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Maternal and genetic correlations between morphology and physical performance traits in a small captive primate, *Microcebus murinus*

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Physical performance traits are key components of fitness and direct targets of selection. Although maternal effects have important influences on integrated phenotypes, their contributions to variation in performance and to phenotypic traits associated with performance remain poorly understood. We used an animal model to quantify the contribution of maternal effects to performance trait variation, in addition to the genetic and maternal correlations between performance and the relevant underlying morphology in *Microcebus murinus*. We showed that bite force is heritable ($h^2 \approx 0.23$) and that maternal effects are an important source of variation, resulting in a medium inclusive heritability ($IH^2 \approx 0.47$). Bite force and head depth showed a significant genetic correlation (0.70), and other genetic correlations were generally high (0.63 for bite force and head width; 0.41 for pull strength and radius length, albeit not significant), as were the maternal correlations for bite force and head dimensions (0.44, 0.73 and 0.29). Finally, we found differences in evolvability for pull strength and bite force that were also consistent with a higher potential for evolutionary change in pull force. This demonstrates clear effects of the maternal environment on performance expression and on the relationships between morphology and performance. This illustrates the importance of accounting for maternal identity when considering the heritabilities of functional traits.

ADDITIONAL KEYWORDS: bite force – genetic correlations – heritability – maternal effects – pull strength.

INTRODUCTION

Phenotypic variation is necessary for natural selection to act. However, understanding the potential for obtaining an evolutionary response to selection first requires quantification of the relevant sources of phenotypic variation and distinguishing among them (Lande & Arnold, 1983; Endler, 1986). Variation in measurable phenotypes is attributable to both genetic and environmental effects, and in the same way that genetic effects can be partitioned into additive and non-additive genetic factors, environmental factors can also comprise multiple components (reviewed by Bonduriansky & Day, 2009, 2018). Maternal effects are an important non-genetic contributor to phenotypic variation, even in animal species that lack maternal care. Maternal effects can have an impact both directly, via adaptive maternal resource allocation or clutch manipulation (Mousseau & Fox, 1998), and indirectly, via incidental effects of maternal age on offspring survival and fitness (Ivimey-Cook & Moorad, 2020; but regarding maternal genetic effects, see also Wilson *et al.*, 2005; Wolf & Wade, 2009). Despite the extensive literature on non-genetic maternal effects, studies within a quantitative genetic framework commonly focus either on direct measures of traits, such as offspring survival, or on offspring phenotypes that are tied to reproduction, such as life-history traits.

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A central principle underlying the field of ecomorphology, which deals with the relationships among fitness, performance and the structural predictors of performance, is that selection acts on performance as opposed to on the underlying anatomy, physiology or genes (Arnold, 1983). In addition to being products of the genome, epigenome and environment, performance traits therefore also capture the integrated output of multiple layers of biological organization (West-Eberhard, 1989; Chen *et al.*, 2013; Glastad *et al.*, 2019). Most studies testing the heritability of physical performance traits to date have focused on locomotor performance (Tsuji *et al.*, 1989; Garland *et al.*, 1990; Sorci *et al.*, 1995; Berwaerts *et al.*, 2008; Noble *et al.*, 2014) and have demonstrated low to moderate heritabilities for specific aspects of locomotion in a range of animal taxa. More generally, not only locomotion, but also overall physical performance appears to be heritable in humans (Bouchard *et al.*, 2011), with ~20% of the variation in the ability to win a medal at the Olympics, for example, being attributable to genetics (Antero *et al.*, 2018). Likewise, overall racing performance appears to be heritable in horses (Bokor *et al.*, 2006; Velie *et al.*, 2014; Sharman & Wilson, 2015). Despite this heritable nature of performance, the extent to which non-genetic factors influence the expression of whole-organism performance phenotypes has seldom been tested within a rigorous quantitative genetic framework.

