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Weak evidence for anticipatory parental effects in plants and animals

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Abstract

The evolution of adaptive phenotypic plasticity relies on the presence of cues that enable organisms to adjust their phenotype to match local conditions. Although mostly studied with respect to nonsocial cues, it is also possible that parents transmit information about the environment to their offspring. Such 'anticipatory parental effects' or 'adaptive transgenerational plasticity' can have important consequences for the dynamics and adaptive potential of populations in heterogeneous environments. Yet, it remains unknown how widespread this form of plasticity is. Using a meta-analysis of experimental studies with a fully factorial design, we show that there is only weak evidence for higher offspring performance when parental and offspring environments are matched compared with when they are mismatched. Estimates of heterogeneity among studies suggest that effects, when they occur, are subtle. Study features, environmental context, life stage and trait categories all failed to explain significant amounts of variation in effect sizes. We discuss theoretical and methodological reasons for the limited evidence for anticipatory parental effects and suggest ways to improve our understanding of the prevalence of this form of plasticity in nature.

Introduction

Parents provide a wide range of inputs that contribute to offspring development. Variation in the environment experienced by the parents can therefore have both positive and negative consequences for offspring fitness (Fig. 1). When environmental states are correlated between generations, parents that respond to their environment by changing their morphology, physiology or behaviour provide a source of information about the environment that will be experienced by the offspring (Shea *et al.*, 2011). Thus, from the offspring's perspective, the parental phenotype is a cue, and selection may shape offspring plasticity to optimize the use of that information (Mousseau & Dingle, 1991; Donohue & Schmitt, 1998; Mousseau & Fox, 1998; Agrawal *et al.*, 1999; Bateson *et al.*, 2004; Gluckman & Hanson, 2006; Marshall & Uller, 2007; Uller, 2008, 2012; Herman & Sultan, 2011; Fig. 1b i,ii). Adaptive plasticity mediated via parental effects goes under different names in the literature, but is often described as 'anticipatory parental effects' (Marshall & Uller, 2007; Marshall *et al.*, 2008), 'adaptive transgenerational plasticity' (Van Dam & Baldwin, 2001; Galloway & Etterson, 2007) or 'adaptive parental programming' (Horton & Stetson, 1990). Each of these terms emphasizes that selection may shape both the parental response to the environment, to maximize the information transmitted to the offspring, and the response of offspring to this variation in parental phenotype, to maximize fitness given the local environment (Mousseau & Fox, 1998; Uller, 2008, 2012; Herman & Sultan, 2011). Analogous to the use of environmental cues in adaptive plasticity, this implies that offspring fitness will be maximized when the parental and offspring environments are accurately matched (i.e. when the appropriate cue is followed by the environment that it predicts; Marshall & Uller, 2007; Monaghan, 2008). Examples of adaptive transgenerational plasticity include the effect of maternal

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environment on the timing of seed germination (Galloway & Etterson, 2007; Donohue, 2009) and predator-induced maternal effects on offspring morphology (Agrawal *et al.*, 1999).

To what extent parental effects allow parents to provide information for offspring that enables expression of adaptive plasticity in heterogeneous environments has received substantial attention in behavioural and evolutionary ecology over the past two decades (Mousseau & Fox, 1998; Marshall & Uller, 2007; Marshall *et al.*, 2008; Mousseau *et al.*, 2009; Maestripieri & Mateo, 2010; Uller, 2012). If such adaptive information transfer does occur, it may substantially facilitate adaptation to variable environments (Beldade *et al.*, 2011; Raubenheimer *et al.*, 2012). Understanding the adaptive significance of transgenerational plasticity is also important to be able to predict the consequences of parental effects for population dynamics and may help inform our understanding of how species, including humans, will respond to rapid environmental change (Bateson *et al.*, 2004; Dyer *et al.*, 2010; Bonduriansky *et al.*, 2012). However, the prevalence and strength of anticipatory parental effects in natural systems remain poorly understood (Sultan *et al.*, 2009; Uller, 2012).

