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LETTER

A mother's legacy: the strength of maternal effects in animal populations

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Abstract

Although mothers influence the traits of their offspring in many ways beyond the transmission of genes, it remains unclear how important such 'maternal effects' are to phenotypic differences among individuals. Synthesizing estimates derived from detailed pedigrees, we evaluated the amount of phenotypic variation determined by maternal effects in animal populations. Maternal effects account for half as much phenotypic variation within populations as do additive genetic effects. Maternal effects most greatly affect morphology and phenology but, surprisingly, are not stronger in species with prolonged maternal care than in species without. While maternal effects influence juvenile traits more than adult traits on average, they do not decline across ontogeny for behaviour or physiology, and they do not weaken across the life cycle in species without maternal care. These findings underscore maternal effects as an important source of phenotypic variation and emphasise their potential to affect many ecological and evolutionary processes.

Keywords

Animal model, life-history theory, meta-analysis, quantitative genetics.

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INTRODUCTION

Phenotypic differences among individuals govern patterns of survival and reproduction. Exploring the sources of this trait variation therefore remains foundational to understanding the trajectories and limits of adaptive evolution (Mousseau & Roff 1987; Kruuk *et al*. 2008), population dynamics (Schoener 2011) and even community assembly (Bolnick *et al*. 2011). Maternal effects – the maternal phenotype's causal influence on the phenotypes of her offspring (Bernardo 1996a) – may be one important source of such variation. Indeed, mothers have substantial capacity to affect their offspring's traits above and beyond the direct transmission of genes (Bernardo 1996a), for example via per-capita provisioning (Dugas *et al*. 2016), host or oviposition-site selection (Mitchell *et al*. 2013) and hormone transfer (Sheriff *et al*. 2010).

While maternal effects were initially considered a nuisance by early geneticists (reviewed in Räsänen & Kruuk 2007; Bonduriansky & Day 2009), they have attracted substantial attention over the last three decades for their own ecological and evolutionary potential (Kirkpatrick & Lande 1989; Mousseau & Fox 1998; Mousseau *et al*. 2009). For instance, maternal effects that have an underlying genetic component (e.g. heritable variation in the size of a female's eggs [Reznick 1982]) can increase the total genetic variation in the offspring phenotype and, thus, its ability to respond to selection (Kirkpatrick & Lande 1989). Many conditions also favour the evolution of adaptive or 'anticipatory' maternal effects, whereby mothers facultatively endow their offspring with traits that are suited to the future environment (Marshall & Uller 2007; Kuijper & Hoyle 2015). Conversely, some maternal effects handicap offspring fitness, as when the impacts of environmental stressors are passively transmitted to offspring (e.g. Beyer & Hambright 2017). In several cases, these maternal effects are even known to underlie evolutionary divergence (Badyaev *et al*. 2002; Pfennig & Martin 2009), demographic patterns (LaMontagne & McCauley 2001; Sheriff *et al*. 2010) and the composition of ecological communities (Duckworth *et al*. 2015; Van Allen & Rudolf 2016). However, for these broader ecological and evolutionary consequences to be widespread, maternal effects must be a common and significant source of phenotypic variation (Bonduriansky & Day 2009) – a largely unexplored proposition. Thus, one way to begin clarifying the ecological and evolutionary potential of maternal effects is to quantify the extent to which they generate trait differences within populations (see Mousseau & Roff 1987; Hoffmann *et al*. 2016 for examples with additive genetic effects).

While illuminating the broader impact of maternal effects will be aided by estimating their average contribution to phenotypic variation, theory and previous empirical work indicate that the strength of maternal effects may also vary in important ways. For example, mothers often have unilateral control over the initial size of their offspring via the energetic provisioning of embryos (Mousseau & Dingle 1991; Bernardo 1996b, 1996b). As a result, maternal effects may influence morphological traits of offspring more strongly than other types of traits. Similarly, in some animals, mothers care for their offspring long after hatching or parturition. Maternal effects may therefore be stronger in these species (Reinhold 2002). Another widespread expectation about maternal effects is that they should weaken throughout ontogeny as offspring encounter more environments (Mousseau & Dingle 1991; Wilson & Réale 2006; Lindholm *et al*. 2006). However, because the environment affects the development of some traits more than others (e.g. life-history vs. morphology [Houle 1992]), maternal effects may weaken most for traits with the greatest environmental sensitivity. Likewise, maternal care often continues until adulthood (e.g. Royle *et al*. 2012; Dugas *et al*.

