FEMALE PROMISCUITY AND MATERNALLY DEPENDENT OFFSPRING GROWTH RATES IN MAMMALS

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Conflicts between family members are expected to influence the duration and intensity of parental care. In mammals, the majority of this care occurs as resource transfer from mothers to offspring during gestation and lactation. Mating systems can have a strong influence on the severity of familial conflict—where female promiscuity is prevalent, conflict is expected to be higher between family members, causing offspring to demand more resources. If offspring are capable of manipulating their mothers and receive resources in proportion to their demands, resource transfer should increase with elevated promiscuity. We tested this prediction, unexplored across mammals, using a comparative approach. The total durations of gestation and lactation were not related to testes mass, a reliable proxy of female promiscuity across taxa. Offspring growth during gestation, however, and weaning mass, were positively correlated with testes mass, suggesting that offspring gain resources from their mothers at faster rates when familial conflict is greater. During gestation, the relationship between offspring growth and testes mass was also related to placenta morphology, with a stronger relationship between testes mass and growth observed in species with a less invasive placenta. Familial conflict could have a pervasive influence on patterns of parental care in mammals.

KEY WORDS: Fecundity, life-history evolution, mating systems, placenta, sexual conflict.

In sexually reproducing species, conflict is expected to be prevalent between parents and offspring over parental care. Parents are equally related to each of their offspring and should, all else being equal, spread their investment evenly among them (Fisher 1930). Each offspring, however, is more related to itself than its siblings, so selection on individual offspring to garner additional parental investment is also expected.

Conflict is also rife between the sexes over the quantity of reproductive allocation each parent makes. Sexual conflict over resource allocation occurs because both parents benefit from any resource allocation to offspring, but parents are separately burdened from the costs of the allocation they make (Lessells and McNamara 2012). In species with predominantly female parental

care, fathers are expected to employ tactics that increase a female's reproductive allocation to their offspring, above the mother's optimal level, as those fathers care less about the mother's future offspring, which could be sired by a different male.

Familial conflict can be considered using Hamilton's rule (Hamilton 1964) and his derived coefficient of relatedness (r). By considering a hypothetical situation of nursing by a female caribou to its newborn calf, Trivers (1974) was the first to demonstrate that conflict should occur between a parent and its offspring over the duration of parental care and quantity of resources transferred. Specifically, the mother should invest in her calf until the cost of investment to her future reproductive success (C) from future offspring (whom she is related by 0.5) outweighs the inclusive
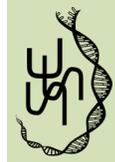
benefits (B) the mother derives from the calf in question (to whom she is also related by 0.5; i.e., when $B_{0.5} < C_{0.5}$). The calf is expected to see this situation differently, as its relatedness to itself ($r = 1$) is greater than its relatedness to its future siblings ($r = 0.5$). As a consequence, the optimal cessation of lactation for the calf will be reached when the costs exceed the benefits from the calf's view (i.e., $B_1 < 2C_{0.5}$). In the vast majority of situations, the offspring's optimal duration of lactation will be longer than the mother's.

Trivers' hypothesis is relevant to all sexually reproducing species that are not completely inbred (Trivers 1974), but at the time of its formulation it was assumed that gene expression was independent of parent of origin. Over the past 20 years it has been revealed that a variety of genes in mammals are expressed differently depending on whether they are inherited from the mother or the father (genomic imprinting). When genes expressed in offspring are imprinted and show differential expression depending on the parent of origin, the consideration they show for future offspring will vary depending on whether they are inherited from the mother or father, and whether these future offspring are likely to be full- or half-siblings (Haig 2002).

Maternally derived genes have a 0.5 probability of being present in her future offspring and will favor allocation in future offspring, over allocation in themselves, only when the benefit to those future offspring is double that which they would gain from allocation to themselves—analogous to the calf's view in Triver's hypothetical example. The probability of paternally derived genes being found in the mother's future or other current offspring (e.g., litter mates) is 0.5 multiplied by the probability that future offspring are sired by the same male. Thus, in the vast majority of instances paternally derived genes will be even less rewarded by allocation to future offspring than maternally derived genes, and this discrepancy will increase with the likelihood that the mother's future offspring will be sired by a different male.

Two comparative studies in birds have separately assessed the relationships between extra-pair paternity and prenatal (Lloyd and Martin 2003) and postnatal (Royle et al. 1999) growth rates, observing that chicks grow quicker in species where extra-pair paternity is high. Because of an absence of genomic imprinting in this taxa, limited prenatal opportunities for offspring manipulation, and a predominance of biparental care, these results have been interpreted in relation to intrabrood sibling competition, where siblings in a brood compete at the same time for a fixed set of resources (Parker 1985; Royle et al. 1999). Faster growth rates may evolve in such situations because of selection on nonimprinted genes inherited from either parent, if this allows individual offspring to attain a greater proportion of the parentally provisioned resources allocated to a brood (Royle et al. 2004).