Maternal effects are defined as the causal influence of the maternal phenotype or genotype on offspring phenotype (Wolf & Wade, 2009; see also Mousseau & Fox, 1998) and are widespread in both plants and animals (Mousseau & Fox, 1998). This means that the 'environment' provided by the mother through any of several avenues, ranging from prenatal resource allocation to postnatal maternal care, can significantly impact offspring developmental trajectories and, ultimately, both juvenile and adult phenotypes (Reinhold, 2002; Power & Schulkin, 2016). Non-genetic factors can significantly influence the expression of phenotypic traits, either independent of genetic effects or through explicit interactions with them (Falconer & Mackay, 1996; Wolf & Wade, 2009), leading some researchers to define the concept of 'inclusive heritability', which encompasses all sources of phenotypic variation that are inherited across generations (Danchin & Wagner, 2010). Estimation of non-genetic effects is necessary to avoid overestimation of additive genetic variance, hence narrow-sense heritability (Wilson *et al.*, 2010). Variance partitioning is useful to estimate the genetic and non-genetic components of phenotypic variation in animal models (Postma & Charmantier, 2007; Hill, 2010); however, this approach requires either an at least moderately powerful breeding design or a pedigree comprising multiple offspring per mother (Kruuk *et al.*, 2004, 2007). Given that these requirements render the estimation of maternal effects in most animal species logistically challenging, and despite the importance of maternal effects to explain phenotypic variability in natural and captive populations for a variety of traits (Forstmeier *et al.*, 2004; Taylor *et al.*, 2012; Ariyomo *et al.*, 2013; Zablocki-Thomas *et al.*, 2019), the relative importance of maternal effects compared to additive genetic effects or common environmental effects on fitness-relevant traits, such as physical performance, have been estimated in only a handful of quantitative genetic studies (Blumstein *et al.*, 2010; Noble *et al.*, 2014).

In addition to being sources of phenotypic variance, both maternal and additive genetic effects also contribute to the phenotypic and genetic correlations among suites of traits. Previous studies have implicated performance traits as integral components of suites of traits, including morphology, behaviour and life history, that are interconnected via genetic correlations (Réale *et al.*, 2007; Le Galliard *et al.*, 2013). Such traits can be selected for and inherited together, as in the case of the lizard *Zootoca vivipara*, in which exploratory behaviour and resting metabolic rate are under correlated selection (Le Galliard *et al.*, 2013). Furthermore, Kern *et al.* (2016) demonstrated the existence of genetic correlations between morphology, performance and personality by selecting individuals on their personality and measuring the change in morphology and performance. Nonetheless, our understanding of the genetic correlations between morphology and the performance capacities driven by variation in morphology is extremely limited. Beyond genetic correlations, maternal correlations, which are attributable to the shared effects of variability in maternal factors on variation in the traits in question (Cheverud *et al.*, 1983), have been measured among traits such as behaviours (e.g. Taylor *et al.*, 2012). Yet, these are seldom considered within the context of performance evolution specifically, despite the potential for maternal effects to affect suites of traits that include performance.

In this study, we take advantage of the reconstructed partial pedigree of a large colony of a small captive primate, the grey mouse lemur (*Microcebus murinus*), to evaluate the relative influence of maternal effects and the additive genetic variance on performance and morphological traits by using an animal model analysis to estimate narrow-sense and inclusive heritability. The grey mouse lemur is a useful model for studying maternal effects given its remarkably fast life cycle, reproducing yearly, with each mother giving birth to two or three offspring per litter. In addition, *M. murinus* can live up to 10 years in captivity, providing the opportunity to study several generations and multiple litters per mother in a short period. Previous studies have demonstrated

the relevance of physical performance capacities to reproductive success in this species; for example, pull strength, a repeatable performance trait, is correlated with the number of offspring in captivity (Thomas *et al.*, 2016). However, the additive genetic and maternal contributions to pull strength and bite force variation, in addition to the genetic and maternal correlations between performance and the underlying morphological traits that determine it, have never been quantified rigorously.

We estimate both the narrow-sense heritabilities and inclusive heritabilities accounting for maternal effects for each of two performance traits (pull strength and bite force) and associated morphological traits. Based on previous studies (Thomas *et al.*, 2015, 2016), we predict that pull strength might be under strong selection in this arboreal species and, as such, will exhibit a lower narrow-sense heritability than bite force (Mousseau & Roff, 1987; Price & Schluter, 1991). We also expect these traits to be moderately repeatable (Thomas *et al.*, 2015, 2016). We expect the evolvability of performance traits to be relatively high (Lailvaux *et al.*, 2010), yet rather low for morphometric traits (Houle, 1992). We further predict that maternal effects should be significant for both performance traits, owing to the significant investment of the mother in the growth of the offspring. Finally, we predict genetic and maternal correlations between functionally relevant morphological traits and the associated performance traits (i.e. head dimensions and bite force; forearm length and pull strength), allowing the evolution of fitness-relevant co-adapted character complexes (Savitzky, 1983; Matioli & Templeton, 1999).

MATERIAL AND METHODS

Animals

We collected data for 486 grey mouse lemurs (*M. murinus*), 247 females and 239 males, aged between 1 and 10 years and housed in large aviaries (167 cm × 60 cm × 70 cm) in the captive colony of mouse lemurs in Brunoy (France) (MNHN; agreement no. F91-114-1). The ambient air temperature is maintained at 25 °C, and humidity is stable around 30%. All individuals are fed *ad libitum*, weighed monthly, and maintained under artificial light conditions mimicking natural seasons. Not all individuals participated in all datasets (morphology, bite force and pull strength) because of availability issues (death, breeding or the involvement in other experiments). All measurements were approved by, and in accordance with, the guidelines of the local institutional ethics committee and with European guidelines for the use of animals in research (Directive 2010/63/EU).