2016), which may result in only modest average declines in the strength of maternal effects across ontogeny for species with maternal care. By evaluating the potential sources of heterogeneity in the strength of maternal effects, we can identify the conditions where their ecological and evolutionary consequences are likely to be greatest.

In this study, we explored the strength of maternal effects in animal populations. We compiled a database of studies that used detailed pedigrees to partition phenotypic variation into its additive genetic, maternal and environmental components (an analytical technique known as the 'animal model', Kruuk 2004; Wilson *et al*. 2010). From these estimates, we assessed the average proportion of phenotypic variation determined by maternal effects (typically known as 'm^2', e.g. Wilson & Réale 2006). This metric has several desirable properties for quantitative synthesis. For instance, just as narrow-sense heritability (h^2) reflects allelic variation at all loci rather than the specific allelic effects of any one locus (Mousseau & Roff 1987; Postma ; Hoffmann *et al*. 2016; Wood *et al*. 2016), m^2 estimates the cumulative influence of many different proximate maternal effects rather than the specific action of one (e.g. hormones, provisioning; McAdam *et al*. 2014). Additionally, m^2 is calculated in a relatively standardised way, aiding comparisons: (1) to h^2 for each trait and (2) among types of traits, taxa and life stages (e.g. Mousseau & Roff 1987; Wilson & Réale 2006; Kruuk *et al*. 2008; Hoffmann *et al*. 2016). Here, we began by considering the mean proportion of phenotypic variation determined by maternal effects and by additive genetic effects. We next tested for differences in the influence of maternal effects among types of traits (e.g. morphology, physiology); between species with and without maternal care; among life stages; between invertebrates and vertebrates; between male and female offspring; between livebearing and egg-laying animals and between amniotes and anamniotes. Finally, we compared ontogenetic declines in maternal effects among types of traits and between species with and without maternal care.

METHODS

Data collection

Our approach to gathering studies largely followed the PRISMA statement checklist (Preferred Reporting Items for Systematic Reviews and Meta-Analyses; Liberati *et al*. 2009; see also Koricheva & Gurevitch 2014; Nakagawa *et al*. 2017). We began by forward-searching Google Scholar for all articles prior to May 2017 that cited Kruuk (2004), which was among the first overviews of the animal-model method. We scanned the methods of each article, retaining it if it used an animal model with a term to partition the phenotypic variance determined by maternal effects (maternal identity; V_M). While most studies that use this technique cite Kruuk (2004), we also forward-searched the more recent overview provided by Wilson *et al*. (2010), retaining those studies that fit animal models with V_M and did not cite Kruuk (2004). Finally, we conducted an additional search on Google Scholar using the keywords 'animal model', 'quantitative genetics' and 'maternal'. Again, we scanned the methods of each returned article, retaining those that fit animal models with V_M and did not cite either of the two forward-searched articles. For studies to be retained and considered further, they needed to report, at minimum: (1) the amount of variance explained by the maternal effects term and (2) the sample size (Fig. S1).

One strength of using m^2 for quantitative synthesis is that each estimate was generated in a similar fashion. To further maximise consistency, we excluded several types of studies. First, we did not include studies of large mammals in zoos or farms. For these populations, it was often uncertain if all of the individuals included in the pedigree were kept at the same location. As quantitative genetic parameters differ among rearing environments (Wood & Brodie 2015), it remains unclear how estimates would be affected if, for example, parents of only some offspring were kept in different locations. Second, in cases where offspring were cross-fostered, we included the study only if it modelled the effects of both the birth mother and the rearing mother. In animals with prolonged parental care, mothers often influence offspring traits both before and after parturition (Dugas 2015), and these maternal effects may not always be the same for each offspring within a family (e.g. Badyaev *et al*. 2002). Thus, when only the effects of the rearing parent are considered, any preparturition maternal effects are subsumed by the additive genetic or residual variance terms, leading to underestimates of the total strength of maternal effects (Kruuk & Hadfield 2007). Finally, researchers occasionally included one or more fixed effects in their animal models to account for phenotypic variation that would otherwise have been in the V_M estimate (e.g. maternal diet or egg size as a predictor of offspring size at hatching). The V_M in these cases is only the phenotypic variation that is not already explained by the fixed effect (McAdam *et al*. 2014; Pick *et al*. 2016). We used estimates from models that did not include these terms when available, and we excluded the study when they were not. Overall, our final database included 151 studies (see Fig. S1 for flow chart and the Supporting Information for a reference list).