Building a strong case for adaptive plasticity requires assessment of the target phenotype (i.e. fitness estimate) in matched vs. mismatched environments (Doughty & Reznick, 2004). In the context of transgenerational plasticity, this means that studies need to conduct a fully factorial design with at least two parental treatments (providing cues) and two offspring treatments (with different phenotypic optima, Marshall &

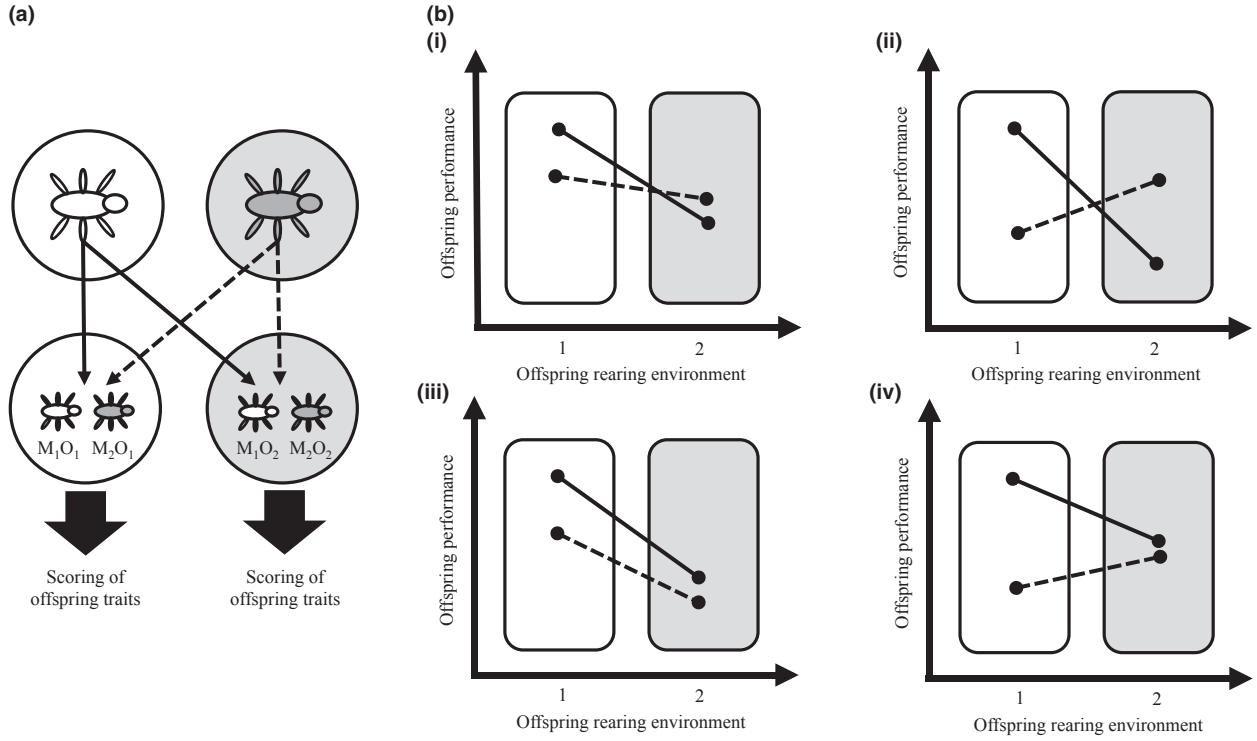

Fig. 1 (a) Schematic of fully factorial designs that test the combined effect of parental (large insects) and offspring (small insects) environment on offspring phenotype. One environment is typically more benign (here, environment 1). (b) Potential scenarios of phenotypic traits in offspring. Dots indicate phenotypic value (higher values have higher fitness). Lines link offspring born to mothers from the same environment (M_1, solid; M_2, dashed). (i) Within offspring environments, fitness is highest when environments persist across generations ('adaptive matching'), but average trait value in O_1 offspring is higher. (ii) Adaptive matching, as above, where the effect of experiencing a matched environment is stronger than the offspring environment effect. (iii) Offspring from M_1 mothers always have higher fitness than offspring from M_2 mothers ('carry-over' or 'silver spoon' effect). (iv) Carry-over and adaptive matching combined: offspring with M_1 mothers have higher fitness than M_2 offspring, yet, within maternal environments, fitness is higher when environments match across generations. Strong evidence that offspring use the parental phenotype as a cue in adaptive plasticity (i.e. anticipatory parental effects or adaptive transgenerational plasticity) would entail showing that maternal fitness is maximized when the cue matches the actual environment (i, ii). In scenario (iv), adaptive plasticity using parental cues may instead be inferred from the presence of reaction norms that are different in sign. In contrast, if transgenerational effects mostly reflect silver spoon effects, for example, due to resource provisioning, offspring from mothers in higher-quality environments should have higher fitness across all environments and slopes should be equal in sign (iii).

Uller, 2007). Although the environments of parents and offspring need not be the same, many adaptive scenarios involve levels of environmental heterogeneity that make offspring more likely to encounter the same environment as their parents rather than a different environment (e.g., Galloway & Etterson, 2007; see Materials and methods). Using meta-analysis, we therefore conducted a quantitative test of the strength of the effects of matching of parental environment (cue) and offspring environment (selective context) on expression of fitness-related traits in offspring. Specifically, on the basis of the predicted patterns of phenotypic variation in offspring from parents exposed to contrasting environments, who themselves are exposed to different environments (Fig. 1), we addressed the following key questions. First, is