Bite force

We used a piezo-electric transducer (Kistler, type 9203, range ±500 N; Kistler, Winterthur, Switzerland) attached to a hand-held charge amplifier (Kistler, type 5995) to record bite force for 401 individuals. The transducer was placed between two plates that animals had to bite, as described by Herrel *et al.* (1999) (for studies on mouse lemurs, see also Chazeau *et al.*, 2013; Thomas *et al.*, 2015). We covered the bite plates with a layer of cloth medical tape to provide grip and to protect the teeth of the animals. Next, we adapted the distance between plates to the size of the lemurs in order to measure bite force during unilateral molar biting, where bite force is maximized in mammals (Dumont & Herrel, 2003). We measured three bites per session, and only the highest bite force for each session was kept for the analysis. We recorded 474 bite forces for 401 individuals, meaning that bite force was repeated 1.18 times per animal on average.

Pull strength

We used a small iron bar mounted on a piezo-electric force platform (Kistler squirrel force plate, ±0.1 N; Winterthur, Switzerland) to measure pull strength. The force platform was positioned on a custom-designed metal base and connected to a charge amplifier (Kistler charge amplifier, type 9865). During sessions lasting 60 s (1 kHz), we allowed the animals to grip a dowel with their hands and pulled them away horizontally from the dowel several times. We extracted peak forces in the horizontal direction with Bioware software (Kistler), and we kept the highest force obtained for the analysis (Thomas *et al.*, 2016). We recorded 486 pull strength trials for 399 individuals, meaning that pull strength was repeated 1.21 times per animal on average.

Morphology

We recorded head length, width and depth and forearm, tibia and metatarsus length for 417 mouse lemurs with digital callipers (±0.01 mm; Mitutoyo, Kanagawa, Japan; see Chazeau *et al.*, 2013). We extracted body weight at birth for the 486 individuals from the database of the colony. We used radius length in our study because it can be measured accurately *in vivo* and represents forearm length. Forearm length is of interest because the muscles used in gripping attach to the ulna and radius; consequently, longer forearms will provide a greater attachment area for muscles involved in gripping. Data on the forearm muscles and anatomical illustrations for the muscles in *M. murinus* have been published previously (Boettcher *et al.*, 2020). Likewise, we measured external head dimensions because they reflect

the space available for the jaw adductor muscles. A recently published study provides data on the cranial muscles and illustrations of these muscles in the grey mouse lemur (Leonard *et al.*, 2020).

Pedigree construction

We used the same pedigree as in the study by Zablocki-Thomas *et al.* (2019), where all maternities were known and the pedigree was stored in an Excel file with three columns to comply with the AsREML-R statistical analysis (for more details, see supplements in the paper by Zablocki-Thomas *et al.*, 2019). In short, we performed DNA extractions for 256 individuals based on skin tissue available in the tissue sample bank of the colony (Invitrogen PureLink Genomic DNA mini Kit) and amplified it (Qiagen REPLI-g Mini Kit). The genetic analysis of these samples allowed us to determine the paternity for 116 infants, initially having two to four potential fathers, using a microsatellite analysis (Radespiel *et al.*, 2001; Wimmer *et al.*, 2002; Hapke *et al.*, 2003). We assigned paternity by eye using ROX Size Standards, and prospective fathers that did not possess the same alleles found in the offspring were eliminated (Supporting Information).

All maternities were known. There were 246 different mothers in the total 486 mouse lemur dataset, leading to an average of 1.98 infants per mother. The pedigree is very complex because mouse lemurs can breed every year during their lifespan of ~10 years (see Supporting Information, Figure S1 and Table S2). Two to five generations are represented in our dataset.

Statistical analysis

Repeatability

We used the RPTR package and the 'rpt' function to estimate repeatability of performance traits (Nakagawa & Schielzeth, 2010) as a verification of the consistency in performance for a given individual (i.e. the intraclass correlation coefficient). To do so, some individuals were tested several times in independent sessions.

Animal models with AsREML-R

We ran animal models using the AsREML-R software (v.4.0) to conduct restricted maximum likelihood estimations of variance and covariance components. We selected models based on log-likelihood comparisons:

$$t_i = \mu(+\text{age})(+\text{sex}) + a_i + m_i(+pe_i) + \varepsilon_i$$

where μ is the mean of the trait, a_i is the additive genetic effect, m_i is the maternal effect explained by the identity of the mother, pe_i is the permanent environmental effect explained by the identity of the individual, and ε_i is the error for the ith individual. We standardized our variables to a variance of one before analysis by dividing them by the overall standard deviation.