From each of these 151 studies, we obtained estimates of m^2 and h^2 for each trait. Most studies reported values in tables, but when estimates were only depicted in figures, we used ImageJ to extract them (Rasband 2012). When only the total maternal or additive genetic variance was reported (i.e. V_M or V_A), we calculated m^2 or h^2 by dividing these variance components by the total phenotypic variance (V_P). Few studies decomposed maternal variance into its genetic (V_{MG}) and environment (V_{ME}) components (2.2% of all estimates), and, in such cases, we pooled these parameters together to create a single V_M that accounted for the effects of both. In cases where animal models were fit to a cross-fostering experimental design, we pooled the common-brood term (V_C) and the overall V_M to account for the maternal effect of post-hatching/parturition maternal care (Kruuk & Hadfield 2007).

For each estimate of m^2 and h^2, we also noted: the study; the species; whether the species exhibits post-hatching/parturition maternal care; whether it was an invertebrate or vertebrate; the taxonomic class (e.g. Insecta, Actinopterygii); whether the species was livebearing or egg-laying; whether the species was an amniote or anamniote; the trait type (e.g. morphology, physiology; see Table S1); the sex of the offspring in

which it was measured; the life stage in which it was measured; the number of individuals for which the trait was measured; and, when reported, the standard error or credible intervals. We took a strict approach to classifying a trait as 'life-history' vs. 'fitness': traits categorised as 'fitness' were integrative estimates across all fitness components (e.g. lifetime number of offspring) and life-history traits were components of fitness that could be parameterised in a life table (e.g. annual survival). Size at a life-history transition (e.g. size at maturity) was scored as a morphological trait. To classify the life stage to which a trait belonged, we used the author's description whenever possible. When authors did not classify the trait, we assigned traits that were measured at a life-history transition (e.g. age at first reproduction) to the later life stage. In the few instances where a study reported multiple m^2 or h^2 estimated for different populations or treatments of an experiment, we considered each estimate to be a separate entry. In some cases, authors could classify a trait as belonging to either the mother or her offspring (e.g. lay date [mother] vs birth date [offspring]). In these instances, we used only those traits that were ascribed to the offspring such that both V_M and V_A were modelled.

Analyses

We used mixed-effects models ('*lme4*', Bates *et al*. 2015) to characterise patterns of m^2 and h^2. Prior to analyses, we arcsine square-root transformed all m^2 and h^2 estimates to improve normality and homoscedasticity. We also weighted the response variable by the $\log_{10}$ of the number of individuals for which the trait was measured. To test the significance of explanatory variables and make pairwise comparisons within each variable, we used the Satterthwaite denominator degrees of freedom approximation (*'lmerTest'*, Kuznetsova *et al*. 2016) and Tukey *post hoc* tests (*'lsmeans'*, Lenth 2016), respectively. Full results of *post hoc* tests are reported in the Supporting Information (Tables S4, S5). Weighting estimates by the number of maternal links in the pedigree produced similar results to these analyses (Supporting Information; Table S2), suggesting little bias arises from differences in pedigree quality.

Quantitative syntheses can present many sources of non-independence, and we attempted to account for these in our analyses. To control for non-independence among estimates from the same study, we included study as a random effect. However, there can also be sources of non-independence within studies – for instance, if two morphological traits measured in one study are more similar to each other than either is to any behavioural trait measured in the same study (Noble *et al*. 2017). Few studies in our database presented this kind of non-independence, and its magnitude was small even in those studies that did (Supporting Information). Furthermore, in nearly all cases, results from supplemental analyses that removed this non-independence are similar to those presented in the main text (Supporting Information). Across studies, estimates from the same species or from closely related species can also be correlated. We did not directly incorporate a phylogeny in our analyses because of the very sparse representation at intermediate levels of taxonomic resolution (e.g. little replication within genera, family, or orders). Instead, we included random effects for species and class (e.g. mammalia, ave, insecta), which were the higher levels of taxonomic resolution with the best replication.

To first estimate the overall mean proportion of phenotypic variance determined by maternal effects and by additive genetic effects, we used random-effects models that included each estimate of m^2 or h^2 as the response and the random-effects structure described above. Then, to compare the strength of maternal effects among the potential sources of heterogeneity, we fit separate mixed-effects models for each of the following comparisons: among trait types; between species with vs. without maternal care; among life stages; between vertebrates and invertebrates; between the sexes; between livebearing and egg-laying animals; and between amniotes and anamniotes. Here, each estimate of m^2 or h^2 was included as the response, the comparison was modelled as a single fixed effect, and the random effects were as described above. As 'fitness' is a feature of the entire life cycle, we did not assign these estimates to any one life stage, and we therefore do not include them in any comparisons among life stages. Moreover, there were only three total estimates for 'performance traits' (e.g. swimming speed), and we did not include these in any comparison among trait types. Finally, to test for differences in the ontogenetic declines of maternal effects among trait types and between species with and without maternal care, we conducted mixed-effects models with fixed effects that included either (1) trait type, life stage and their interaction; or (2) maternal care status, life stage and their interaction. Because embryonic traits were poorly represented across groups, we only considered juvenile and adult traits for these ontogenetic decline analyses.

