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### Citation for published version:

Gauzere, J, Pemberton, J, Morris, S, Morris, A, Kruuk, L & Walling, C 2020, 'The genetic architecture of maternal effects across ontogeny in the red deer', *Evolution: International Journal of Organic Evolution*, vol. 74, no. 7, pp. 1378-1391. <https://doi.org/10.1111/evo.14000>

### Digital Object Identifier (DOI):

[10.1111/evo.14000](https://doi.org/10.1111/evo.14000)

### Link:

[Link to publication record in Edinburgh Research Explorer](#)

### Document Version:

Publisher's PDF, also known as Version of record

### Published In:

Evolution: International Journal of Organic Evolution

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# The genetic architecture of maternal effects across ontogeny in the red deer

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Received February 18, 2020

Accepted May 4, 2020

Maternal effects, either environmental or genetic in origin, are an underappreciated source of phenotypic variance in natural populations. Maternal genetic effects have the potential to constrain or enhance the evolution of offspring traits depending on their magnitude and their genetic correlation with direct genetic effects. We estimated the maternal effect variance and its genetic component for 12 traits expressed over the life history in a pedigreed population of wild red deer (morphology, survival/longevity, breeding success). We only found support for maternal genetic effect variance in the two neonatal morphological traits: birth weight ( $h^2_{Mg} = 0.31$ ) and birth leg length ( $h^2_{Mg} = 0.17$ ). For these two traits, the genetic correlation between maternal and direct additive effects was not significantly different from zero, indicating no constraint to evolution from genetic architecture. In contrast, variance in maternal genetic effects enhanced the additive genetic variance available to respond to natural selection. Maternal effect variance was negligible for late-life traits. We found no evidence for sex differences in either the direct or maternal genetic architecture of offspring traits. Our results suggest that maternal genetic effect variance declines over the lifetime, but also that this additional heritable genetic variation may facilitate evolutionary responses of early-life traits.

**KEY WORDS:** *Cervus elaphus*, cross-sex correlation, genetic constraint, life-history traits, maternal genetic effects, total heritability.

Maternal effects, either environmental or genetic in origin, are widespread in animals and have important consequences for offspring development and fitness (reviewed in Mousseau and Fox 1998; Moore et al. 2019). However, the strength and nature of these effects can vary between different traits. In particular, because maternal effects likely arise from gestation and maternal care, a strong expectation is that these effects are more prevalent for traits expressed early in life. A recent meta-analysis has confirmed that maternal effects explained more phenotypic variance in juvenile traits than in adult morphological and life-history traits (Moore et al. 2019). Yet, within-population studies looking in detail at the ontogeny of maternal effects are rare, especially in wild populations (but see Wilson and Réale 2006 in domestic mammals; Lindholm et al. 2006; White and Wilson 2019 in captive bred fish populations; or Cheverud et al. 1983 in laboratory mice).

Additionally, we know that maternal investment can vary with offspring sex. For instance, in many sexually selected mammal species, males are typically born heavier and have a higher growth rate than females and usually require more maternal care (Clutton-Brock 2016). Life-history theory also suggests that the sex with the greater variance in reproductive success, usually the males in polygynous species, should be more sensitive to variation in early-life conditions (Trivers and Willard 1973). For these reasons, we expect maternal effects to vary with offspring sex for species showing pronounced sexual dimorphism and different reproductive roles (Clutton-Brock et al. 1985; Kruuk et al. 2008). Published studies testing for such sex-specific maternal effects are limited, and have mostly found no differences between the sexes (e.g., Lindholm et al. 2006; Moore et al. 2019; but see Kruuk et al. 2015). More generally, there is a need to better identify the factors affecting the expression and importance of

maternal effects within wild animal populations (Moore et al. 2019).

Despite a substantial literature on the existence of maternal effects and their ecological consequences, we still have a poor insight into the evolutionary consequences of maternal effects in the wild. To assess the evolutionary consequences of maternal effects, we need to partition the sources of phenotypic variation in maternal performance, that is, in the maternal traits underlying maternal effects, and particularly their genetic determinism (Räsänen and Kruuk 2007). Quantitative genetics offers a useful framework to investigate the genetic basis of maternal effects. Indeed, maternal effects can most easily be modeled within a framework that does not require us to identify the actual maternal traits explaining maternal performance for a given offspring trait, but rather treats them as a general feature of individual mothers and estimates the variance between mothers (Willham 1972—in contrast to a “trait-based” approach that requires identification of the relevant trait(s) in the mother; Kirkpatrick and Lande 1989). This strategy, also called “variance partitioning” approach, only requires phenotypic measurement of offspring trait(s), measures on multiple offspring per mother, and a pedigreed population over several generations (Kruuk 2004).

Using the variance partitioning approach, several studies of wild animals have now shown substantial variance between mothers in their impact on offspring phenotype, but separating this variance into its genetic and environmental components is more challenging (Räsänen and Kruuk 2007). In livestock, the importance of heritable maternal effects has been acknowledged and fully integrated into breeding programs for decades (e.g., Meyer 1992; Miller and Wilton 1999; Wilson and Réale 2006). Over the last 20 years, some long-term individual-based studies have managed to quantify maternal genetic effect variance for different offspring traits and species in wild animal populations (e.g., red squirrels, McAdam et al. 2002; McFarlane et al. 2015; Soay sheep, Wilson et al. 2005; Béréños et al. 2014; roe deer, Quéméré et al. 2018; red deer, Kruuk and Hadfield 2007), but such studies are still relatively rare.

The genetic architecture of maternal effects has the potential to enhance or constrain the response of offspring traits to selection (Willham 1972; Kirkpatrick and Lande 1989; Räsänen and Kruuk 2007). Let us assume that a focal offspring trait  $Y$  is affected by both direct additive genetic effects and indirect maternal effects. Following the Willham model, the decomposition of the genetic value of this trait has three components: two additive genetic values, one for the focal individual and one for the maternal performance for the trait, and one component of correlation between these genetic effects (Willham 1963). Consequently, the rate and direction of evolution of  $Y$  depends on the inheritance of maternal performance for that trait, even if the trait itself is not under selection (Kirkpatrick and Lande 1989).

More specifically, first, the additional heritable genetic variation conferred by variance in maternal genetic effects can increase the “total heritability” of the trait  $Y$  (Willham 1972) and its evolutionary potential, that is, the amount of additive genetic variation available for selection (Falconer and MacKay 1996). Second, assuming only directional selection on the offspring trait, a negative covariation between direct additive and indirect maternal genetic effects,  $\sigma_{AM_g}$ , should constrain the evolution of  $Y$  (Kirkpatrick and Lande 1989; Räsänen and Kruuk 2007; Hadfield 2012). In contrast, a positive  $\sigma_{AM_g}$  should facilitate its response to selection. The ultimate impact of maternal effects on evolutionary dynamics is thus heavily dependent on the direction and magnitude of the direct-maternal genetic covariance. However, to date, the number of studies estimating the covariation between additive and maternal genetic effects in wild animals is insufficient to determine the general effect of  $\sigma_{AM_g}$  on the evolutionary potential of traits in wild populations (positive for growth in red squirrels, McAdam et al. 2002; nonsignificant or negative depending on the trait in Soay sheep, Wilson et al. 2005; see also Wilson and Réale 2006 for estimates in livestock; or Riska et al. 1985 in laboratory mouse lines).