there overall support for the prediction that offspring should have higher performance when the parental and offspring environments are matched compared with when they are mismatched? Second, if this is not the case, are the general patterns of offspring plasticity most consistent with compensatory responses that reduce the negative impact of poor or stressful maternal environments or with silver spoon effects (i.e. positive effects of being born to mothers from relatively good environments)? Third, can we explain variation in the patterns within and among studies based on the types of environments considered, the organism under study or characteristics of the traits that are measured? Our results show that the experimental evidence for anticipatory parental effects is limited and that the effects, when they occur, are subtle compared with the direct effect of offspring environment.

Materials and methods

We conducted a comprehensive search for studies that reported results from a fully factorial design with at least two parental (typically maternal) and two offspring treatments. We first searched for papers on ISI Web of Science, using the following keywords singly or in combination: maternal effect(s), paternal effect(s), parental effect(s), transgenerational, plasticity, adaptive, experiment*, maternal, environment*, offspring, fitness. We also made use of the references in major reviews and books on the topic (e.g. Mousseau & Fox, 1998). Abstracts were scanned for indications that the study fulfilled our basic criterion and candidate papers were read in detail. We also solicited data from authors that we had seen presenting suitable unpublished results at conferences during 2011–2012 and emailed authors of papers to ask for supplementary information when the published data did not allow extraction of effect sizes. The search generated a total of 58 studies that contained at least one useful data point. The data from studies included in our analyses can be found in Table S1 in the Supporting Information. It is important to emphasize that our criteria explicitly excluded studies that inferred the presence of adaptive transgenerational plasticity based on a single offspring environment, which is common in, for example, studies of maternal transfer of immunity (review in Hasselquist & Nilsson, 2009). Although immunoglobulins and other immune factors transferred from mothers to offspring may carry information about disease risk, assessing offspring fitness in a single environment does not rule out the possibility that receiving and responding to those factors would be beneficial across environments and hence this design is unsuitable as strong tests of adaptive plasticity (Doughty & Reznick, 2004). In other words, it is unclear whether they represent cues about future environments or are better seen as resources. On the basis that adaptive significance of plasticity is difficult to assess without planned experiments (Doughty & Reznick, 2004), we also excluded purely correlative data, which ruled out, for example, human studies.