To assess the sensitivity of our results to different analytical approaches, we conducted two sets of supplemental analyses. First, because sample-size-weighted analyses such as ours can produce upwardly biased estimates (Morrissey 2016), we conducted 'formal' meta-analyses on estimates of m^2 and h^2 for which measurement error was directly reported. We report these analyses in the Supporting Information because this subset of the data set was quite small (37% of estimates), and because formal meta-analytic techniques can also be biased when each estimate's magnitude and measurement error are correlated (Hamman *et al*. 2018) – as is the case here (m^2: $r = 0.257$, $t_{285} = 4.50$, $P < 0.001$; h^2: $r = 0.261$, $t_{285} = 4.57$, $P < 0.001$). Nonetheless, results from the formal meta-analyses were similar to those of the analyses reported in the main text (Supporting Information; Tables S8, S9). Second, some of the 151 studies reported checking the significance of V_M and removing it when non-significant (29.9% of all estimates). This practice is questionable because it inflates estimates of V_A (Kruuk & Hadfield 2007) and researchers rarely remove V_A when it is non-significant. Nevertheless, we also performed analyses where we included these non-significant, unreported m^2 estimates as 0's. While the inflated number of 0's reduced the estimated-marginal means relative to the analyses reported here, the qualitative patterns were the same (Tables S10, S11). Further supporting the exclusion of these unreported estimates, Egger's regressions on models reported in the main text showed no evidence of

publication bias for m^2 (see Supporting Information; Table S12).

RESULTS

Across 770 estimates of m^2 from 116 studies, 64 species and 9 classes, maternal effects determined 10.8% of all phenotypic variance (95% confidence range, hereafter: 8.6–13.3%). Additive genetic effects explained 21.6% of the variation (16.8–26.7%; Fig. 1). Although publication bias was implicated for some analyses of h^2 (Table S13; but not m^2, Table S12), this synthetic average is similar to others derived from animal-model studies (e.g. ~30%, Postma ; ~10–30%, Wood *et al*. 2016).

Because mothers may control some offspring traits more than others, we compared the strength of maternal effects among types of traits. As predicted, there are differences ($F_{5,723.8} = 10.2$, $P < 0.001$). In general, maternal effects are stronger on morphology than on most other types of traits. Maternal effects also tend to be stronger on phenology than other trait types, though none of these pairwise differences were significant (Fig. 2a; Table S4).

We continued by examining if maternal effects tend to explain more phenotypic variance in species with maternal care. However, there was no average difference between these groups ($F_{1,48.8} = 2.0$, $P = 0.159$; Fig. 2b).

We next tested the hypothesis that maternal effects weaken across the offspring life cycle. Maternal effects indeed differ among life stages ($F_{2,431.9} = 22.7$, $P < 0.001$), exhibiting greater influence over juvenile than adult traits (Fig. 2c, Table S4). In contrast, this analysis also showed stronger maternal effects on juvenile traits than embryonic traits (Fig. 2c, Table S4).

No significant differences were observed between invertebrates and vertebrates ($F_{1,105.3} = 1.2$, $P = 0.272$, Fig. 2d), between males and females ($F_{1,223.4} = 1.5$, $P = 0.227$; Fig. 2e), between livebearing and egg-laying animals ($F_{1,26.4} = 0.2$, $P = 0.692$, Fig. 2f) or between amniotes and anamniotes ($F_{1,48.1} = 2.2$, $P = 0.146$, Fig. 2g).

Finally, we explored the extent to which maternal effects weaken across the offspring life cycle among trait types and between species with vs. without prolonged maternal care. Ontogenetic declines in the strength of maternal effects were only significant for morphology, life history and phenology (life stage × trait type interaction: $F_{4,666.8} = 7.7$, $P < 0.001$; Table S5 for *post hoc* tests). In contrast, maternal effects did not weaken across the offspring life cycle for physiology or behaviour (Fig. 3a; Table S5). Additionally, ontogenetic declines in the strength of maternal effects were larger in species with prolonged maternal care than in species without ($F_{1,362.2} = 4.6$, $P = 0.033$; Fig. 3b; Table S5).