In mammals, offspring provisioning during lactation and gestation is conducted solely by the mother. Genomic imprinting is present and there is a much greater scope for familial conflict over both inter- and intralitter provisioning. Female promiscuity is expected to elevate conflict between the sexes and intensify selection on paternally derived alleles in offspring that increase offspring growth (and fitness) at the expense of the mother's future reproductive success (Haig 1999). If females produce litters of offspring, sibling competition and selection on nonimprinted genes that increase growth may also be increased, because offspring have less genetic interest in sharing resources with their litter mates. Gestation is expected to be the reproductive period when offspring and paternally derived genes have the best possible opportunity to exert control over maternal resource transfer (Haig 2010). The placenta provides offspring with an intimate association to maternal tissues and, for some species, direct access to the mother's blood. Genomically imprinted genes strongly impact placental development and invasiveness, consistent with the view that the evolution of the placenta has been rife with realized familial conflict (Frost and Moore 2010). Paternally expressed genes usually enhance offspring growth, whereas maternally expressed genes tend to have the opposite effect (DeChiara et al. 1991; Wang et al. 1994; Leighton et al. 1995; Lefebvre et al. 1998).

Two studies have explored the evolutionary consequences of familial conflict on neonatal growth rates across mammals. In one study, the relative conflict between siblings within litters, when determined by the number of offspring competing for each nipple, was found to correlate positively with fetal growth rates within carnivores and insectivores (Stockley and Parker 2002). In the second study, fetal growth rates correlated positively with relative testes mass, an indicator of female promiscuity, across primates (Long 2005). Although the relationships between fetal growth rates and familial conflict have been examined within some mammalian orders, no study has yet examined whether this relationship holds across a range of mammalian orders. Further, fetal growth rates have previously been assessed by using the birth mass/gestation length ratio. This procedure has been criticized because it does not follow the rules of allometric relationships by assuming proportionality between mass and time (Lindstedt and Calder 1981), and because it is not possible to determine whether the independent variable of interest affects the numerator or denominator, or whether one is traded-off against another (Capellini et al. 2011).

Long (2005) also noted that the relationship between relative testes mass and fetal growth rates was stronger within Haplorhine (i.e., tarsiers and anthropoids) than Strepsirhine (i.e., lemuroidea and lorisoidea) primates. This difference, Long suggested, might be attributable to the differences in placenta morphology between these species. Haplorhine primates are notable for having a highly invasive hemochorial placenta, where fetal tissues deeply invade the maternal endometrium and gain direct access to the mother's

blood (Mossman 1987). By contrast, Strepsirhine primates have an epitheliochorial placenta, which is less invasive and has three layers of maternal tissue separating the fetus from the mother's blood (Mossman 1987). Long (2005) suggested that mothers may have more control over the outcome of conflict in species with an epitheliochorial placenta, as the fetus is limited from direct access to the mother's blood. In hemochorial species, the outcome of parent–offspring conflict may be more in favor of offspring and paternally derived genes, as an invasive placenta could allow the offspring greater influence over resource transfer.

Here we test whether fetal growth rates are positively related to testes mass (corrected for body mass), the best marker of female promiscuity and multiple paternity in the absence of behavioral or genetic data (Harcourt et al. 1995; Soulsbury 2010). As our dataset contained species from a range of orders, we also test whether placental invasiveness influences the relationship between intensity of parent–offspring conflict and fetal growth rates. Specifically, we compare the relationship between testes mass and offspring growth within species with a noninvasive epitheliochorial placenta (cetartiodactyles, Strepsirhine primates, American mole) to those with invasive hemochorial (some rodents, Haplorhine primates, hyena) or endotheliochorial (carnivores, elephants, some rodents) placentation. Endotheliochorial placentas show partial invasion of maternal tissues, although the endothelial walls of the mother's blood vessels remain intact (Mossman 1987).

We also test whether the duration of lactation, and the amount of resources transferred over this period, correlate with the level of female promiscuity. If offspring and paternally derived genes are capable of manipulating resource transfer during lactation then a further positive correlation may be observed between relative testes mass and resource transfer over this period. Alternatively, a negative relationship between pre- and postnatal growth rates may be observable in mammals (Stockley and Parker 2002), possibly because growth during these periods can trade-off against each other, or because offspring have less growth to achieve during lactation as a consequence of their fast prenatal development. There may also be greater energetic costs to solicitation of resources during lactation (Stockley and Parker 2002), which may reduce energy allocation to growth or make solicitation of resources less profitable.

Methods
DATASET
Data on combined testes mass (measured without epididymis) and adult body mass come from several published reviews (Kenagy and Trombulak 1986; Tourmente et al. 2011). This dataset was then supplemented with information on gestation length, age at weaning, birth mass, weaning mass, and litter size recorded

in various published databases (Ernest 2003; Jones et al. 2009; Capellini et al. 2011) for all species for which testes mass data were available. We then added information on placenta type using recently published data (Garratt et al. 2013), where the placenta of each individual species was categorized according to the primary literature. Overall, we collected data on 103 mammalian species (notably including 24 rodents, 24 carnivores, 31 cetartiodactyles, and 18 primates). However, the sample size differed between the analyses due to a lack of information for particular traits in some species. All these data and their associated references are provided in Table S1.