From the available data in text, tables or figures, we extracted the means and standard deviations, with transformations applied where appropriate (Table S1), for each of the four groups from a 2×2 factorial design (i.e. offspring in environment 1 born to mothers in environment 1 and all other combinations of offspring and maternal environments; Fig. 1). Note that it is not necessary that the environments of parents and offspring are the same (e.g. photoperiod), only that the former correlate with the latter (e.g. short photoperiod followed by cold temperature). Thus, in our paper, a 'matched environment' simply implies that the environment of the offspring is appropriately matched to the environment of the parent, so that the parental phenotype can provide the accurate cue about offspring environment given the natural environmental heterogeneity. It does not require that the environments are the same. Nevertheless, in the vast majority of empirical studies, the adaptive scenario did involve the same environment across generations (e.g. mothers in light gaps are more likely to have offspring growing up in light gaps; Galloway, 2005), and we therefore use this to imply match vs. mismatch in the figures. Six papers did not explicitly discuss adaptive scenarios but focused on estimating variance in offspring phenotype (e.g. Schmitt *et al.*, 1992). However, as these studies considered similar types of environmental heterogeneity as other studies, we have included them here; exclusion of these data had no qualitative effect on the results. In studies involving more than two treatment levels, we only used data from the two extreme groups, unless there were *a priori* reasons for including all combinations (e.g. when there were several different types of treatment rather than different levels of the same treatment). Where data were presented separately for different categories of individuals (e.g. males and females or different source populations), we entered separate lines for each category unless specified otherwise. Some trait

values were negatively associated with fitness (e.g. development time) or known to exhibit contrasting trait–fitness relationships in different environments (e.g. helmet and predator presence in *Daphnia*): in these cases, we multiplied the appropriate values by -1 to allow general interpretations.

We also recorded details about the study system, the study design, the experiment and the species, which could be used as moderators in our mixed-effects models (see Table 1 for a complete list of moderators and brief summary of the rationale for each and online supplementary methods for further details on data extraction and classification). We based our choice of moderators on the following *a priori* predictions. First, there are several reasons to expect the relative fitness effects of matched vs. mismatched environments to depend on the quality of the environment encountered by the parent and the offspring (see Introduction and Fig. 1). Indeed, most studies explicitly contrasted environments of different quality, such as low and high food availability. We therefore scored environments within each study design as 'good' vs. 'poor' whenever this was supported by the experiment and the details provided by the authors of the paper (see Supplementary Information). When there was no predicted direct environmental effect on maternal reproductive state (i.e. environments could not unambiguously be classified as 'good' and 'poor'), we classified the environmental quality as 'undefined'. Second, we expected that positive fitness effects of matched environments would be more common in plants than in animals. This prediction is based on the more sedentary nature of plants that is likely to be associated with an environmental grain that selects for adaptive transfer of information across generations (e.g. Galloway & Etterson, 2007). Third, we predicted that adaptive responses would be more common at early life stages, where offspring have relatively limited ability to assess their own environment and the maternal phenotype may be a more accurate cue to the offspring environment (Uller, 2008). Fourth, we expected stronger support for adaptive responses for traits that are more closely related to fitness, such as reproduction and survival, than for traits with relatively weak or unclear fitness effects (Table 1). Thus, we compared different types of traits to examine whether any adaptive parental effects were manifested more strongly among life-history, survival or fecundity-related traits compared with morphological or physiological traits.

Table 1 Description of moderators used to explain heterogeneity in effect size estimates for matching and carry-over analyses.