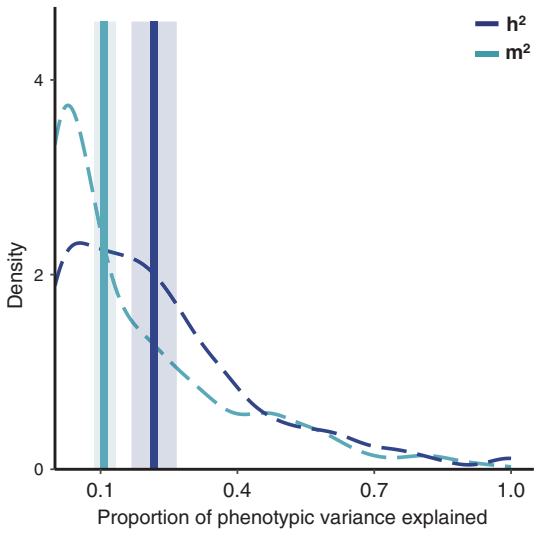

Figure 1 Density distributions and estimated-marginal means of m^2 (green) and h^2 (purple) in animal populations. Dashed lines are the smoothed histograms showing the distribution of all estimates in analyses. Vertical lines are means and shaded regions are 95% confidence intervals.

DISCUSSION

Although maternal effects are known to underlie ecological and evolutionary processes in some remarkable cases (Sinervo 1990; Agrawal *et al*. 1999; Badyaev *et al*. 2002; Sheriff *et al*. 2010), their general contribution to phenotypic variation remains unclear. Drawing on a large database of quantitative genetic estimates from animal populations, we provide the most comprehensive synthesis of the strength of maternal effects to date. Overall, our analyses reveal that maternal effects determine half as much phenotypic variance within animal populations as do additive genetic effects. Though substantial variation in the magnitude of these estimates remains to be explained (Tables S14-S20), maternal effects appear strongest on morphological and phenological traits, and tend to be strongest in the juvenile stage. Given the role of standing genetic variation in ecological and evolutionary processes (Barrrett & Schluter 2008), the observed strength of maternal effects relative to additive genetic effects suggests that maternal effects could indeed have broad impacts.

Limitations

To understand the importance of maternal effects, researchers typically explore either the total phenotypic variance determined by maternal effects or the consequences of particular non-genetic mechanisms by which mothers govern the traits of their offspring (McAdam *et al*. 2014). Here, we synthesised the variance-partitioning approach, which estimates the total impact of the many proximate mechanisms (provisioning, hormones, epigenetics) by which mothers influence their offspring's phenotypes. However, in light of some of the patterns that we observed, estimating and synthesizing the contributions of different proximate maternal effects across a broad range of taxa remains vital (e.g. for avian-focused examples, see Gorman & Williams 2005; Gil *et al*. 2007; Bentz *et al*. 2016). It is also important to note that the variance-partitioning approach synthesised here may not capture every non-genetic maternal influence on offspring (McAdam *et al*. 2014). Because each estimate of V_M is derived from the consistent effect that individual mothers have on all of their offspring, m^2 underestimates the contribution of maternal effects that increase the variance among siblings (e.g. Badyaev *et al*.