COMPARATIVE ANALYSIS
Species can share many characteristics in response to their common ancestry (Felsenstein 1985) and this nonindependence between species needs to be accounted for when testing for possible covariation between biological traits along the tree of life (Harvey and Pagel 1991). In our study, we applied phylogenetic generalized least-squared models (PGLS, Martins and Hansen 1997) that assume a "Brownian model" of evolution (i.e., traits evolved gradually through time independently of the current state [Freckleton et al. 2002]) to control for the nonindependence of datapoints due to phylogenetic relatedness. PGLS analyses provide an estimate of the phylogenetic signal (designed as "λ"), computed through a maximum-likelihood approach (Freckleton et al. 2002). The "λ" value generally varies between 0 (complete absence of phylogenetic signal) and 1 (phylogenetic structure in agreement with a Brownian model of evolution) and is introduced into the regression model to account for the confounding influence of phylogeny on the tested relationships (Freckleton et al. 2002). For these analyses, we used the phylogenetic supertree of mammals published by Bininda-Emonds et al. (2007) that provides information on both topology and branch length.

METRICS AND STATISTICAL TESTS
We examined how relative testes mass (a proxy of multiple paternity) was related to the dependent variable of interest (e.g., gestation length, birth mass, lactation length, or weaning mass) while also including several other covariates in the model that explain variance in the dependent variable (Lemaitre et al. 2009; Ramm and Stockley 2010). To avoid the use of residuals that can lead to biased estimates (Freckleton 2009), we measured relative testes mass by adding male body mass as a covariate in models including testes mass, with both variables log-transformed to satisfy allometric rules (Lemaitre et al. 2009; Ramm and Stockley 2010). In our dataset, the relationship between testes mass and body mass was linear ($n = 103$, slope $\pm$ SE: 1.06 ± 0.08, $t = 13.42$, $P < 0.001$) with no evidence for nonlinearity (Fig. S1). Diagnostic tests for collinearity problems were conducted, and

Table 1. Phylogenetically corrected models testing the influence of key life-history traits and invasive versus noninvasive placenta morphology on gestation length and birth mass.

	Independent variables	Estimate	t	P
Gestation length, $n = 79$, $\lambda = 0.99$	Intercept	0.60 ± 0.25	2.39	0.02
	Body mass	0.11 ± 0.03	4.23	<0.0001
	Testes mass	0.03 ± 0.03	0.81	0.42
	Litter size	-0.38 ± 0.08	-4.61	<0.0001
Birth mass, $n = 75$, $\lambda = 0.99$	Intercept	-0.39 ± 0.38	-1.01	0.31
	Body mass	0.44 ± 0.04	10.90	<0.0001
	Testes mass	0.14 ± 0.05	2.86	0.006
	Litter size	-0.23 ± 0.13	-1.68	0.10
	Gestation length	1.34 ± 0.17	7.89	<0.0001
Birth mass with placentation, $n = 75$, $\lambda = 0.99$	Intercept	-0.30 ± 0.38	-0.78	0.44
	Body mass	0.46 ± 0.04	11.61	<0.0001
	Testes mass	0.06 ± 0.05	1.11	0.27
	Litter size	-0.23 ± 0.13	-1.79	0.077
	Gestation length	1.37 ± 0.16	8.36	<0.0001
	Placenta	-1.01 ± 0.40	-2.52	0.014
	Placenta × testes	0.25 ± 0.08	2.96	0.004
Birth mass: noninvasive placenta, $n = 23$, $\lambda = 0.99$	Intercept	-1.90 ± 0.87	-2.20	0.041
	Body mass	0.55 ± 0.08	7.25	<0.0001
	Testes mass	0.30 ± 0.08	3.89	0.001
	Litter size	-0.27 ± 0.27	-1.01	0.33
	Gestation length	1.17 ± 0.45	2.59	0.018
Birth mass: invasive placenta, $n = 51$, $\lambda = 0.99$	Intercept	-0.17 ± 0.43	-0.39	0.70
	Body mass	0.43 ± 0.05	8.85	<0.0001
	Testes mass	0.09 ± 0.06	1.40	0.17
	Litter size	-0.21 ± 0.15	-1.43	0.16
	Gestation length	1.41 ± 0.18	7.63	<0.0001

All variables are log-transformed. The effect of litter size on birth mass was weak and removal of this variable from the model does not affect the results (Table S3).

in each model the variance inflation factors (VIFs) and tolerance were below critical values (Dormann et al. 2013; see Table S2).