Heritability and evolvability analysis

To assess the relative contribution of genes to the phenotype, we first assessed the heritability of our phenotypic variables with univariate models (asreml-R package), with performances and morphometric traits as response variables. We tested the significance of the fixed effects of age and sex with a conditional Wald test (Wilson *et al.*, 2010). To test for the significance of additive genetic variance, we ran the same model as selected with the pedigree component removed and tested against a χ^2 distribution with one degree of freedom. To estimate the variance of maternal effects, we added maternal identity as a random factor. For bite force and pull strength measures, which were measured multiple times for each individual, we added individual identity to estimate permanent environmental effects (Kruuk, 2004). We calculated the total phenotypic variance as the sum of the variance of all random components (Falconer & Mackay, 1996).
Narrow-sense heritability:

$$h^2 = \frac{Va}{Vp}$$

In our models, Vp is divided in three to four parameters, depending on whether there are several measurements or not:

$$Vp = Va + Vm + Vpe + \varepsilon$$

where Va is the additive genetic variance, Vm is the maternal effect explained by the identity of the mother, and Vpe is the permanent environmental variance explained by the identity of the individual (Kruuk, 2004; Wilson *et al.*, 2010). Likewise, we defined maternal heritability as the ratio of the variance of maternal effect over the total phenotypic variance. We also defined inclusive heritability, which is a measure of the variance transmitted from one generation to another (genetic or non-genetic) over the total phenotypic variance (Danchin & Wagner, 2010) as:

$$IH^2 = \frac{Va + Vm}{Vp}$$

We did not include Vpe in the variance that is transmitted to the next generation, because Vpe accounted for the variance attributable to the individual identity when it was tested several times, as was the case for bite force and pull strength.

To estimate heritabilities and their standard errors, we used the vpredic() function. We reported the coefficient

of variation of additive genetic variance (*CVa*) (Houle, 1992; Hansen *et al.*, 2011) as a measure of evolvability to be able to compare the potential of evolution between tested traits and the 'opportunity of selection' (*Ia*):

$$CVa = 100 \times \sqrt{\frac{Va}{\bar{X}}}$$

$$Ia = \frac{Va}{\bar{X}^2}$$

where *Va* is the additive genetic variance and $\bar{X}$ is the mean for the variable.

Genetic and maternal correlations

We fitted bivariate models to estimate covariance components between traits using a linear mixed modelling approach. We conducted bivariate analyses to test for genetic and maternal correlations among traits, with performance and morphometric traits as response variables. We tested for the significance of two fixed effects, sex and age, by comparing likelihood ratios with and without the effect, with one degree of freedom, first with sex and then with age. We tested for the significance of the maternal effect as a random parameter by comparing the general model with a model in which the covariance attributable to maternal effects (*COVm*) = 0, using a likelihood ratio test with one degree of freedom. When the maternal effect caused problems with convergence, we removed it from the model. We tested for the significance of covariance attributable to additive genetic effects (*COVa*) as previously described, by comparing the general model with a model in which *COVa* = 0 (Wilson *et al.*, 2010) and calculated it as follows:

$$\frac{COVa}{\sqrt{Va.\text{Trait } 1 * Va.\text{Trait } 2}}$$

We calculated maternal correlations, in the same manner but with *COVm* and *Vm*. For bivariate models involving physical performance with repeated measures and other traits without repeated measures, we fixed the residual variance of the non-repeated trait to zero. When we encountered convergence problems, we calculated genetic correlations directly using the corgh() function (Supporting Information, Table S2). When genetic correlations were close to one, standard errors could not be calculated because they exceeded the range of possible values provided by the function.

Data availability

The data underlying this study are available to download from the Dryad Data Repository (Zablocki-Thomas *et al.*, 2021).

RESULTS

Repeatability

Bite force and pull strength were significantly repeatable (Table 1), as demonstrated previously (Thomas *et al.*, 2015, 2016).

Analysis of heritability and evolvability

We found a significant additive genetic variance for bite force, but not for pull strength ($h^2 = 0.23 \pm 0.088$ for bite force and $h^2 = 0.10 \pm 0.096$ for pull strength; Supporting information, Figure S2). Additive genetic variances were significant for the dimensions of the limbs (radius, tarsus and tibia), birth weight and only one head dimension (depth) (Table 1; Supporting Information, Table S3). Maternal effects were significant for most of the tested traits, except for tarsus length and head length, and accounted for substantial proportions of the variance (maternal heritability), sometimes higher than additive genetic variance as was the case for bite force, head width and birth weight.