In the present study, we aimed to provide a comprehensive analysis of the impact of maternal genetic effects on the evolutionary potential of offspring traits for a wild mammal species, the red deer (*Cervus elaphus*). We used the long-term individual-based study of the red deer population of the Isle of Rum National Nature Reserve (Scotland). This species has extended maternal care, and sex differences in juvenile growth and life history (Clutton-Brock et al. 1982, 1987; Kruuk et al. 1999). Previous studies have estimated variance due to maternal effects in this population (Kruuk et al. 2000; Kruuk and Hadfield 2007; Walling et al. 2014; Logan et al. 2016; Bonnet et al. 2019). They found substantial maternal effect variance for birth weight, explaining more than 20 % of the phenotypic variance (Kruuk et al. 2000; Kruuk and Hadfield 2007), and moderate maternal effect variance for some life-history traits, such as survival to breeding age, age at first reproduction and annual breeding success, with 3–17% of the phenotypic variation explained (Kruuk et al. 2000; Walling et al. 2014). However, no significant variance in maternal effects was found for other life-history traits (e.g., male lifetime breeding success and adult longevity; Kruuk et al. 2000; Walling et al. 2014), or adult morphological traits (leg length, jaw length, endocranial volume; Kruuk et al. 2000; Logan et al. 2016). Among these studies, only one decomposed maternal effects into genetic and environmental components, and estimated larger maternal genetic than maternal environmental effects for birth weight (Kruuk and Hadfield 2007).

In a summary, previous studies have highlighted that maternal effects are an important source of phenotypic variation for some traits in the study population, but they did not investigate

the ontogeny of maternal effects, or test for sex-specific maternal effects. More importantly, the consequences of maternal genetic effects for the evolutionary potential of traits in this population are still unexplored, as no study has estimated the genetic covariance between additive and indirect maternal genetic effects and the total heritability of traits. Here, we address the following questions: (1) How do maternal genetic effects change for traits expressed at different life stages? (2) Do maternal effects vary with offspring sex? (3) Does the covariation between additive and maternal genetic effects constrain or enhance the potential evolutionary response of offspring traits to selection? Compared with relevant previous studies on the deer population (Kruuk et al. 2000; Kruuk and Hadfield 2007; Walling et al. 2014), we used at least 10 years' more data and a more complete pedigree to provide a systematic picture of the genetic architecture of maternal effects across the ontogeny of red deer.

## Materials and Methods

### STUDY SPECIES AND POPULATION

The red deer is a uniparous, polygynous mammal, for which maternal siblings born in different years can share maternal effects. Female red deer are philopatric, meaning that female relatives tend to live together and in their natal range. Adult females provide substantial maternal investment to their offspring, with an average gestation period of about 34 weeks, and a lactation period of 6 months, which can continue into a calf's second year if a mother does not conceive again the following year (Clutton-Brock et al. 1982). This maternal investment is likely a critical determinant of the size at which calves enter the winter, and consequently of their chances of survival (Clutton-Brock et al. 1982). Observations also suggest that maternal investment is higher in male calves than in females; males have heavier weight at birth, more frequent suckling, and higher juvenile growth rate (Clutton-Brock et al. 1982), although there are no differences in gestation period (Clements et al. 2011). Males usually leave their natal range around the age of 2 years, while young and old females associate in loose matrilineal groups (Clutton-Brock et al. 1982; Stopher et al. 2012).

Here, we use the red deer population of the Isle of Rum as a study system to investigate the importance of maternal effects in the wild. This population, located in the Inner Hebrides (Scotland; 57°03'N, 06°21'W), has been intensively studied since 1972, with about 5000 individuals tagged and monitored throughout their life. Regular censusing, close observations during the rut and calving seasons, and mortality searching allow us to retrieve complete life-history data for most of the individuals living within the 12 km<sup>2</sup> study area. The calving period generally extends over 6 weeks, from mid-May to late June. Most calves born within the study area are caught soon after birth (typically within

24 h), sampled for genetic analysis (ear punch), and measured (details below). Immigrant individuals (mostly males) were also sampled for genetic analysis from postmortem tissue samples and cast antlers. All individuals found dead are measured for further morphological traits. Since 1973 there has been no culling within the study area, but individuals born in the study area are occasionally shot as part of management culls if they range elsewhere on the island.

### RELATEDNESS INFORMATION

As far as possible all sampled individuals within the study area were genotyped at 38k polymorphic SNPs spaced throughout the genome. A subset of 440 SNPs and the R-package SEQUOIA were used to construct a pedigree for the population (see Huisman 2017 for more details). The main advantage of this method is that it allows efficient assignment of parentage from SNP data, and accurate determination of more distant relationships (second- and third-degree relationships) even if parents are not sampled. Where SNP genotyping failed (for 8% of sampled individuals), we used maternal links from observation and paternal links previously found using 15 microsatellite loci and paternity inference methods implemented in MasterBayes (Hadfield et al. 2006) and COLONY2 (Wang and Santure 2009; see Walling et al. 2010 for more details). In total, the reconstructed pedigree had 4429 mother-offspring links, 2995 father-offspring links and the pairwise relatedness matrix ( $A$ ) derived from the pedigree had 6,118,033 nonnull elements (representing 43% of the elements in  $A$ ; see also Part S1 in Supporting Information). Huisman (2017) showed that this SNP-based SEQUOIA pedigree was more comparable to the actual pairwise genomic relatedness than the microsatellite-based pedigree used previously in the red deer population (e.g., Kruuk et al. 2000; Walling et al. 2014).

### QUANTITATIVE TRAITS

We analyzed a range of morphological and life-history traits, expressed at different ages, to investigate changes in the magnitude of maternal effects across life. We included individuals resident in the study area with known maternal identity in all cohorts up to 2017 (sample sizes for each trait are provided in Table 1). Morphological traits were measured on calves soon after birth and on adults once found dead (via measurement of clean bones). Life-history traits were determined from the detailed monitoring of births and deaths within the study area, as well as from parentage assignments. For the survival and longevity analyses, we only included individuals that died from natural causes, that is, we removed culled individuals, which constituted 15% of monitored individuals. See Table 1 for summary statistics on the traits studied.

**Table 1.** Description and summary statistics for the traits analyzed in this study.

Trait	$n_O$	$n_M$	Average	CV	Unit	Fixed effects
<b>Neonatal traits</b>						
Birth weight <sup>a</sup>	2421	678	6.93	0.202	kg	Sex + AgeHrs + PopDens + MatAge + MatAge <sup>2</sup> + ReproStatus + Measurer + Region
Birth leg length <sup>a</sup>	1596	521	281.34	0.065	mm	Sex + AgeHrs + PopDens + MatAge + MatAge <sup>2</sup> + ReproStatus + Measurer + Region
Neonatal survival	3553	832	0.82	0.47	rate	Sex + ReproStatus + PopDens + MatAge + MatAge <sup>2</sup> + Region
<b>Early-life traits</b>						
Survival age 1	2726	745	0.70	0.680	rate	Sex + ReproStatus + PopDens + MatAge + MatAge <sup>2</sup> + Region
Survival age 2	2544	710	0.59	0.882	rate	Sex + ReproStatus + PopDens + MatAge + MatAge <sup>2</sup> + Region
AFR ♀	729	408	4.07	0.191	years	Region
<b>Late-life traits</b>						
ABS ♀	671	375	0.61	0.80	calves	ReproStatus + Age + Age <sup>2</sup> + Region
ABS ♂	592	344	0.45	2.73	calves	Age + Age <sup>2</sup> + Region
Adult longevity	697	330	10.95	0.343	years	Sex + Region
Jaw length	839	421	263.29	0.053	mm	Sex + AgeDeath + Measurer + Region
Endocranial volume	568	330	338.04	0.089	mL	Sex + AgeDeath + Measurer + Region
Leg length	440	259	190.31	0.046	mm	Sex + AgeDeath + Measurer + Region

Note:  $n_O$  provides the number of offspring trait values,  $n_M$  the number of mothers, *average* is the average phenotypic value in the population, and *CV* the coefficient of variation of each trait, with  $CV = \text{standard deviation}/\text{mean}$ . We also provide the list of the fixed effects used to fit model (1), with *AgeHrs* the age at capture, *PopDens* the number of females sharing the same region during spring, *MatAge* the maternal age, *ReproStatus* = {"naive," "milk," "yield," "winter yield," "summer yield"} *Region* = {"Kilmory," "Shamhnan Insir," "Intermediate area," "Mid glen," "North glen," "South glen"} *AgeDeath* the age at death, and "Measurer" a measurer effect for the morphological traits. Note that the spatial/population information, *Region* and *PopDens* are measured based on the mother's location for the neonatal and early-life traits, and based on the focal individual location for the late-life traits. AFR = age at first reproduction; ABS = annual breeding success; ABS was analyzed separately for males and females.