The relationship between testes mass and the lengths of gestation and lactation were analyzed while additionally including body mass and litter size as covariates (both log-transformed), as both these variables had an impact on the durations of these reproductive periods (Tables 1 and 2). When analyzing the relationship between testes mass and fetal growth rate, birth mass was included as the dependent variable and gestation length was added as an additional covariate; again, both variables were log-transformed. This modeling approach allowed us to test for an effect of testes mass on fetal growth (Capellini et al. 2011). Placenta type was added as an independent variable and coded as a factor (hemochorial and endotheliochorial placentas were coded as 0 and epitheliochorial placenta was coded as 1). Species were categorized in relation to whether they had an invasive (hemochorial/endotheliochorial) or noninvasive (epitheliochorial) placenta because no differences were apparent between species

with hemochorial and endotheliochorial placentas (comparison between these species for birth mass: $t = -1.43$, $P = 0.16$, interaction between these aspects of placentation and testes mass for birth mass: $t = 1.28$, $P = 0.21$; comparison between these species for weaning mass: $t = -0.30$, $P = 0.78$, interaction between these aspects of placentation and testes mass for weaning mass: $t = -59$, $P = 0.55$). This is consistent with two recent studies that demonstrated species with hemochorial placentas do not differ from those with endotheliochorial placentas in various aspects of life history (Capellini et al. 2011; Garratt et al. 2013) and these species were similarly combined for analysis.

In a second step, we analyzed the relationship between testes mass and early postnatal growth rate (i.e., growth between birth and weaning). We followed the same modeling approach, including weaning mass as the dependent variable and weaning age as a covariate, although weaning age was subsequently dropped from the model as it did not correlate with weaning mass.

Table 2. Phylogenetically corrected models testing the influence of key life-history traits and placenta morphology on weaning age and weaning mass.

	Independent variables	Estimate	t	P
Weaning age, $n = 92$, $\lambda = 0.72$	Intercept	3.45 ± 0.33	10.42	<0.0001
	Body mass	0.15 ± 0.04	3.94	0.0002
	Testes mass	0.07 ± 0.05	1.32	0.19
	Litter size	-0.54 ± 0.12	-4.48	<0.0001
Weaning mass without placentation, $n = 61$, $\lambda = 0.31$	Intercept	1.55 ± 0.47	3.32	0.002
	Body mass	0.65 ± 0.06	10.39	<0.0001
	Testes mass	0.19 ± 0.08	2.22	0.031
	Litter size	-0.70 ± 0.17	-4.10	0.0001
	Lactation length	0.13 ± 0.15	0.87	0.39
Weaning mass with placentation, $n = 49$, $\lambda \leq 0.0001$	Intercept	1.14 ± 0.45	2.53	0.014
	Body mass	0.65 ± 0.07	9.91	<0.0001
	Testes mass	0.20 ± 0.09	2.14	0.038
	Litter size	-0.53 ± 0.17	-3.10	0.003
	Placenta	0.64 ± 0.22	2.92	0.006
	Placenta $\times$ testes	0.21 ± 0.14	1.53	0.13

All variables are log-transformed. Lactation length was dropped from the final model of weaning mass (without placentation).

Results

FETAL GROWTH RATE

Gestation length, after controlling for body mass and litter size, was not detectably related to testes mass (Table 1; Fig. 1A), a proxy of female promiscuity and parent–offspring conflict.

Birth mass increased with gestation length and adult body mass and tended to decrease with litter size. After accounting for these relationships previously reported in comparative studies of mammalian life-history variation, birth mass also increased with relative testes mass (Table 1; Fig. 1B), in support of the prediction that prenatal maternal allocation increases in situations of heightened conflict. We also found an interaction between placenta invasiveness and testes mass on fetal growth (Table 1). The effect of relative testes mass on fetal growth was stronger in species with an epitheliochorial placenta than in those with an invasive (hemochorial or endotheliochorial) placenta (Table 1; Fig. 1B).

EARLY POSTNATAL GROWTH RATE

Age at weaning markedly increased with adult body mass ($n = 92$, $t = 3.94$, $P = 0.0002$). Relative testes mass tended to increase with weaning age ($n = 92$, $t = 1.94$, $P = 0.055$; Fig. 1C), but this effect vanished when litter size was also included in the model (Table 2).

After controlling for the effects of body mass and litter size, weaning mass was positively correlated with testes mass (Table 2; Fig. 1D). Weaning mass was also influenced by placentation, although there were no interactive effects between placenta type

and testes mass as reported for fetal growth (Table 2). For a given relative testes mass, the weaning mass of species with an epitheliochorial placenta is heavier than in species with a more invasive placenta (Fig. 1D), which may relate to the faster pace of life previously reported in species with an epitheliochorial placenta (Garratt et al. 2013).

Discussion

Our results reveal that across mammals, relative testes mass—a strong correlate of female promiscuity—predicts maternally dependent offspring growth rates during gestation and lactation. These findings are consistent with theoretic predictions that parent–offspring conflict and sexual conflict could lead to offspring soliciting more resources from mothers in species where females mate with multiple males and where there is a greater probability of siblings only being related via maternal genes (Trivers 1974; Haig 1999; Bossan et al. 2013).