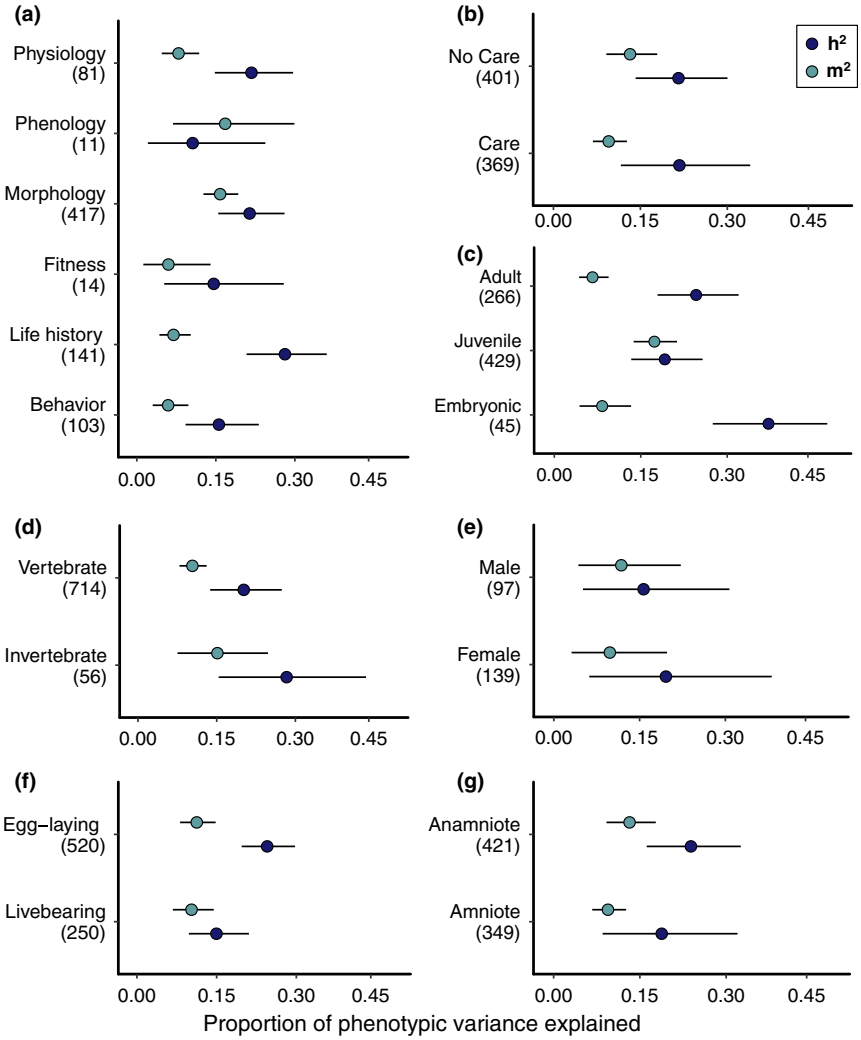

Figure 2 Estimated-marginal mean proportion of phenotypic variance (± 95% CIs) determined by maternal effects (green) and additive genetic effects (purple) among types of traits (a), between species with and without maternal care (b), among life-history stages (c), between vertebrates and invertebrates (d), and between male and female offspring (e), between egg-laying and livebearing species (f) and between anamniotes and amniotes (g). Numbers below categories indicate sample sizes.

2002). Likewise, when a single V_M is estimated using siblings across different cohorts, the impact of maternal effects may be underestimated if different mothers experience and respond to each year's environment in exactly the same way. The strength of maternal effects reported here is thus likely to be conservative. Moreover, important proximate maternal effects are stronger in plants than animals (e.g. transgenerational chromatin marking; Hu & Barrett 2017). Our estimates for the cumulative influence of maternal effects should therefore be viewed as conservative for the broader diversity of eukaryotes as well. Nevertheless, as these conservative estimates are still substantial relative to additive genetic effects on the same traits, our results indicate that maternal effects are an important source of phenotypic variation.

Variation in the strength of maternal effects

Mothers can greatly control some offspring phenotypes, such as morphology and phenology (Mousseau & Dingle 1991; Bernardo 1996b). Indeed, maternal effects on these traits are of similar strength to the corresponding additive genetic effects (Fig. 2a). The effect of maternal provisioning offers one appealing explanation for these patterns because its pronounced influence on offspring body size and the energetic reserves may govern subsequent morphological variation and the timing of key developmental events (i.e. phenology; Berven & Chadra 1988; Bernardo 1996b). Other proximate maternal effects, such as hormone transfer, also control aspects of the offspring phenotype (Love *et al*. 2013), but may not influence any particular type of trait as strongly as maternal provisioning acts on morphology and phenology. Disentangling the relative influence of different proximate maternal effects remains necessary, but our analysis indicates that maternal effects are likely to have their greatest ecological and evolutionary significance on morphology and phenology.

An earlier review of morphological and behavioural traits in hybrids suggested that post-hatching or post-parturition maternal care is the most important proximate maternal effect