<sup>a</sup>The summary statistics are provided for the weight and leg length measured at capture.

### Neonatal morphological traits

We analyzed the weight (kg) and leg length (from the back of the hock to the back of the hoof; mm) of calves caught within 7 days of birth and born before August 1 (following Huisman et al. 2016). All models for juvenile morphological traits accounted for the effect of calf sex, age at capture (hours) and measurer (a 24-level factor). We also considered some effects related to maternal condition, namely maternal age (in years; linear and quadratic effects) and maternal reproductive status, which characterizes a female's breeding status in the previous year as to whether she (i) calved and the calf survived to at least May 1, the year after birth (milk), (ii) calved and the calf died during the winter after birth, between October 1 and May 1 (winter yield), (iii) calved and the calf died during the summer, before October 1 (summer yield), (iv) female did not calve (true yield), and (v) had never calved (naïve) (following Stopher et al. 2012; Huisman et al. 2016). Note that in previous studies, a calf's birth date was frequently used as a fixed effect to analyze neonatal traits (e.g., Huisman et al.

2016), but birth date was not included here as it is a maternal trait and including it could lead to an underestimate of the strength of maternal effects for the focal traits.

### Adult morphological traits

We analyzed three different morphological traits measured on all dead mature individuals (i.e., aged 3 years or more) found in the study area: (1) back leg length (mm), measured as the distance between proximal and distal metatarsal canal openings on the cannon bone, an approximation of its length; (2) endocranial volume (mL; see Logan et al. 2016 for measurement details); (3) length of the jaw bone (mm), measured from the back of the jaw to the base of the first incisor. To account for the fact that these traits were measured on adults of different ages and some may still have been growing, the age at death was included in models of postmortem morphological traits. We also considered an effect of the sex and measurer (a five-level factor).



### Life-history traits

**Neonatal and juvenile survival:** Most mortality occurs in the first 2 years of life. We defined neonatal survival as survival from the date of birth to September 30 in the year of birth. During this period, mortality is due to a variety of causes including being stillborn, predation by eagles, and accidents. Further mortality occurs during the two first winters after birth and depends on calves' body condition before experiencing harsh conditions. We analyzed the first-year winter survival of the calves from October 1 in the year of birth to the following May 1. Survival to the age of 2 years was similarly defined as survival of the 19-month period from October 1 of the year of birth to May 1 of the second year following birth. Models for neonatal and juvenile survival accounted for the effect of calf sex, maternal reproductive status, and maternal age (i.e., similar to neonatal morphological traits).

**Age at first reproduction:** For females, we analyzed the age at which an individual first calved (in years). Females that never calved, mostly because they died before the age of maturity, had no information for this trait and were excluded. Females that calved at least once and were later shot were included. We did not estimate this trait for males, as even males that consistently breed in the study area may have had a small number of matings outside the study area before establishing a regular breeding site.

**Adult annual breeding success:** For both sexes, we estimated the number of calves produced each year from maturity (at 3 years) to death. For females this number can only be 0 or 1, while for males it ranges from 0 to 14 per year. Females were included only if they survived up to the age of 6 (following Walling et al. 2014) and males if they rutted within the study area in a given year. Because of their inherently different distributions, this trait was analyzed separately for males and females. Models for annual breeding success accounted for the effect of individual age (in years; linear and quadratic effects) and, for females only, we included the effect of the female's breeding status in the previous year.

**Longevity:** We estimated the age at death (in years) of all individuals with known birth and death years that reached the age of 3 and died from natural causes. Models for longevity accounted for the effect of individual sex.

Because our data came from the long-term study on the Rum red deer population, part of these data have already been used in previously published studies. Compared with previous works investigating maternal effect variance in the study population (Kruuk et al. 2000; Kruuk and Hadfield 2007; Walling et al. 2014), we had at least 10 years of additional data. For juvenile traits, this substantially increased the amount of data analyzed; for example, for birth weight, we analyzed 45% more individuals than Kruuk and Hadfield (2007). For a recently measured adult trait, adult endocranial volume, we analyzed a very similar dataset to the one published by Logan et al. (2016).

### VARIANCE PARTITIONING APPROACH

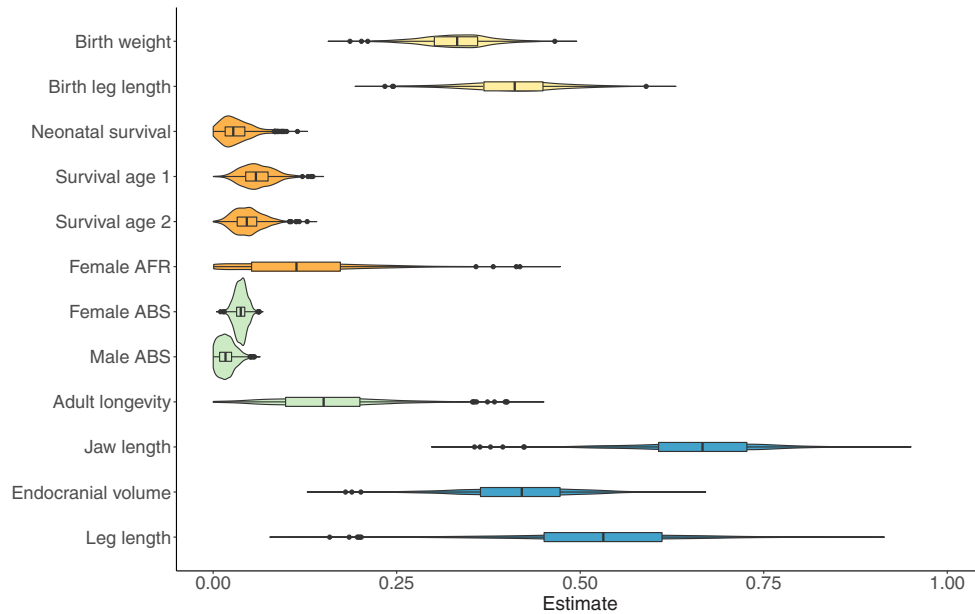
We used a univariate mixed model framework to partition the total phenotypic variance in each trait into the sum of fixed and random effects. More specifically, we considered that four random effects can affect the phenotypic variance: the effects of the individual's additive genetic value (contained in the vector  $a$ ), maternal additive genetic value ( $m_g$ ), maternal environmental value ( $m_e$ ), and the effect of the cohort ( $c$ ; i.e., its year of birth). For the traits with repeated measures, we also considered an effect of the year of measurement ( $yr$ ) and a permanent environmental effect on individual's phenotype ( $pe$ ). We did not consider dominance genetic effects, as full-sibs constituted a very small proportion of the relatives in the pedigreed population (1% in comparison to half-sibs) and dominance variance is generally low for quantitative traits (Hill et al. 2008). The general matrix form of this model is