The opportunity for selection (*Ia*) ranged between 1.19×10^{-4} and 4.23×10^{-4} for head width and radius length, respectively, and between 1.69×10^{-4} and 9.58×10^{-4} for bite force and pull strength, respectively (Table 1). It increased to 1.01×10^{-3} for head depth, and the highest value was found for birth weight, the only life-history trait we included here (4.38×10^{-3}) (Supporting Information, Table S3).

The coefficient of variation of additive genetic variance (*CVa*) ranged from 5.04 to 11.03 for morphological traits and from 7.77 to 9.85 for bite force and pull strength, respectively (Table 1). It increased to 12.89 for head depth, and the highest value was found for birth weight (16.94) (Supporting Information, Table S3).

Genetic and maternal correlations

Bite force exhibited a significant genetic correlation with head depth and a high but not significant genetic correlation with body weight and head width. Pull strength exhibited a high and non-significant genetic correlation with radius length (Table 2).

DISCUSSION

Maternal identity can affect the evolutionary trajectories of offspring phenotypes via maternal effects on single traits or via maternal correlations among suites of traits, yet such effects are seldom estimated for performance traits that capture vital aspects of organismal functional ecology. We applied an animal model to a captive population of *M. murinus*

lemurs to test several predictions related to additive genetic and maternal effects and correlations between two performance traits and the underlying morphology.

Our first prediction, that the heritability of pull strength would be lower than that of bite force, was supported. Bite force showed a moderate and significant heritability, whereas that for pull strength was not significantly different from zero. Both of these estimates are within the range of heritabilities previously reported for performance traits in other organisms, which tend to show moderate (e.g. $h^2 = 0.58$ for maximal crawling speed in *Thamnophis sirtalis* snakes; Garland *et al.*, 1990; $h^2 = 0.30–0.32$ for jump power and jump distance in *Teleogryllus commodus* crickets; Lailvaux *et al.*, 2010) to low heritabilities [e.g. $h^2 = 0.081$ (not significantly different from zero) in the lizard *Zootoca vivipara*; Sorci *et al.*, 1995] depending on the taxa and traits in question. The moderate narrow-sense heritability of bite force in *M. murinus* (0.23) is similar to that reported for locomotor performance in marmots (Blumstein *et al.*, 2010) and for the thermal sensitivity of locomotor performance in a parasitic hymenopteran (Gilchrist, 1996). Despite the common interpretation that low heritabilities are emblematic of traits subject to strong selection, inferring the intensity of selection on a trait based on the magnitude of its heritability alone is perilous, because a low heritability could be a consequence of either low additive genetic variation or high environmental variation (Wilson *et al.*, 2005). However, in the present study the heritabilities of bite force and pull strength are based on the same population and conditioned on similar effects (Table 1) and are thus directly comparable, suggesting that selection might have eroded the additive genetic variation underlying pull strength to a greater extent than that for bite force. Consequently, although bite force is important to fitness in a variety of taxa and contexts, our data support the idea that pull strength is a more important component of fitness in *M. murinus*.

We also expected evolvability (i.e. the capacity to generate heritable and selectable phenotypic variation; Kirschner & Gerhart, 1998) to be relatively high for performance traits (Lailvaux *et al.*, 2010) but rather low for morphometric traits (Houle, 1992). We assessed this through the coefficient of variation of additive genetic variance (*CVa*) and the opportunity of selection (*Ia*). Houle (1992) showed that fitness-related traits, such as fecundity, exhibit high evolvability, whereas morphometric traits, in contrast, generally have lower evolvabilities. Studies containing estimates of the evolvability both of performance traits and of other key fitness-related traits linked to performance are rare, which makes it difficult to put our results in comparative context. However, Lailvaux *et al.* (2010) reported that the evolvability of a locomotor performance trait, jumping ability, estimated as the *CVa*, was similar to that of life-history traits measured from the same breeding design in *Teleogryllus commodus* crickets. In our study, the *CVa* was lower for bite force and pull strength (*CVa* = 7.77 and 9.85, respectively) compared with the study by Lailvaux *et al.* (2010; *CVa* = 16–20), although it is important to note that the differences in functional ecology between these species and traits are vast. With regard to the opportunity for selection, the *Ia* of morphometric traits in *M. murinus* ranged from 1.30×10^{-4} to 1.01×10^{-3}. As a comparison, the opportunity of selection for birth weight was about four times higher than the highest value ($Ia = 4.38 \times 10^{-3}$). The *Ia* was also ~10 times higher for pull strength than for bite force, suggesting a greater potential for evolutionary change in pull strength compared with bite force. In a previous study, pull strength in females was demonstrated to be associated with a higher number of offspring in captivity (Thomas *et al.*, 2016), suggesting that pull strength could be subject to selection in this captive population.