Moderator	Levels	Rationale/prediction
Offspring environment	Good, poor, undefined	Influence of maternal environment may depend on offspring environment (Fig. 1)
Life stage	Embryo, juvenile, adult	Stronger effects for earlier life stages
Kingdom	Plant, Animal	Stronger matching effects for plants
Trait type	Life history, morphology, physiology, reproduction, survival	Stronger effects for survival and reproduction

Calculation of effect sizes and statistical analyses

We used two approaches to test the generality of adaptive transgenerational plasticity (as defined above) and whether we could explain some of the heterogeneity within and among studies in terms of our moderators. In the first approach, we standardized each mean by the pooled standard deviation for each set of four means (for each treatment, e.g. M_1O_1, within each trait-within-study combination) according to the formula:

$$\tilde{X}_{ji} = \bar{X}_{ji}/\bar{\sigma}_{Xj}$$

where $\tilde{X}_{ji}$ is the standardized mean for the ith treatment within the jth trait–study combination, $\bar{X}_{ji}$ is the mean for each treatment, and $\bar{\sigma}_{Xj}$ is the pooled standard deviation across treatments within the jth trait-within-study, calculated as

$$\bar{\sigma}_{Xj} = \sqrt{\frac{\sum_{i=1}^{4}[\sigma_{ji}^2(n_{ji}-1)]}{\sum_{i=1}^{4} n_{ji} - 4}}$$

where σ_{ji} and n_{ji} are the standard deviation and sample size, respectively, for the ith treatment within the jth trait–study combination. The associated measurement standard error for each standardized mean value was calculated using the formula:

$$SE(\tilde{X}_{ji}) = \frac{\sigma_{ji}}{\bar{\sigma}_{Xj}\sqrt{(n_{ji}-1)}}$$

This approach allowed us to test directly the effect of offspring and maternal environment, and the effect of matched environments, on trait expression in offspring. This calculation was only possible for those studies where the trait–fitness relationship was the same in both environments. In this first analysis, we simply considered offspring environment (good or poor), maternal environment (good or poor) and whether offspring environment and maternal environment were matched, on variation in the standardized mean trait value (i.e. data with environmental quality 'undefined' were excluded for this analysis).

In our second approach, the data were used to calculate two sets of effect sizes based on the difference in offspring traits within and between treatments (Fig. 1). We calculated values for Hedges' *d* and corresponding measurement error variance for both offspring treatments, based on the difference between offspring phenotype in the matched treatment and the offspring phenotype in the mismatched treatment (i.e. M_1O_1-M_2O_1 and M_2O_2-M_1O_2; Fig. 1). For a reduced data set including only studies where the environment could be confirmed to have a direct effect on the maternal state (i.e. 'good' vs 'poor' environments), we also calculated effect sizes for which the sign reflected whether offspring had larger phenotypic values if they were born to mothers from good environments (i.e. M_1O_1-M_2O_1 and M_1O_2-M_2O_2, where the quality of $M_1 > M_2$; Fig. 1). Although the only difference between these two sets of effect sizes is that the sign of the effect size in offspring environment 2 is reversed, they directly capture the predictions of higher fitness for matched compared with mismatched offspring and positive carry-over effects of a good maternal environment, respectively (see Fig. 1). By including offspring environment as a moderator in our analyses, we could assess whether the effects are stronger within any given environment, which helps to identify if positive carry-over effects drive any positive overall effect on environmental matching. For example, an overall positive effect size across the first data set ('matching') implies that offspring do better when environments are matched, in general. If, however, there is also a positive effect size in the second data set ('carry-over') for offspring in poor environments (indicating higher fitness if born to good-environment mothers compared with poor-environment mothers), this pattern suggests that the apparent matching effect is, at least partly, driven by positive carry-over effects.