$$y = Xb + Z_1a + Z_2m_g + Z_3m_e + Z_4c + Z_5yr + Z_6pe + e \quad (1)$$

with  $y$  the vector of phenotypic observations,  $b$  the vector of fixed effects fitted in the model, and  $e$  the vector of residual error. The design matrices  $X$  and  $Z$  link the individual observations to the relevant fixed and random effects. Each random effect is distributed following a normal distribution with  $a \sim N(0, A\sigma_A^2)$ ,  $m_g \sim N(0, A\sigma_{Mg}^2)$ ,  $m_e \sim N(0, I\sigma_{Me}^2)$ ,  $c \sim N(0, I\sigma_C^2)$ ,  $yr \sim N(0, I\sigma_{YR}^2)$ ,  $pe \sim N(0, I\sigma_{PE}^2)$ , and  $e \sim N(0, I\sigma_R^2)$ .  $A$  is the genetic relatedness matrix derived from the pedigree, and  $I$  the identity matrix.

We used an animal model, a specific mixed-effect model that uses pedigree information to dissociate both the direct additive genetic effects ( $a$ ) and the maternal genetic effects ( $m_g$ ) from the other effects specified in model (1). Note that in this model, the maternal environmental effects ( $m_e$ ) were modeled as permanent differences between mothers, with no genetic basis. We compared the three covariance matrices used to estimate  $a$ ,  $m_g$ , and  $m_e$ , which are  $A$ , the matrix of pairwise genetic relatedness between calves;  $A_m$ , the matrix of pairwise genetic relatedness between mothers; and  $PME$ , the matrix of maternal identities, respectively. We found that the matrices were partly correlated, with  $cor(A, PME) = 0.27$ ,  $cor(A, A_m) = 0.60$ , and  $cor(A_m, PME) = 0.59$ . Yet, there was still a substantial proportion of uncorrelated variance that should allow us to decompose the different sources of variation in offspring traits (see more details in Part S1).

We first ran model (1) without considering covariation between additive and maternal genetic values (i.e.,  $\sigma_{AM_g} = 0$ ) to estimate the additive genetic variance  $\sigma_A^2$ , maternal genetic variance  $\sigma_{Mg}^2$ , maternal environmental variance  $\sigma_{Me}^2$ , among cohort variance  $\sigma_C^2$  (as well as the year of measurement variance  $\sigma_{YR}^2$  and permanent environment variance  $\sigma_{PE}^2$  for repeated measures



**Figure 1.** Total heritability ( $h^2_{Tot}$ ) of the study morphological and life-history traits represented as probability density plot and box plot of the posterior distributions. Note that for all traits, except birth weight and leg length, we found nonsignificant maternal genetic effect variance and therefore  $h^2_{Tot} = h^2$ . Adult morphological traits (blue) exhibited higher heritabilities than neonatal morphological traits (yellow), and morphological traits overall exhibited higher heritabilities than life-history traits (orange: early-life traits; green: late-life traits). AFR= age at first reproduction; ABS= annual breeding success.

only) and total phenotypic variance  $\sigma_p^2$  of the studied traits. We also fitted a model for “total” maternal effects ( $m$ ), that included maternal genetic and environmental effects combined, that is,  $m = m_g + m_e$  and with  $m \sim N(0, I_{de} \sigma_M^2)$ . This model allowed us to estimate the total contribution of maternal effect variance (not partitioned into genetic and environmental components) to the phenotypic variance in offspring traits, called  $m^2$ , and provided results more comparable to former studies on this population (Kruuk et al. 2000; Kruuk and Hadfield 2007; Walling et al. 2014; Logan et al. 2016; Bonnet et al. 2019; more details in Parts S2 and S3).

Spatial and population effects were modeled as fixed effects in model (1). The birth and lifetime locations were determined based on census data, and each individual could be categorized as living in one of six regions of the study area: Kilmory, Shamnan Insir, Intermedite area, Mid glen, North glen, or South glen (following Huisman et al. 2016). For neonatal and early-life traits, models also accounted for the effect of population density, as the number of adult females in each region during spring. For neonatal and early-life traits, the spatial and population information were based on the mother’s location; for late-life traits they were based on the focal individual’s lifetime location (or individuals’ location each year in adult life for annual breeding success).

**GENETIC COVARIANCES AND SEX-SPECIFIC MODELS**

For the traits for which there was evidence for significant maternal genetic effect variance, we investigated further how these indirect genetic effects could impact the evolutionary potential of offspring traits, and sex-specific differences in maternal performance. We first estimated the genetic covariation between additive and maternal genetic effects, using model (1) and considering a variance-covariance matrix with the following structure:  $\begin{bmatrix} \sigma_A^2 & \sigma_{AM_g} \\ \sigma_{AM_g} & \sigma_{M_g}^2 \end{bmatrix}$ . A negative  $\sigma_{AM_g}$  would reduce the evolutionary potential of a trait and, assuming only directional selection on the focal offspring trait, suggest some evolutionary constraint (Hadfield 2012).

For these traits, we also fitted sex-specific models to estimate separate variance components in males and females ( $\sigma_{\sigma}^2$ ,  $\sigma_{\phi}^2$ ), and the covariance between the genetic effects (direct or indirect) expressed by the two sexes ( $\sigma_{\sigma\phi}$ ). This model follows the same general form as model (1), but it includes sex-specific fixed effects and the general structure of the variance-covariance matrix for each random effect follows:  $\begin{bmatrix} \sigma_{\sigma}^2 & \sigma_{\sigma\phi} \\ \sigma_{\sigma\phi} & \sigma_{\phi}^2 \end{bmatrix}$ , with  $\sigma$  = male,  $\phi$  = female. A correlation between the genetic effects expressed in males and females different from one ( $\frac{\sigma_{\sigma\phi}}{\sigma_{\sigma}\sigma_{\phi}} < 1$ ) would potentially result in independent evolutionary trajectories for the

trait between the sexes. Based on previous evidence (Kruuk et al. 2000), we also tested for sex-specific total maternal effects ( $m$ ) for adult traits (see Part S5 for more details).

### FITTING PROCEDURE AND OUTPUT

We fitted model (1), and its extended versions (covariance structure, sex-specific model), using the Markov Chain Monte Carlo (MCMC) method implemented in the R-package MCMCglmm (Hadfield 2010). For all traits, the MCMC parameterization was defined such that the lag between two sampled iterations produced a low autocorrelation of the chain (autocorrelation  $<0.15$ ) and the total number of sampled iterations equals 1000 (equivalent to a number of iterations = 300,000 and thinning interval = 250 for most univariate models). A burn-in of 50,000 iterations was sufficient to reach convergence. We used noninformative parameter expanded priors for all analyses, so that the posterior distributions of our models were little influenced by a priori expectations about the parameter distributions. The error distribution used to analyze each trait was chosen based on the nature of the trait (categorical vs. continuous) and the adequacy of its fit with model assumptions. We thus chose Gaussian error models to analyze all morphological traits, as well as age at first reproduction and adult longevity. We chose a threshold model to analyze neonatal and juvenile survival as well as female annual breeding success (with residual variance fixed to 1), and a Poisson model to analyze male annual breeding success. Note that to date, maternal effects for life-history traits in wild populations have rarely been studied using these more appropriate error models rather than assuming Gaussian error distribution (but see McFarlane et al. 2015).