Our second prediction, that maternal effects would be significant for both performance traits, was also supported. We found significant maternal effects for bite force and pull strength, leading to inclusive heritabilities of 0.47 (±0.07) and 0.10 (±0.09), respectively (Table 1), again within the range of heritabilities for locomotor performance traits observed in the literature. These results are consistent with earlier studies that have shown evidence for maternal effects on performance traits in several taxa (e.g. Vanhooydonck *et al.*, 2001). For example, egg size is one of several traits affecting locomotor performance in larval *Bombina orientalis* frogs (Parichy & Kaplan, 1995), and variation in brood ball size, which is under maternal influence, has complex effects on pulling strength in male *Euonticellus intermedius* dung beetles (Reaney & Knell, 2015). Noble *et al.* (2014) quantified additive genetic and maternal effects on two types of locomotor performance in the lizard *Eulampris quoyii* and found significant maternal influence on one (sprint speed) but not the other (endurance), suggesting that maternal effects on performance are not universal. Beyond the performance phenotypes alone, we also found significant maternal effects contributing to the phenotypic variation in head width and birth weight in *M. murinus*. Given the general effects of allometry on performance, in addition to the impact of the juvenile environment on performance development (reviewed by Lailvaux & Husak, 2014), the maternal influence on birth weight constitutes a further avenue for maternal variation to influence performance expression in offspring. Noble *et al.* (2014) also reported similar maternal effects on offspring mass in *Eulampris quoyii*, lending further support to the notion that maternal effects on performance

Table 1. Summary of the univariate analysis on bite force, pull strength, head width and radius length calculated with the AsREML-R animal model (v.4)

			Model selected			
	Mean ± SD	Repeatability [CI] *P*[Permutation]	Fixed effects	Random effects	*Va*	*Vm*
Bite force (N = 401)	35.75 ± 7.52 N	0.329 [0.114, 0.511] P = 0.003	SEX* AGE*	MOTHER*	$2.159 \times 10^{-1} \pm 0.0850$* P = 0.046	$2.293 \times 10^{-1} \pm 0.0728$* $P < 0.001$
Pull strength (N = 399)	10.12 ± 1.72 N	0.346 [0.156, 0.512] P = 0.001	SEX*	MOTHER*	$9.805 \times 10^{-2} \pm 0.0917$ P = 0.25	$4.760211 \times 10^{-7} \pm$ NA* $P < 0.001$
Head width (N = 417)	21.22 ± 0.95 cm	–	SEX* AGE*	MOTHER*	0.054 ± 0.093 P = 0.59	0.11 ± 0.056* P = 0.02734345
Radius length (N = 417)	28.71 ± 1.21 cm	–	AGE*	MOTHER*	0.349 ± 0.116* $P < 0.001$	0.157 ± 0.0603* P = 0.0018

The response variable is presented in the first column. In the third and fourth columns, fixed (SEX and AGE) and random (maternal identity: MOTHER) effects are indicated in capitals. *Va* is the additive genetic variance; *Vm* is the maternal effect explained by the identity of the mother; *Vpe* is the permanent environmental variance explained by the identity of the individual; and *Vr* is the residual variance. We also report the coefficient of variation of the additive genetic variance (*CVa*) and the 'opportunity of selection' (*Ia*).
*Significant result.
NA, not available.

Table 2. Summary of the bivariate analysis on bite force, pull strength, body weight, head width and radius length calculated with the AsREML-R animal model (v.4)

	Model selected				
	Fixed effects	Random effects		*Va*	*Vm*
Bite force and head depth	SEX* AGE*	MOTHER*	Bite force	$2.291 \times 10^{-1} \pm 0.0845$	$2.443 \times 10^{-1} \pm 0.0735$
			Head depth	$2.911 \times 10^{-1} \pm 0.122$	$9.311 \times 10^{-2} \pm 0.0699$
Bite force and head length	SEX* AGE*	MOTHER*	Bite force	$2.221 \times 10^{-1} \pm 0.0847$	$2.355 \times 10^{-1} \pm 0.0729$
			Head length	$2.554 \times 10^{-1} \pm 0.127$	$6.365 \times 10^{-2} \pm 0.0671$
Bite force and head width	SEX* AGE*	MOTHER*	Bite force	$2.27 \times 10^{-1} \pm 0.084$	$2.29 \times 10^{-1} \pm 0.073$
			Head width	$7.52 \times 10^{-2} \pm 0.082$	$9.97 \times 10^{-2} \pm 0.048$
Pull strength and radius length (with corgh)	SEX* AGE*	MOTHER*	Pull strength	0.131 ± 0.0907	0.0111 ± 0.0207
			Radius length	0.425 ± 0.121	0.169 ± 0.06

The response variables are presented in the first column. In the second and third columns, fixed (SEX and AGE) and random (maternal identity: MOTHER) effects are indicated in capitals. Covariances were not calculated when the corgh function was used. *Va* is the additive genetic variance; *Vm* is the maternal effect explained by the identity of the mother; *Vpe* is the permanent environmental variance explained by the identity of the individual; and *Vr* is the residual variance. *COVa*, *COVm* and *COVr* are the covariance attributable to additive genetic effects, maternal effects and residuals, respectively.
*Significant result.
NA, not available.

can operate both directly on performance itself and indirectly, via maternal effects on key determinants of performance.