Our results generalize Long's (2005) findings that neonatal mass/gestation length, a proxy of fetal growth, was positively correlated with relative testes mass across primates (Long 2005). By controlling for phylogenetic dependence among species and for allometric rules, we show that in species with relatively larger testes, neonates weigh more at parturition for a given gestation length. Yet gestation length itself is not related to relative testes mass. Perhaps more importantly, we demonstrate that a strong positive relationship between fetal growth and relative testes mass
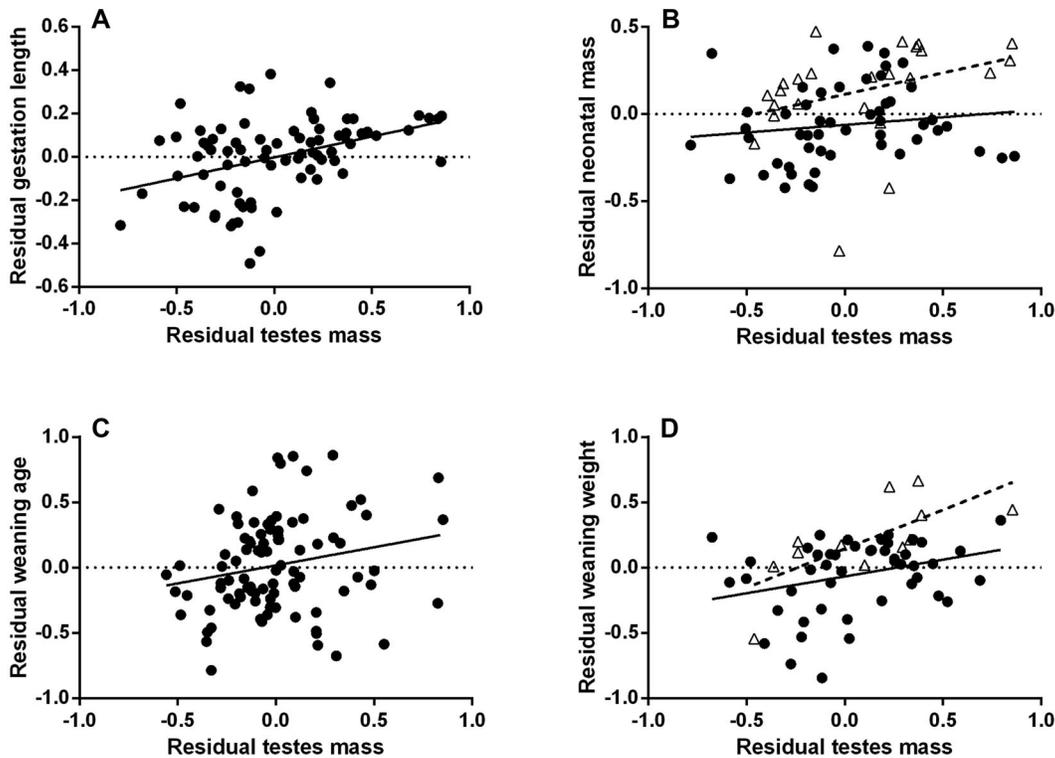
Figure 1. The relationship between relative testes mass and (A) gestation length, (B) birth mass, (C) weaning age, and (D) weaning mass. The *x*-axis represents unstandardized residuals from an ordinary least squares (OLS) regression of log-transformed testes mass against log-transformed body mass. (A) *Y*-axis represents unstandardized residuals from OLS regression of log-transformed gestation length against log-transformed body mass. (B) Residual birth mass represents unstandardized residuals of log-transformed birth mass against both log-transformed body mass and gestation length. (C) Residual weaning age represents unstandardized residuals of log-transformed weaning age against log-transformed body mass. (D) Residual weaning mass represents unstandardized residuals of log-transformed weaning mass against both log-transformed body mass and weaning age. Where a statistically significant effect of placentation was detected, epitheliochorial species are denoted with open triangles, and invasive species (hemochorial and endotheliochorial species) with black dots.

occurs broadly across the Eutheria, rather than just within the primate order.

Stockley and Parker (2002) reported that sibling rivalry, a component of familial conflict, was correlated with faster neonatal growth in species of carnivores and insectivores. However, sibling rivalry was then correlated with slower postnatal growth relative to size at birth. We examine a different component driving familial conflict—female promiscuity—and find this is also correlated with faster neonatal growth, but we do not observe the same trade-off with postnatal growth rates. Our analysis was not restricted to a particular mammalian family and we also examine a different component of conflict, which may be at least partly driven by selection on paternally derived genes. Stockley and Parker (2002) assessed sibling rivalry and the number of offspring per nipple, so predictions with regard to postnatal growth may be different. These authors also used a different method of analysis for postnatal growth and included neonatal mass as a covariate. Because we include two other covariates in our study, and because

there is a strong correlation between neonatal and weaning mass in mammals (Lee et al. 1991), we are unable to include neonatal mass in our analysis without generating substantial collinearity (Table S2). This difference in method might also contribute to the different results. Stockley and Parker's slowdown in postnatal growth with sibling rivalry is in relation to birth mass, while our study demonstrates that female promiscuity is correlated with the absolute mass of offspring at weaning.