The significance of variance components was visually assessed from posterior distributions and the significance of covariance components was evaluated based on whether the 95% credible intervals of the estimate overlapped zero. The contribution of each random effect to the determinism of the phenotypic trait was evaluated as the proportion of variance explained by a given effect, estimated as its variance component divided by the total phenotypic variance. For the additive genetic variance, this proportion is an important evolutionary parameter known as the narrow-sense heritability:  $h^2 = \frac{\sigma_A^2}{\sigma_P^2}$ . We also calculated the “total heritability” of the traits, considering that  $\sigma_{M_g}^2$  and  $\sigma_{AM_g}$  also determine the amount of additive genetic variation available for selection (Dickerson 1947):  $h_{Tot}^2 = \frac{\sigma_A^2 + 1/2 \sigma_{M_g}^2 + 3/2 \sigma_{AM_g}}{V_P}$ , and neglecting the effect of  $\sigma_{AM_g}$ :  $h_{Tcov0}^2 = \frac{\sigma_A^2 + 1/2 \sigma_{M_g}^2}{V_P}$ . The impact of the covariance component  $\sigma_{AM_g}$  on the “total heritability” was thus evaluated by comparing  $h_{Tot}^2$  and  $h_{Tcov0}^2$ . Note that for traits with no detectable maternal genetic effect variance ( $\sigma_{M_g}^2 = 0$  and  $\sigma_{AM_g} = 0$ ), we only estimated  $h^2$ . For the non-Gaussian models of neonatal/juvenile survival and adult breeding success,

we computed the variance components and the heritability on the data-scale using the R-package QGglmm (de Villemereuil et al. 2016).

### Results

We found significant additive genetic variance for all morphological traits, survival to the age of both 1 and 2 years, female annual breeding success and adult longevity, but no significant additive genetic variance for neonatal survival, age at first reproduction in females or male annual breeding success (Tables 2 and S1). The proportion of phenotypic variance explained by these direct additive effects varied across traits. In line with previous studies in red deer (Kruuk et al. 2000), we found higher heritabilities for morphological than life-history traits, with an average heritability  $\bar{h}^2 = 0.43$  ranging from 0.18 to 0.63 for morphological traits, and an average  $\bar{h}^2 = 0.06$  ranging from 0.001 to 0.13 for life-history traits (Table 2 and Fig. 1). Among the morphological traits, adult traits had higher heritabilities than juvenile traits, with  $\bar{h}^2 = 0.53$  for adult traits, while  $\bar{h}^2 = 0.26$  for juvenile traits (Table 2 and Fig. 1), which is also consistent with previous estimates (Kruuk et al. 2000; Logan et al. 2016). Note that neonatal survival was the only study trait for which none of the random components fitted explained a significant proportion of phenotypic variance, reflecting the low mechanistic understanding we have about these multicausal deaths.

We found significant total maternal effect variance for birth weight, birth leg length, first- and second-year survival ( $m^2$ ; Table S2). Maternal effect variance explained a negligible proportion of phenotypic variance for neonatal survival, age at first reproduction and late-life traits. Significant maternal genetic effect variance was only found for the two morphological traits measured soon after birth (Table 2). These effects represent 31% [24%; 43%] (95% credible intervals) of the total phenotypic variance ( $V_P$ ) in birth weight, and 17% [6%; 25%] of the total phenotypic variance in birth leg length. There was a small, but nonsignificant, maternal genetic variance estimated for neonatal and juvenile survival (explaining 2–3% of  $V_P$ , but with posterior distribution close to zero; Tables 2 and S1). For first- and second-year survival, our results thus suggest that we lacked the statistical power to properly decompose maternal effect variance  $m^2$  into significant genetic and environmental components  $h_{M_g}^2 + h_{Me}^2$ . For traits expressed later in life, that is, life-history and adult morphological traits, maternal genetic effects were negligible (explaining less than 0.1% of  $V_P$ ; Table 2).

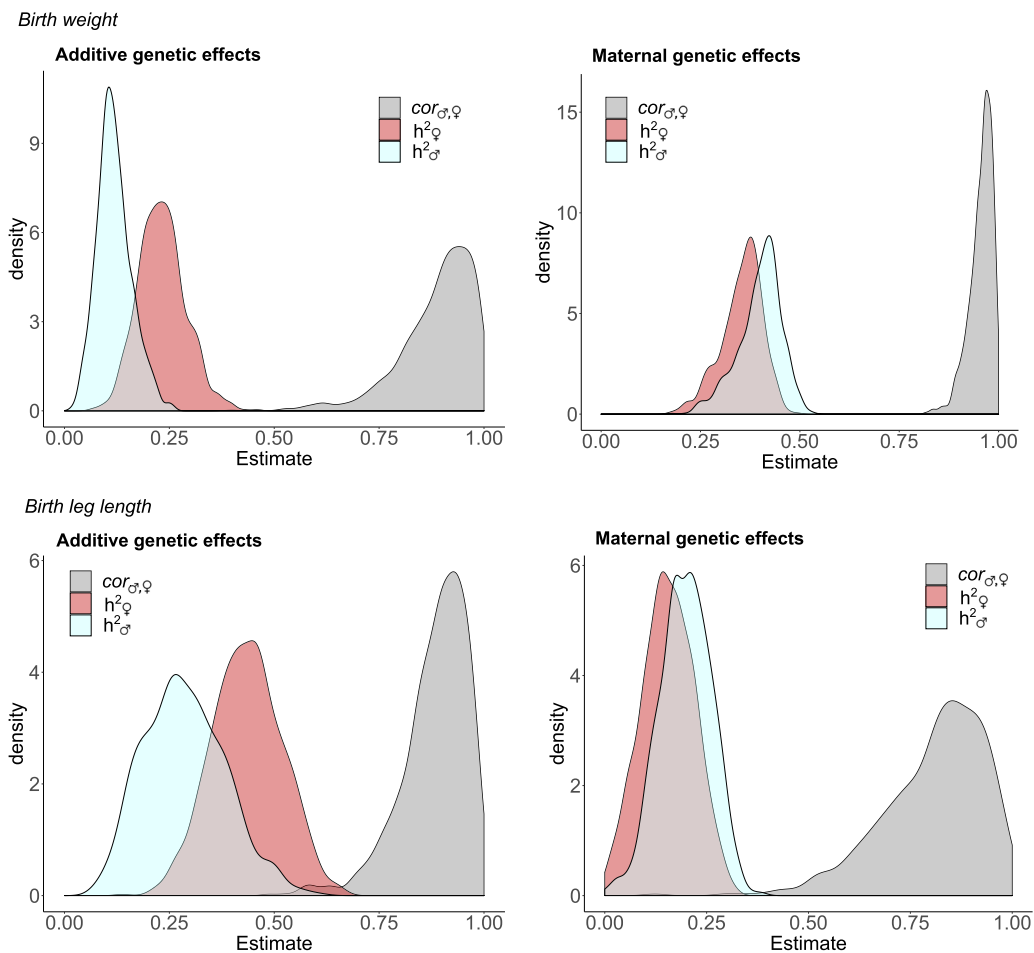
Maternal environment effects variance was not significant for any trait and explained a lower proportion of phenotypic variance than additive and maternal genetic effects, with only on average 0.8% of  $V_P$  explained by these effects. Maternal



**Table 2.** Posterior modes and 95% credible intervals for the proportion of total phenotypic variance explained by each random component in model (1).