In addition, the measured morphological variables themselves showed significant heritabilities. Morphometric dimensions are some of the most

Vpe	*Vr*	Narrow-sense heritability (h^2) (estimate ± SE)	Maternal heritability (estimate ± SE)	Inclusive heritability (IH^2)	*CVa*	*Ia*
6.0631×10^{-8} ± NA	4.936×10^{-1} ± 0.0610	0.23 ± 0.088	0.24 ± 0.071	0.47 ± 0.07	7.77	1.69×10^{-4}
2.031×10^{-1} ± 0.117	6.480×10^{-1} ± 0.0869	0.10 ± 0.096	5.01×10^{-7} ± 3.30×10^{-8}	0.10 ± 0.09	9.85	9.58×10^{-4}
–	0.59 ± 0.091	0.072 ± 0.12	0.14 ± 0.071	0.22 ± 0.12	5.04	1.19×10^{-4}
–	0.383 ± 0.0971	0.39 ± 0.12	0.18 ± 0.064	0.57 ± 0.12	11.03	4.23×10^{-4}

Vpe	*Vr*	Narrow-sense heritability (estimate ± SE)	*COVa*	*COVm*	*COVr*	Genetic correlation	Maternal correlation
5.0543×10^{-8} ± NA	4.751×10^{-1} ± 0.0589	0.24 ± 0.086	1.810×10^{-1} ± 0.0729	6.57×10^{-2} ± 0.052	-5.168×10^{-2} ± 0.058	0.70 ± 0.26 *P* = 0.01*	0.44 ± 0.32 *P* = 0.23
–	5.978×10^{-1} ± 0.101	0.30 ± 0.12					
5.0543×10^{-8} ± NA	4.856×10^{-1} ± 0.0602	0.23 ± 0.087	2.651×10^{-2} ± 0.0701	8.86×10^{-2} ± 0.050	-3.662×10^{-2} ± 0.0613	0.11 ± 0.29 *P* = 0.69	0.73 ± 0.49 *P* = 0.085
–	–	0.25 ± 0.12					
5.054×10^{-8} ± NA	4.85×10^{-1} ± 0.059	0.24 ± 0.085	8.27×10^{-2} ± 0.06	4.48×10^{-2} ± 0.043	-2.63×10^{-2} ± 0.05	0.63 ± 0.45 *P* = 0.14	0.29 ± 0.27 *P* = 0.30
–	4.94×10^{-1} ± 0.077	0.11 ± 0.12					
0.153 ± 0.113	0.657 ± 0.0879	0.14 ± 0.09	–	–	–	0.41 ± 0.26 *P* = 0.15	0.99 ± NA *P* = 0.28
–	0.306 ± 0.95	0.47 ± 0.12					

studied traits in quantitative genetic studies and, generally, show significant additive genetic variances and medium to high narrow-sense heritablities. For example, in snakes (h^2 = 0.41 for ventricle mass; Garland *et al.*, 1990) and in Darwin finches (h^2 = 0.76 for averaged morphological data; Boag, 1983) high

heritabilities were found using parent–offspring regressions and sib comparisons. In the house sparrow, results were more variable ($0.12 < h^2 < 0.68$; Jensen *et al.*, 2003) and appeared to be sex dependent. Likewise, in sticklebacks, heritabilities were also highly variable ($h^2 = 0.67$, 0.92 and 0.34 for body length, body shape and relative spine length, respectively), depending on the trait (Dingemanse *et al.*, 2009). Compared with the literature, our heritability estimates for morphological traits are low in addition to being non-significant, and ranged from very low ($h^2 = 0.07$) to moderate ($h^2 = 0.24$ for radius length, tibia length and tarsus length) (see Supporting Information, Table S3). Notably, values for *Va* were always significant for limb dimensions but not for head dimensions in our mouse lemur colony, resulting in higher narrow-sense heritabilities for limb dimensions than for head dimensions. This suggests that head dimensions might be under stronger selection than limb dimensions, probably as a consequence of strong selection on bite force (Arnold, 1983). Future studies on both wild and captive populations of *M. murinus* should consider explicitly the form and intensity of selection acting on bite force and associated head morphology through the utility of such traits for securing access to food resources (Génin, 2004; Viguier, 2004) and in intersexual interactions (Eberle & Kappeler, 2004).