Offspring growth rates could be linked to female promiscuity through several different avenues. Some theory predicts that heightened female promiscuity will increase selection on paternally derived genomically imprinted genes that favor faster offspring growth (Haig 1999, 2002), although selection on maternally derived genes may counteract this response. There is evidence that some paternally derived genomically imprinted genes do increase offspring growth, because inhibiting the expression of these genes in neonates causes offspring to be smaller at birth (DeChiara et al. 1991; Lefebvre et al. 1998). A similar

effect has also been observed with a genomically imprinted gene that influences growth during lactation (Itier et al. 1998). It is important to note, however, that within brood extra-pair paternity in birds is also positively correlated with offspring growth rates (Royle et al. 1999; Lloyd and Martin 2003), even though this taxa does not show genomic imprinting (Arnqvist and Rowe 2005). In these studies, it has been suggested that elevated conflict between siblings may select for nonimprinted genes that increase growth rates, because being relatively bigger than brood mates allows chicks to win scrambles or contests for food (Royle et al. 1999). It is difficult to draw direct comparisons between our study and the two previously published studies in birds, particularly as prenatal growth rates were not assessed in relation to body mass (Lloyd and Martin 2003) and postnatal growth was determined using a logistic function (Royle et al. 1999). Nonetheless, mammalian siblings also compete for access to postnatally provided maternal resources, so sibling competition may also be driving selection for increased offspring growth, and genomic imprinting is not necessary for such relationships to occur across animals.

A drawback of such broad-scale comparative studies is the inability to separate cause and effect. It is also possible that a larger relative birth mass or a larger relative mass at weaning facilitates development of relatively larger testes, or different mating systems select for differences in growth rates during parental care. If multiple mating enhances the genetic quality of offspring, this could also enhance offspring growth rates. In a recent meta-analysis, the genetic benefits of polyandry were found to have only a weak effect on offspring growth (Slatyer et al. 2012), but it would be incautious to discard this alternative possible explanation without further evidence. Another possibility is that enhanced growth rates are not a consequence of greater resource allocation by the mother, but due to offspring allocating a greater proportion of the resources they gain to growth, and away from other biological functions, such as somatic maintenance. Under the latter hypothesis, the enhanced growth rate would be expected to trade-off against some other life-history trait in offspring, while under our original hypothesis the enhanced growth rate is facilitated by a greater maternal allocation in resources and so may be less costly for offspring. Our results show that the predicted correlations are present in nature—we urge further experimental studies to determine the causal factors underlying these relationships.

The durations of gestation and lactation, after accounting for differences in body mass and litter size, were not associated with relative testes mass. Trivers (1974) predicted that there would be conflict between parents and offspring over the duration of resource transfer, with offspring favoring a longer duration of parental care in most situations. Likewise, Haig (2010) suggested that offspring fitness will be enhanced by longer gestations. Thus, in situations of heightened conflict we expected the duration of both gestation and lactation to increase if offspring exert a level of

control over the duration of these maternal allocation periods. Our results suggest that this is not the case. It is possible that mammalian mothers have a high degree of control over the duration of gestation and lactation, with offspring having little scope to manipulate mothers into extending the duration of their reproductive allocation. It has also been suggested that there could be an arms race between paternally derived genes that increase resource transfer to offspring and maternal genes that reduce the length of reproductive transfer to offset these effects (Capellini et al. 2011). Thus, maternal control over the duration of reproductive provisioning may provide a counterstrategy that allows parents to limit their investment in individual offspring.

By using a dataset encompassing species from most mammalian lineages we were also able to test whether placental invasiveness moderates the effects of relative testes mass, and therefore family conflict, on fetal growth. The only previous examination of this subject compared just two lineages of primate with different placenta types (Long 2005). It was a long-held assumption that the highly invasive hemochorial placenta evolved from the less invasive epitheliochorial placenta in situations of heightened familial conflict, to allow greater control by offspring over resource transfer (Haig 1993; Crespi and Semeniuk 2004). However, it has recently been determined that a more invasive placenta type was the ancestral type in mammals and epitheliochorial placentation has evolved at least three times independently (Wildman et al. 2006). Furthermore, epitheliochorial species have a faster pace of life than hemochorial species (Garratt et al. 2013), with such a life history generally expected to lead to heightened conflict (Bossan et al. 2013; Garratt et al. 2013).

After correcting for adult body size, litter size, and phylogeny, the effect of testes mass on birth mass was stronger in species with a noninvasive epitheliochorial placenta, when compared to species with either a hemochorial or endotheliochorial placenta. This contrasts with Long's (2005) conclusion that the link between testes mass and offspring growth was stronger in Haplorhine primates—with invasive hemochorial placentas—when compared to just Strepsirhine primates—which generally have less invasive epitheliochorial placentas. The species used in our analysis are taxonomically more diverse when compared to those of Long (2005), with our dataset containing species from each major evolutionary transition in placentation (Garratt et al. 2013). It should be noted though that the total number of major transitions in placental invasiveness are few (Wildman et al. 2006) and epitheliochorial placentation has only evolved independently in cetartodactyla, Strepsirhine primates, and the American mole. The majority of epitheliochorial species in our study are derived from cetartiodactyla, as we only included species for which a primary placentation reference could be found, and this group has been by far the best studied with regards to placentation. Within cetartiodactyla, Soay sheep, *Ovis aries*, which are frequently used