Trait	$h^2$	$h^2_{Me}$	$h^2_{MR}$	$h^2_C$	$h^2_{YR}$	$h^2_{PE}$
<b>Neonatal trait</b>						
Birth weight	0.177 [0.098; 0.241]	0.307 [0.243; 0.426]	0.081 [0; 0.129]	0.047 [0.024; 0.078]	–	–
Birth leg length	0.335 [0.21; 0.457]	0.17 [0.065; 0.253]	0.001 [0; 0.128]	0.026 [0.007; 0.064]	–	–
Neonatal survival	0.022 [0; 0.072]	0.021 [0; 0.039]	0 [0; 0.018]	0 [0; 0.003]	–	–
<b>Early-life traits</b>						
Survival age 1	0.051 [0.019; 0.099]	0.026 [0; 0.052]	0 [0; 0.04]	0.115 [0.084; 0.185]	–	–
Survival age 2	0.052 [0.015; 0.087]	0.026 [0; 0.048]	0 [0; 0.035]	0.101 [0.064; 0.151]	–	–
AFR ♀	0.001 [0; 0.261]	0.001 [0; 0.137]	0.001 [0; 0.132]	0.077 [0.031; 0.145]	–	–
<b>Late-life traits</b>						
ABS ♀	0.04 [0.021; 0.052]	0 [0; 0.012]	0 [0; 0.006]	0 [0; 0.004]	0.035 [0.024; 0.061]	0 [0; 0.016]
ABS ♂	0.018 [0; 0.038]	0 [0; 0.015]	0 [0; 0.016]	0.009 [0; 0.02]	0 [0; 0.005]	0.029 [0.012; 0.054]
Adult longevity	0.13 [0.019; 0.3]	0.001 [0; 0.123]	0.001 [0; 0.103]	0 [0; 0.067]	–	–
Jaw length	0.447 [0.269; 0.559]	0.001 [0; 0.128]	0 [0; 0.052]	0 [0; 0.045]	–	–
Endocranial volume	0.629 [0.501; 0.824]	0.001 [0; 0.142]	0 [0; 0.101]	0 [0; 0.025]	–	–
Leg length	0.502 [0.293; 0.764]	0.001 [0; 0.159]	0.001 [0; 0.126]	0.001 [0; 0.054]	–	–

Note:  $h^2$ ,  $h^2_{Me}$ ,  $h^2_C$ ,  $h^2_{YR}$ , and  $h^2_{PE}$  represent the contribution of additive genetic effects, maternal genetic effects, maternal environmental effects, cohort effects, year of measurement effects and permanent environmental effect on individual's phenotype, respectively. For survival and annual breeding success, we used the MCMCglmm output and QGglmm package to provide the parameters estimates on the data scale. AFR = age at first reproduction; ABS = annual breeding success; ABS was analyzed separately in males and females.



**Figure 2.** Posterior distributions of the sex-specific heritabilities ( $\sigma$  = male in blue,  $\varphi$  = female in red) and the correlation between the effects expressed in males and females ( $cor_{\sigma,\varphi}$  in gray) for the two neonatal morphological traits. The proportion of variance explained by additive (left) and maternal (right) genetic effects were similar for the two sexes (overlapping distributions). The fact that the gray distribution overlaps one also highlights that the genetic effects expressed for males and females are identical. For birth weight the correlation between maternal genetic effects  $cor_{\sigma,\varphi} = 0.97$  [0.90; 1.0], and for birth leg length  $cor_{\sigma,\varphi} = 0.86$  [0.57; 1] (posterior mode and 95% credible intervals).

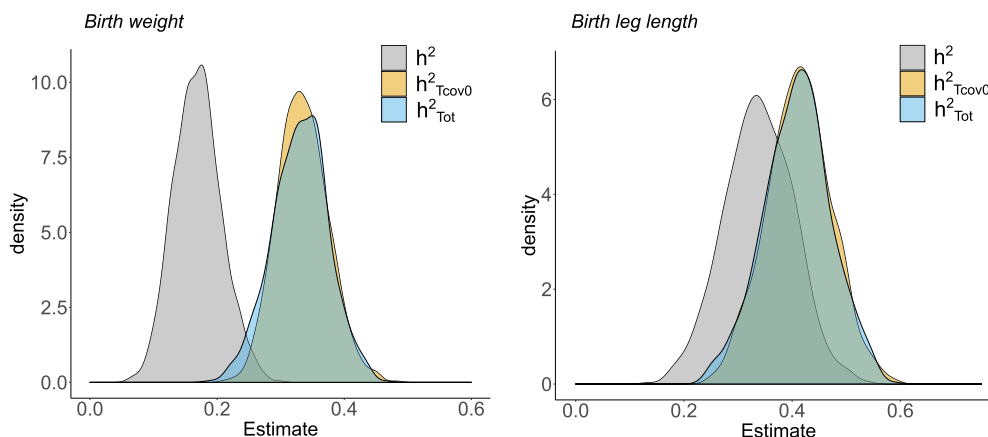
environmental effects were slightly more important for birth weight than other traits, but then did not change much across traits expressed at different times of the life history (Table 2).

For the two neonatal morphological traits exhibiting significant maternal genetic effect variance, the covariance between direct additive and indirect maternal genetic effects was not significantly different from zero, with  $\sigma_{AM_g} = 0.011$  [−0.10; 0.09] for birth weight, and  $\sigma_{AM_g} = 1.375$  [−30.2; 20.6] for birth leg length (Table S3 and Fig. S1).

We also found no evidence for differences in the sex-specific variance components, with overlapping posterior distributions for variance components estimated in males and females (Table S4). In particular, the proportion of variance explained by additive and maternal genetic effects was similar for the two sexes (Fig. 2), and the correlation between direct and indirect genetic effects expressed in males and females was very close to 1 (for

birth weight  $cor_{\sigma,\varphi} = 0.93$  [0.72; 1.0] for additive genetic effects and  $cor_{\sigma,\varphi} = 0.97$  [0.90; 1.0] for maternal genetic effects; Fig. 2). Note that these sex-specific models also indicated a low contribution of maternal environment effects to the total phenotypic variance in birth weight and birth leg length for the two sexes (for birth weight  $h^2_{Me\sigma} = 0.0005$ ;  $h^2_{Me\varphi} = 0.0006$ ; Table S4). For the adult traits, we used sex-specific models to check that there was no evidence for maternal effects variance in either sex (Table S5), a contrast to previous findings (Kruuk et al. 2000).

Finally, from the posterior distributions of  $\sigma_A^2$ ,  $\sigma_{M_g}^2$ , and  $\sigma_{AM_g}$ , we calculated a total heritability  $h^2_{Tot} = 0.30$  [0.23; 0.41] for birth weight, and  $h^2_{Tot} = 0.38$  [0.27; 0.53] for birth leg length (Fig. 3). Maternal genetic effects thus increased the amount of additive genetic variance by almost twofold for birth weight and by more than a third for leg length. Note that the calculation



**Figure 3.** Posterior distributions of the heritability ( $h^2$ ) and total heritabilities ( $h^2_{Tot}$  and  $h^2_{Tcov0}$ ) for the two neonatal morphological traits with significant maternal genetic effect variance. For birth weight, the posterior mode and 95% credible intervals are  $h^2_{Tot} = 0.36$  [0.23; 0.45],  $h^2_{Tcov0} = 0.33$  [0.26; 0.41], and for birth leg length  $h^2_{Tot} = 0.42$  [0.27; 0.55],  $h^2_{Tcov0} = 0.39$  [0.29; 0.55] ( $h^2$  estimates are provided Table 2).

of  $h^2_{Tot}$  using the posterior distribution of  $\sigma_{AM_g}$  or setting up  $\sigma_{am_g} = 0$  provided very similar estimates and credible intervals ( $h^2_{Tcov0}$ ; Fig. 3 and Table S3). This result highlights again the low contribution of  $\sigma_{AM_g}$  to the evolutionary potential of offspring traits.