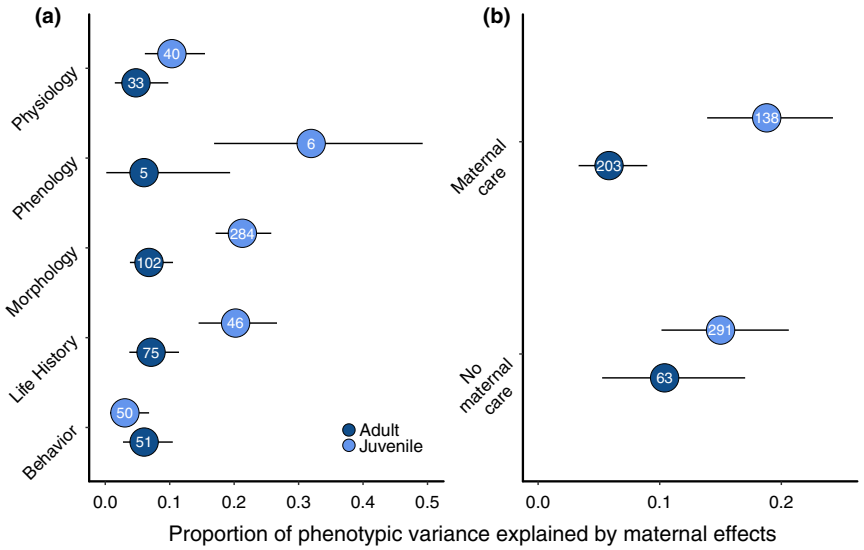

Figure 3 Estimated-marginal mean proportion of juvenile and adult phenotypic variance (± 95% CIs) determined by maternal effects among different types of traits (a) and between species with and without maternal care (b). Numbers within characters indicate sample sizes.

(Reinhold 2002). As maternal effects are not stronger in species with prolonged care than without, our findings do not support this contention. If animals share most other proximate maternal effects irrespective of whether or not they provide care (e.g. hormones, chromatin marking), it would be somewhat surprising that species that also have prolonged interactions with offspring after hatching or parturition do not exert greater average control over offspring phenotypes. Thus, rather than acting additively, it is plausible that the effects of prolonged care mask, or even trade off with, the effects of other proximate maternal effects (Williams 1994; Monteith *et al*. 2012). Alternatively, because mothers often care for their offspring unequally (Mock *et al*. 2011; Caro *et al*. 2016), maternal care may generate as much or more phenotypic variation within families as it does among them. Nonetheless, our results demonstrate that species without maternal care have a similar non-genetic impact on offspring traits as do species with prolonged care.

Theory also predicts that maternal effects should weaken across ontogeny. Consistent with this, maternal effects are weaker on adult traits than juvenile traits. Several explanations for this pattern seem plausible. Encountering more environments throughout ontogeny is thought to inflate the total phenotypic variance, and then, even if the absolute strength of maternal effects persists through time, their relative contribution simply becomes smaller (Mousseau & Dingle 1991; Wilson & Réale 2006). All else being equal, this phenomenon should also depress the proportional contribution of additive genetic effects (Houle 1992). As we did not find such declines in additive genetic effects, this explanation seems unlikely. Rather than the compounding of environmental effects, hormonal or other transcriptional shifts during reproductive maturation may curtail the importance of most maternal effects but not genetic effects. However, direct tests of this hypothesis remain necessary. In contrast to expectations of overall ontogenetic declines, there was also evidence for stronger maternal effects on juvenile than embryonic traits, though this effect was not found in all analyses (Supporting Information). More estimates of m^2 on embryonic traits are therefore needed to better understand these patterns. Finally, although weaker on adult than juvenile traits, maternal effects still explain a non-trivial amount of adult phenotypic variation (cf. Møller & Jennions 2002). This rejects the common assertion that maternal effects in adulthood are unimportant, showing instead that maternal effects can significantly contribute to the adult phenotype.

While stronger on juvenile traits than adult traits, there was also substantial variation in the extent to which maternal effects weakened across the offspring life cycle. For instance we predicted that life-history traits would show the largest ontogenetic declines in maternal effects due to their greater environmental sensitivity (Houle 1992). However, ontogenetic declines were similar for morphology, life-history and phenology, but were much smaller or non-existent for physiology and behaviour. Maternal effects also only weakened across ontogeny in species with post-hatching or post-parturition maternal care. The effect of maternal provisioning, which may disproportionately influence some traits and may be particularly influential in species with maternal care, again offers a unifying explanation. Whereas hormone or epigenetic transmission can regulate offspring gene expression throughout an individual's lifetime (Love *et al*. 2013; Hu & Barrett 2017), the influence of maternal provisioning may diminish after investment is ceased (Janzen & Warner 2009; Svanfeldt *et al*. 2017; but see Moore *et al*. 2015). This could lead to the larger declines in the strength of maternal effects across ontogeny for these trait types and taxa. It is also intriguing that maternal effects influence adult traits as greatly as they do juvenile traits for physiology and behaviour, as well as in species without prolonged maternal care. In this broad range of conditions, adult selective pressures may therefore be an overlooked factor shaping the evolutionary significance of maternal effects (see also Coulson *et al*. 2003; Warner & Shine 2008; Marshall & Monro 2012; Moore *et al*. 2015).