as an illustrative example of a very promiscuous mating system, have a relatively large fetal mass in relation to their gestation length. At the other end of the spectrum, moose, *Alces alces*, which are known to have a mating system of low female promiscuity, have a relatively low neonatal mass. Despite the low levels of evolutionary replication, we can conclude that the link between promiscuity and offspring growth is clearly present within species with an epitheliochorial placenta, at least across the subset of species for which placentation has been studied. This highlights that this aspect of placenta morphology does not inhibit faster neonatal growth in situations of heightened conflict. We suggest that the opposite relationship is found here because species with an epitheliochorial placenta type have a faster pace of life (Garratt et al. 2013), which may further elevate parent–offspring conflict (Bossan et al. 2013). It is also worth noting that many species with epitheliochodrial placentation, such as most ungulates, produce precocial offspring (Martin 2008). It is thus possible that offspring gestating at a later development stage could have greater control over resource transfer. Comparisons of neonatal growth at different gestational periods and stages of neonatal development may provide a novel test of this hypothesis.

Ultimately, the currency of familial conflict is parental investment—any parental expenditure in an offspring that reduces a parent's ability to invest in future offspring (Trivers 1972). Our results show that in situations of heightened conflict offspring are relatively heavier at the end of gestation and lactation. Thus, the total amount of resources they receive is likely to be greater (i.e., higher maternal allocation). How this impacts on a mother's future ability to allocate resources to future offspring (i.e., maternal investment) is unknown, but life-history theory predicts that increased investment in current offspring will trade-off against future reproductive investment or some other aspect of life history (Stearns 1992). Such effects remain to be determined and could be fruitful areas for experimental and comparative works.

LITERATURE CITED

Arnqvist, G., and L. Rowe. 2005. Sexual conflict. Princeton Univ. Press, Princeton, NJ.

Bininda-Emonds, O. R. P., M. Cardillo, K. E. Jones, R. D. E. MacPhee, R. M. D. Beck, R. Grenyer, S. A. Price, R. A. Vos, J. L. Gittleman, and A. Purvis. 2007. The delayed rise of present-day mammals. Nature 446:507–512.

Bossan, B., P. Hammerstein, and A. Koehncke. 2013. We were all young once: an intragenomic perspective on parent–offspring conflict. Proc. R. Soc. B 280:20122637.

Capellini, I., C. Venditti, and R. A. Barton. 2011. Placentation and maternal investment in mammals. Am. Nat. 177:86–98.

Crespi, B., and C. Semeniuk. 2004. Parent-offspring conflict in the evolution of vertebrate reproductive mode. Am. Nat. 163:635–653.

DeChiara, T. M., E. J. Robertson, and A. Efstratiadis. 1991. Parental imprinting of the mouse insulin-like growth factor II gene. Cell 64:849–859.

Dormann, C. F., J. Elith, S. Bacher, C. Buchmann, G. Carl, G. Carré, J. R. G. Marquéz, B. Gruber, B. Lafourcade, P. J. Leitão, et al. 2013. Collinearity: a review of methods to deal with it and a simulation study evaluating their performance. Ecography 36:027–046.

Ernest, S. K. M. 2003. Life history characteristics of placental nonvolant mammals. Ecology 84:3402.

Felsenstein, J. 1985. Phylogenies and the comparative method. Am. Nat. 125:1–15.

Fisher, R. A. 1930. The genetical theory of natural selection. Clarendon Press, Oxford, U.K.

Freckleton, R. P. 2009. The seven deadly sins of comparative analysis. J. Evol. Biol. 22:1367–1375.

Freckleton, R. P., P. H. Harvey, and M. Pagel. 2002. Phylogenetic analysis and comparative data: a test and review of evidence. Am. Nat. 160:712–726.

Frost, J. M., and G. E. Moore. 2010. The importance of imprinting in the human placenta. PLoS Genet. 6.

Garratt, M., J.-M. Gaillard, R. C. Brooks, and J.-F. Lemaître. 2013. Diversification of the eutherian placenta is associated with changes in the pace of life. Proc. Natl. Acad. Sci. USA 110:7760–7765.

Haig, D. 1993. Genetic conflicts in human pregnancy. Q. Rev. Biol. 68:495–532.

———. 1999. Multiple paternity and genomic imprinting. Genetics 151:1229–1231.

———. 2002. Genomic imprinting and kinship. Rutgers Univ. Press, New Brunswick.

———. 2010. Transfers and transitions: parent-offspring conflict, genomic imprinting, and the evolution of human life history. Proc. Natl. Acad. Sci. USA 107:1731–1735.

Hamilton, W. D. 1964. The genetical evolution of social behavior. J. Theor. Biol. 7:1–52.

Harcourt, A. H., A. Purvis, and L. Liles. 1995. Sperm competition—mating system, not breeding-season, affects testes size of primates. Funct. Ecol. 9:468–476.

Harvey, P. H., and M. Pagel. 1991. The comparative method in evolutionary biology. Oxford Univ. Press, Oxford, U.K.