## Discussion

### MATERNAL GENETIC EFFECTS INCREASE THE TOTAL HERITABILITY

Theory has shown that indirect genetic effects, such as maternal effects, can substantially impact the evolutionary trajectories of quantitative traits (Willham 1972; Kirkpatrick and Lande 1989; Wolf et al. 1998; Wolf and Wade 2016). More particularly, the genetic architecture of maternal effects can have complex consequences for the response of offspring traits to selection (Willham 1972; Kirkpatrick and Lande 1989). Analyzing one of the largest pedigrees for a wild mammal population, we found high maternal genetic effect variance for neonatal morphological traits, namely weight and leg length measured at birth. However, we found no empirical support for the hypothesis that these effects may mediate a trade-off between different components of phenotype and constrain the evolutionary potential of offspring traits. On the contrary, our results highlight that maternal genetic effects can substantially increase the amount of genetic variation available for selection ( $h^2_{Mg} = 0.31$  and  $0.17$  for birth weight and leg length, respectively). For birth weight, the trait most affected by maternal genetic effects, the additional additive genetic variation contributed by maternal effects doubles its estimated “total heritability” ( $h^2 = 0.18$  and  $h^2_{Tot} = 0.36$ ). For both birth weight and leg length, the covariation (and correlation) between direct and indirect genetic effects was not significantly different from zero.

This result suggests that the evolutionary response of offspring traits will not be impeded or facilitated because of a correlated response of maternal performance. Although we estimated quite large credible intervals around the correlation, this uncertainty is similar to that reported for non-significant components in similar study systems (e.g., in Soay sheep; Wilson et al. 2005).

To our knowledge, only two other studies have estimated the covariance between additive and maternal genetic effects in natural populations and for traits comparable to ours (McAdam et al. 2002; Wilson et al. 2005). Wilson et al. (2005) estimated a nonsignificant component of covariance for birth weight in Soay sheep, and McAdam et al. (2002) estimated a positive covariance for growth in body mass, and a nonsignificant component of covariance for growth in skeletal size in red squirrels. Since the 1980s, several studies in laboratory mice have also reported a positive covariance between direct and indirect genetic effects for body weight (Cheverud 1984a; Riska et al. 1985). Evidence that the genetic architecture of maternal effects can constrain the evolutionary potential of offspring traits in wild populations is thus sparse. However, a consistent and strong result among wild mammal studies (including ours) is that maternal effects are an important source of genetic variation that increase the evolutionary potential of juvenile traits (McAdam et al. 2002; Wilson et al. 2005; Kruuk and Hadfield 2007; Bérénos et al. 2014; Quéméré et al. 2018).

Our study was specifically designed to investigate the consequences of the genetic architecture of maternal effects for the evolutionary potential of offspring traits. We thus aimed at providing robust estimates of the strength of maternal genetic effects in the wild. Nevertheless, the biology of the red deer, especially the philopatry of the females (Clutton-Brock et al. 1985), may hamper the accurate dissociation of genetic and permanent

environment effects. Neglecting shared environment effects could notably mask genetic correlations among traits (Morrissey et al. 2012) and inflate the contribution of genetic and maternal effects to the total phenotypic variation (Stopher et al. 2012). In an attempt to address this issue, we have included spatial effects as a fixed term in our model, thus estimating the contribution of direct additive and maternal genetic effects on the remaining phenotypic variance. Additionally, we found that the pedigree contained a substantial amount of variance in the genetic relatednesses uncorrelated to the permanent (maternal) environment effects, that should allow us to decompose the genetic and non-genetic sources of variation in offspring traits (see Part S1).

We also found that maternal environmental effects had a very low and nonsignificant impact on the traits studied, which suggests that variation in maternal performance for offspring traits has a low environmental basis. This is unexpected given that most quantitative traits are largely determined by non-genetic factors. However, we know that maternal traits and performance in red deer are highly plastic (Froy et al. 2019). The variance partitioning approach taken here estimates only “permanent environment” maternal effects, in the form of consistent differences between mothers, and so does not incorporate any plastic maternal effects (e.g., that vary from year to year). Plasticity in maternal effects will be captured by the relevant fixed effects, or will be included in the residual variance (Kruuk 2004), and as such its impact is not apparent via the modeling approach used here. Here, we may therefore substantially underestimate the magnitude of maternal environmental effect variance in the study population. A “trait-based” approach would probably be more suitable to quantify the strength of these environmental effects (Kirkpatrick and Lande 1989).

Finally, it is important to highlight that the evolutionary consequences of genetic correlations between offspring trait and maternal performance can only be interpreted in the light of the selective pressures actually acting on these traits. The Willham model (Willham 1972), developed in the context of animal breeding, assumes directional selection is limited to offspring traits. However, in natural populations, it is likely that maternal performance comes with a cost in terms of maternal survival or reproductive success (parent-offspring conflict), and so maternal performance is also likely to be under selection (Cheverud 1984b; Hadfield 2012; Rollinson and Rowe 2015; Thomson et al. 2017). Note that this more realistic framework does not change our conclusions about the evolutionary consequences of the genetic architecture of maternal effects, as we found that the genetic effects for neonatal morphological traits and for maternal performance were independent (i.e.,  $\sigma_{am_g} = 0$ ). However, this means that in natural populations some evolutionary constraints may still arise from antagonistic selection on offspring trait and maternal performance. Testing whether maternal effects can explain the ob-

served evolutionary stasis of traits in nature (Merilä et al. 2001; Kruuk et al. 2008; Pujol et al. 2018) thus requires understanding both of the quantitative genetic (co)-variance components and of selective pressures (e.g., see Thomson et al. 2017).

### DECLINE IN MATERNAL GENETIC EFFECT VARIANCE OVER INDIVIDUAL LIFE HISTORY

A common expectation about maternal effects is that their relative contribution to the total phenotypic variation,  $m^2$ , should decrease over the ontogeny, as the importance of environmental effects increases (Mousseau and Dingle 1991; Lindholm et al. 2006; Wilson and Réale 2006; Moore et al. 2019). In some animal species such as the red deer, maternal care can be important up to the age at maturity and beyond (Clutton-Brock et al. 1985; Andres et al. 2013), which may attenuate the decline of  $m^2$  over the ontogeny for species providing such extended care (Moore et al. 2019). We found that variation in maternal effects was mostly genetic in origin and that its magnitude decreased rapidly over the red deer life history, with the only evidence of maternal genetic effect variance being for the neonatal morphological traits. Despite significant broad maternal effect variance for both first- and second-year survival ( $m^2 = 0.03$  [0.01; 0.06] for first-year survival; see also Walling et al. 2014), we lacked the statistical power to decompose these effects into significant genetic and environmental components. However, this power issue likely only affects the estimated credible intervals, but does not bias our point estimates. Therefore, we can sensibly conclude that the magnitude of maternal genetic variance was much lower for juvenile survival than for neonatal morphological traits (average  $h_{Mg}^2 = 0.05$  for the juvenile survival measures compared with  $h_{Mg}^2 = 0.177$  for leg length and 0.335 for birth weight). The greater relative magnitude of maternal genetic effect variance for birth weight than birth leg length is consistent with the idea that mothers may have a larger influence on fetal condition (through energetic provisioning of embryos) than on more deterministic traits such as offspring skeletal size (Bernardo 1996). Overall, our results are in line with the conclusion of a recent meta-analysis showing that total maternal effects (i.e., both genetic and environmental combined) are more important in juvenile than adult traits, and that the strength of these effects is not more important in species with maternal care after birth (Moore et al. 2019).