The evolutionary consequences of maternal effects

When maternal effects themselves have a heritable component, they increase the offspring trait's total genetic variation and, thus, its ability to respond to selection (e.g. McFarlane *et al*. 2015). For example, even if an individual's genotype does not influence its size at hatching, a selective advantage for mothers with genes that produce larger eggs will promote the evolution of larger hatchlings (Wolf *et al*. 1998). Although the paucity of estimates of 'maternal genetic effects' limits our understanding of genetic variation in maternal effects, the ubiquity of gene × environment interactions (West-Eberhard 2003) suggests a heritable component should exist for most maternal effects (Kruuk *et al*. 2008; Bonduriansky & Day 2009). In such cases, our analyses indicate that maternal effects may boost the average trait's total genetic variation by up to 50% (Fig. 1) – a large increase in its evolutionary potential (Hoffmann *et al*. 2016; but see Houle 1992). However, the rate and even direction of any ensuing response to natural selection depends on the association between the direct genetic effects and the maternal genetic effects ('direct-maternal genetic covariance'; Kirkpatrick & Lande 1989; Räsänen & Kruuk 2007). When direct genetic effects are positively associated with maternal genetic effects, as when genes for greater maternal provisioning are physically or pleiotropically linked with those for faster growth (Kölliker *et al*. 2000; McAdam *et al*. 2002), the additional genetic variation contributed by maternal effects should expedite adaptation. Conversely, when direct-maternal genetic covariances are negative (e.g. Wilson *et al*. 2005), large maternal effects can impede responses to selection or even guide them in non-intuitive directions (Kirkpatrick & Lande 1989; Bonduriansky & Day 2009). As direct-maternal genetic covariances tend to be negative in domesticated animals (Wilson & Réale 2006), this latter scenario may be common if wild populations are similar.

Beyond modifying the evolutionary potential of offspring phenotypes, maternal effects can themselves evolve such that mothers facultatively adjust offspring traits to match the future environment (Mousseau & Fox 1998; Marshall & Uller 2007). For such effects to arise, theory predicts that: (1) maternal effects must substantially influence offspring traits; (2) there must be some underlying genetic variation in the maternal effect; (3) aspects of the maternal environment must reliably predict the offspring environment; and (4) natural selection on offspring must be strong (Kuijper & Hoyle 2015; Walsh *et al*. 2016). Our findings indicate that the first condition will commonly be met. However, our analysis cannot reveal if most maternal effects are currently adaptive. As a recent meta-analysis found only modest experimental support for adaptive maternal effects as a general phenomenon (Uller *et al*. 2013, but see Burgess & Marshall 2014), the maternal effects documented here might primarily act as passive conduits of environmental variation from one generation to the next (Bonduriansky & Day 2009). Nevertheless, when the maternal environment predicts the offspring environment, selection on the considerable phenotypic variation determined by maternal effects should favour their adaptive evolution. Exploring the genetic (McAdam *et al*. 2014) and environmental (Marshall & Monro 2012; Tschirren *et al*. 2014; Moore *et al*. 2016) factors that promote and/or limit the evolutionary origins and maintenance of adaptive maternal effects will remain an exciting area for inquiry (e.g. Walsh *et al*. 2016).

CONCLUSIONS

We have provided the most comprehensive estimate of the strength of maternal effects to date. If we begin with the premise that standing genetic variation is an important contributor to phenotypic diversity within and among animal populations (Barrrett & Schluter 2008), then our findings indicate that maternal effects should also be seen as an influential source of phenotypic variation. Our results show that maternal effects have their greatest influence on offspring morphology and phenology and are most important in the juvenile life stage. Many factors likely underlie these patterns, and exploring the relative importance of different proximate maternal effects (e.g. provisioning, hormones) will provide insight into why some types of traits and life stages are influenced more than others (e.g. Pick *et al*. 2016). Although much remains to be discovered, this study ultimately indicates that maternal effects have the potential to shape many ecological and evolutionary processes.

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AUTHORSHIP

MPM and HHW conceived the study. MPM designed the study and collected the data. MPM and RAM analysed the data. MPM wrote the manuscript with input from HHW and RAM.

DATA AVAILABILITY STATEMENT

Data available from the Dryad Digital Repository (https://doi.org/10.506/dryad.360v97q).

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SUPPORTING INFORMATION

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

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