Our third prediction, that we would find genetic and maternal correlations among performance and the underlying morphological traits enabling performance, was also supported, but the correlations lacked significance, except in one occurrence between bite force and head depth (Table 2). Bite force and head dimensions were also correlated (high correlations but only one tendency) with regard to maternal identity, indicating that females that produced infants with larger heads also produced infants with higher bite forces. Although these results suggest an important role for the underlying musculature in driving variation in head dimensions and, consequently, bite force (see also Fabre *et al.*, 2018; Leonard *et al.*, 2020), the direct mechanical link between muscle size and architecture and bite force in *M. murinus* remains to be tested. Previous studies have reported strong maternal correlations among ecologically relevant phenotypes even in the face of low heritabilities; for example, Taylor *et al.* (2012) showed that activity and aggression both exhibited substantial genetic and maternal correlations in red squirrels, illustrating the importance of both sources of variation in linking groups of covarying traits. Our data suggest that similar relationships exist between each performance trait and the underlying morphology, although the magnitudes of those relationships varied (Table 2). Genetic correlations between morphometric traits are generally close to one and positive (Jensen *et al.* 2003), but also sometimes negative (Roff, 1996). Although we detected few significant genetic correlations between morphological traits in our dataset (Supporting Information, Table S4), head width and head length were exceptions. We note, however, that we could not take maternal effects into account in these models, which could have led to inflated estimations.

Although our study is the first to estimate not only maternal and genetic heritabilities, but also maternal and genetic correlations for performance traits, our results come with an important caveat. The grey mouse lemur has a promiscuous mating system, even in captivity, which means that three to four potential fathers mated with the mother of each individual. Importantly for the present study, the housing regimen precluded multiple matings for captive females, which meant that we were unable to estimate additive maternal effects (i.e. the component of phenotypic variation that covaries with offspring genotype owing to relatedness between mother and offspring; Wolf & Wade, 2016). An ideal examination of maternal effects on performance evolution would consider this additive maternal effect, in addition to the additive maternal correlations among traits of interest (Wilson *et al.*, 2005).

Our results represent a clear advance in our understanding of performance-related trait evolution and are an important step towards comprehension of the factors affecting the evolutionary trajectories of functional traits with clear links to fitness. Indeed, they demonstrate the contribution of genetic and maternal effects in the transmission of, and correlations between, phenotypes involved in similar functions (van Oers *et al.*, 2005). Additional studies to evaluate the heritability of performance-related traits for other taxa and the role of maternal effects are needed to gain a better understanding of the importance of these types of traits in an evolutionary context.

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the study. P.Z.-T., A.H. and E.P. collected data. P.Z.-T., A.H. and S.L. analysed the data and interpreted the results. P.Z.-T., S.L. and A.H. wrote the manuscript with the help of F.A. and E.P. The authors declare no conflicts of interest.

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SUPPORTING INFORMATION

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

FigureS1. Visual comparison of the variance components for bite force, pull strength, head width and radius length displayed in Table 1. Variance is displayed as white circles, and the error bars represent the standard deviation.

Figure S2. Representation of the pedigree for the 486 individuals present in the study. This figure was drawn with the freely available 'PEDIGREE VIEWER' software. Individual identities are represented in white, with maternal links in yellow and paternal links in red. Each line represents a generation; however, this does not mean that individuals on the same line are the same age, only that the software is minimizing the number of lines.

Table S1. Summary of the univariate analysis on head depth, head length, tibia length, tarsus length and birth weight calculated with the ASREML-R animal model (v.4). The response variable is presented in the first column. In the third and fourth columns, fixed (SEX and AGE) and random (maternal identity: MOTHER) effects are indicated in capitals. *Va* is the additive genetic variance; *Vm* is the maternal effect explained by the identity of the mother; and *Vr* is the residual variance. We also report the coefficient of variation of the additive genetic variance (*CVa*) and the 'opportunity of selection' (*Ia*).

Table S2. Summary of the bivariate analysis on head and limb dimensions, head width and radius length calculated with the ASREML-R animal model (v.4). The response variables are presented in the first column. In the second and third columns, fixed (SEX and AGE) and random (maternal identity: MOTHER) effects are indicated in capitals. *Va* is the additive genetic variance; *Vm* is the maternal effect explained by the identity of the mother; *Vpe* is the permanent environmental variance explained by the identity of the individual; and *Vr* is the residual variance. *COVa*, *COVm* and *COVr* are the covariance attributable to additive genetic effects, maternal effects and residuals, respectively.

TableS3. Microsatellites used for paternity assignments.

Table S4. Pedigree information for the dataset with the 486 individuals. This pedigree was used to draw the pedigree with 'PEDIGREE VIEWER' and was not suitable for the heritability analysis, which includes 'founder individuals'.