Itier, J. M., G. L. Tremp, J. F. Leonard, M. C. Multon, G. Ret, F. Schweighoffer, B. Tocque, M. T. Bluet-Pajot, V. Cormier, and F. Dautry. 1998. Imprinted gene in postnatal growth role. Nature 393:125–126.

Jones, K. E., J. Bielby, M. Cardillo, S. A. Fritz, J. O'Dell, C. D. L. Orme, K. Safi, W. Sechrest, E. H. Boakes, C. Carbone, et al. 2009. PanTHERIA: a species-level database of life history, ecology, and geography of extant and recently extinct mammals. Ecology 90:2648.

Kenagy, G. J., and S. C. Trombulak. 1986. Size and function of mammalian testes mass in relation to body size. J. Mammal. 67:1–22.

Lee, P. C., P. Majluf, and I. J. Gordon. 1991. Growth, weaning and maternal investment from a comparative perspective. J. Zool. 225:99–114.

Lefebvre, L., S. Viville, S. C. Barton, F. Ishino, E. B. Keverne, and M. A. Surani. 1998. Abnormal maternal behaviour and growth retardation associated with loss of the imprinted gene Mest. Nat. Genet. 20:163–169.

Leighton, P. A., R. S. Ingram, J. Eggenschwiler, A. Efstratiadis, and S. M. Tilghman. 1995. Disruption of imprinting caused by deletion of the H19 gene region in mice. Nature 375:34–39.

Lemaitre, J. F., S. A. Ramm, R. A. Barton, and P. Stockley. 2009. Sperm competition and brain size evolution in mammals. J. Evol. Biol. 22:2215–2221.

Lessells, C. M., and J. M. McNamara. 2012. Sexual conflict over parental investment in repeated bouts: negotiation reduces overall care. Proc. Biol. Sci. 279:1506–1514.

Lindstedt, S. L., and W. A. Calder. 1981. Body size, physiological time, and longevity of homeothermic animals. Q. Rev. Biol. 56:1–16.

Lloyd, J. D., and T. E. Martin. 2003. Sibling competition and the evolution of prenatal development rates. Proc. R. Soc. Lond. Ser. B 270:735–740.

Long, T. A. F. 2005. The influence of mating system on the intensity of parent-offspring conflict in primates. J. Evol. Biol. 18:509–515.

Martin, R. D. 2008. Evolution of placentation in primates: implications of mammalian phylogeny. Evol. Biol. 35:125–145.

Martins, E. P., and T. F. Hansen. 1997. Phylogenies and the comparative method: a general approach to incorporating phylogenetic information into the analysis of interspecific data. Am. Nat. 149:646–667.

Mossman, H. W. 1987. Vertebrate fetal membranes. Rutgers Univ. Press, New Brunswick.

Parker, G. A. 1985. Models of parent-offspring conflict. V. Effects of the behaviour of the two parents. Anim. Behav. 33:519–533.

Ramm, S. A., and P. Stockley. 2010. Sperm competition and sperm length influence the rate of mammalian spermatogenesis. Biol. Lett. 6:219–221.

Royle, N. J., I. R. Hartley, I. P. F. Owens, and G. A. Parker. 1999. Sibling competition and the evolution of growth rates in birds. Proc. R. Soc. B 266:923–932.

Royle, N. J., I. R. Hartley, and G. A. Parker. 2004. Parental investment and family dynamics: interactions between theory and empirical tests. Popul. Ecol. 46:231–241.

Slatyer, R. A., B. S. Mautz, P. R. Backwell, and M. D. Jennions. 2012. Estimating genetic benefits of polyandry from experimental studies: a meta-analysis. Biol. Rev. 87:1–33.

Soulsbury, C. D. 2010. Genetic patterns of paternity and testes size in mammals. PLoS One 5:A152–A157.

Stearns, S. C. 1992. The evolution of life histories. Oxford Univ. Press, Oxford, U.K.

Stockley, P., and G. A. Parker. 2002. Life history consequences of mammal sibling rivalry. Proc. Natl. Acad. Sci. USA 99:12932–12937.

Tourmente, M., M. Gomendio, and E. R. S. Roldan. 2011. Sperm competition and the evolution of sperm design in mammals. BMC Evol. Biol. 11:12.

Trivers, R. L. 1972. Parental investment and sexual selection. Pp. 136–179 *in* B. Campbell, ed. Sexual selection and the descent of man 1871–1971. Aldine, Chicago.

———. 1974. Parent-offspring conflict. Am. Zool. 14:249–264.

Wang, Z.-Q., M. R. Fung, D. P. Barlow, and E. F. Wagner. 1994. Regulation of embryonic growth and lysosomal targeting by the imprinted Igf2/Mpr gene. Nature 372:464–467.

Wildman, D. E., C. Y. Chen, O. Erez, L. I. Grossman, M. Goodman, and R. Romero. 2006. Evolution of the mammalian placenta revealed by phylogenetic analysis. Proc. Natl. Acad. Sci. USA 103:3203–3208.

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Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's website:

Table S1. Supplementary dataset.
Table S2. Collinearity diagnostics.
Table S3. Final models for gestation length and birth mass with litter size removed.
Figure S1.