Here, we provide a comprehensive analysis of the change in maternal genetic effect variance over individuals' life history. This within-population study avoids introducing bias due to the sampled population or the statistical models used when comparing maternal effects at different ages. However, in contrast to studies on domestic or laboratory species (Lindholm et al. 2006; Wilson and Réale 2006; White and Wilson 2019), we do not have repeated measurements of a specific trait over the entire ontogeny to track the changes in maternal effect variance.

Instead, we compared different traits expressed at different ages. One possible issue here is that we may confound the change in maternal effect variance due to the ontogeny with that due to the different genetic architecture of morphological and life-history traits. Indeed, many studies have reported a lower contribution of genetic effects (direct or indirect) to the total phenotypic variance of traits more closely related to fitness, notably as a consequence of their higher residual variance, which is probably due to larger environmental effects on these traits (Merilä and Sheldon 1999; also found in red deer Kruuk et al. 2000). Our results are consistent with these expectations and empirical findings, as we estimated much lower heritability for life-history traits than morphological traits (the highest heritability being found for adult morphology). We also found small or nonsignificant variance in maternal genetic effects for the life-history traits analyzed, even when fitting a “total” maternal effect. Therefore, differences in the genetic architecture of traits may also explain the striking change in maternal genetic effect variance measured for morphological neonatal traits and for neonatal and juvenile survival, two types of traits expressed early in life.

Finally, the evidence (or lack of evidence) of maternal effect variance for traits expressed at different ages may be related to variation in sample sizes over the red deer life history. Indeed, we had lower sample sizes for late-life (including age at first reproduction) than for early-life traits (neonatal morphology, juvenile survival). This may reduce our capacity to detect significant variance in maternal effects for late-life traits, especially when this variance is low. Yet again, if we solely compare the point estimates (which we assume unbiased), our conclusion about higher maternal effect variance in early-life than late-life traits holds. However, our lack of statistical power to detect this significant variance for life-history traits conflicts with the previous studies in this population of red deer. Kruuk et al. (2000) and Walling et al. (2014) showed significant maternal effect variance for age at first reproduction, longevity, and total fitness despite smaller samples sizes and a less accurate pedigree than ours. This contrast probably reflects differences in the models used, such as the distributional assumptions (life-history traits formerly assumed Gaussian) or the influence of environmentally derived maternal effects that we removed by the fitted fixed spatial effects in our study. Finally, the low heritability of the life-history traits studied is consistent with former studies in the red deer population (Kruuk et al. 2000; Morrissey et al. 2012; Walling et al. 2014).

#### **NO SEX DIFFERENCES IN THE GENETIC ARCHITECTURE OF MATERNAL EFFECTS**

In species with strong sexual dimorphism, sexually antagonistic gene expression is expected to play an important role in the maintenance of genetic variation (Foerster et al. 2007; Kruuk et al.

2008). Although many studies have looked at the genetic architecture of traits in males and females independently, only a few studies have estimated the correlation between the genetic effects expressed by the two sexes (also called “cross-sex correlation”; reviewed by Kruuk et al. 2008; but see Walling et al. 2014). Similarly, we can expect sex-specific maternal effects to have evolved in natural populations, as the result of different selective pressures acting on males and females, or because of different sensitivity to maternal effects between the sexes (e.g., Badyaev et al. 2002; Badyaev 2005). However, very few studies have estimated cross-sex genetic correlation for maternal effects (e.g., Kruuk et al. 2015), especially in wild populations (but see Svensson et al. 2009).

Here, we tested for sex-specific direct and indirect genetic effects for the two neonatal morphological traits with significant maternal genetic effects. In the red deer, we know that maternal investment differs with offspring sexes, even at very early stages of life, with males having a longer gestation and being born heavier than females (Clutton-Brock et al. 1982). Nevertheless, we found no evidence for differences in the amount of maternal genetic variation, and direct heritabilities, expressed in male and female offspring. Female and male traits thus have the same potential to respond to natural selection. More importantly, we estimated a very high (and nonsignificantly different from one) cross-sex correlation for both additive and maternal genetic effects for the two neonatal traits. Our results thus indicate a very conserved genetic architecture for maternal effects on morphological traits in males and females. Mothers that produced large sons also produced large daughters, and there was no evidence for a genetic trade-off in maternal performance. These findings are consistent with a literature review showing cross-sex genetic correlations are generally positive for morphological traits (Kruuk et al. 2008).

In this study, we found low to negligible maternal genetic effect variance for other traits than neonatal morphology. However, in other study systems with stronger maternal genetic effect variance for fitness-related traits, it would be interesting to look further for sex-specific effects. Furthermore, to understand the evolution of sexual dimorphism and the maintenance of genetic variation in natural populations, it is critical not only to investigate the sexually differentiated genetic architecture of traits, but also to estimate the selective pressures acting on these traits.

### *Conclusion*

Using one of the largest pedigreed wild mammal population datasets, we evaluated the contribution of maternal genetic effects to traits expressed at different ages. We found substantial maternal genetic effect variance for the two morphological traits



measured at birth, and negligible variance thereafter. These genetic effects were expressed similarly in the two sexes. We found no evidence that the genetic architecture of maternal effects, and particularly their covariation with direct genetic effects, constrains the evolutionary response of these neonatal traits. On the contrary, neglecting the contribution of maternal genetic effects as a source of additive genetic variation leads to substantial underestimation of the evolutionary potential of offspring traits. Forthcoming selection analyses on offspring traits and maternal performance for these traits will provide a complete picture of the evolutionary consequences of maternal effects.

#### AUTHOR CONTRIBUTIONS

JMP and CAW designed the research. SM and AM collected field data. JG performed the analyses. JG, JMP, LEBK, and CAW discussed and interpreted the findings. JG wrote the first draft of the manuscript and JMP, CAW, and LEBK contributed substantially to revisions.

#### ACKNOWLEDGMENTS

This work builds on pilot analyses conducted by I. Dimopoulos and J. Huisman. We thank F. Guinness, M. Baker, C. Logan, and many others for collecting field data, and T. Clutton-Brock and S. Albon for their contributions to the long-term project. We are grateful to J. Huisman for generating the pedigree data, and to S. Johnston, J. Hadfield and J. Pick for useful discussions. We also thank Scottish Natural Heritage for permission to work on the Isle of Rum National Nature Reserve and the Wellcome Trust Clinical Research Facility Genetics Core in Edinburgh for performing the SNP genotyping. The long-term project and this research were funded by the UK Natural Environment Research Council, and most of the SNP genotyping was supported by a European Research Council Advanced Grant to JMP.

#### DATA ARCHIVING

The data used in this article are available from Dryad at <https://doi.org/10.5061/dryad.gtht76hhs>.

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Associate Editor: K. McGuigan  
Handling Editor: T. Chapman

## Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

**Table S1:** Variance components estimated from the main model.

**Table S2:** Proportion of phenotypic variance explained by total maternal effects.

**Table S3:** Estimates and credible intervals for the covariance and correlation between additive and maternal genetic effects, and the derived total heritability.

**Fig. S1:** Posterior distributions for the covariance and correlation between additive and maternal genetic effects.

**Table S4:** Variance components estimated from the sex-specific model for neonatal morphological traits.

**Table S5:** Variance components estimated from the sex-specific model for adult traits.

**Table S6:** Estimates and p-values for the fixed effects fitted in the main model.