We adopted a meta-regression approach to assess the effect of several categorical predictors in one model (reviewed in Nakagawa & Santos, 2012), applying Bayesian mixed-effects meta-analysis (BMM) using the library *MCMCglmm* (version 2.16, Hadfield, 2010) in the statistical environment R (version 2.15, R Core Team, 2013). As our data set included repeated measures on traits within studies, as well as replication across several taxonomic levels, we included the random factors of class, study and trait as random terms in our BMM models and estimated the variance contributed by each (Hadfield & Nakagawa, 2010). Because we had few replicates of studies for any species (only 7 of 38 species featured in more than one study), we could not estimate additional variance at the species level. For the random effects, we used an inverse Wishart prior with $V = 0.002$ and $nu = 1$, which is widely used in the statistical literature (Gelman & Hill, 2007). For each statistical model, we ran three MCMC chains (i.e. three independent runs of *MCMCglmm* models) to test for convergence of model parameters among the chains. For every chain, we used the same settings for sampling: (i) the number of iterations of 2 000 000, (ii) the thinning interval of 500 and (iii) the number of burn-in of 1 500 000. This setting resulted in 1000 samples, which constituted our posterior distributions for all parameter estimates (fixed and random effects). We checked convergence of model parameters (or posterior distributions) using the Gelman–Rubin statistic (Gelman & Rubin, 1992). All sets of three chains had a PRS factor < 1.018, and hence, model convergence was appropriate. We only used posterior distributions from the first of three chains to report our results. If 95% credibility intervals of fixed effects did not span across zero, we deemed these effects were statistically supported. As an index of heterogeneity, we assessed the proportion of the total variance (the total of all variance components in a model) accounted for by a particular random factor (Nakagawa & Santos, 2012). For each of our three analyses, we first fit an intercept-only model to test for an overall effect and subsequently fit a second model including all moderators of interest (see Table 2 for the matching and carry-over analysis). We present our results graphically using the function coefplot in the R library arm (Gelman *et al.*, 2012), modified for MCMCglmm (code provided at http://ms.mcmaster.ca/~bolker/R/misc/coefplot_new.R). Finally, we tested for publication bias through visual inspection of funnel plots and by conducting Egger's regression analysis (Egger *et al.*, 1997).

Results

Standardized mean trait values were strongly influenced by offspring environment (Fig. 2), being lower in poor environments in general (95% highest posterior density, HPD: −1.288, −0.903), but there was no additional effect of maternal environment (HPD: −0.248, 0.131) or whether the maternal and offspring environments were matched (HPD: −0.082, 0.302). Heterogeneity among data was high ($I^2_{[total]}$: 95.73%, Table 2),

Table 2 Summary output from the intercept-only (meta-analytic) models fit to each data set. For the data set on standardized means, the full model included parental environment, offspring environment and the interactions between parental and offspring environment. Shown are the overall heterogeneities (I^2) for each model or the percentage of total variance explained by all random factors and heterogeneities attributed to random factors (taxonomic class, study, trait).

Data set	$I^2_{[class]}$%	$I^2_{[study]}$%	$I^2_{[trait]}$%	$I^2_{[residual]}$%	Heterogeneity (I^2)%
Means	0.09	0.06	91.61	4.27	95.73
Matching	2.86	0.25	0.21	87.19	7.45
Carry-over	0.32	22.71	46.31	23.86	74.15

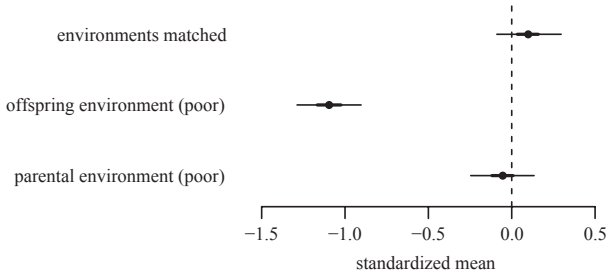

Fig. 2 Forest plot showing effect size, standard deviation (thick line) and 95% credible interval (thin line) for moderators in the model on standardized means (intercept not included).

with most of the variance being accounted for by variation among traits within studies ($I^2_{[trait]}$: 91.61%).

Our analysis of variation in Hedge's *d* showed a weak and nonsignificant positive effect of matching (β_{mean} = 0.186, HPD: −0.030, 0.393; Fig. 3a). Any effect of matching appeared to be reduced for traits measured in offspring reared in poor environments compared with those in good environments (HPD poor, relative to good: −0.273, 0.017), whereas the effect in undefined and good environments did not differ (HPD undefined, relative to good: −0.103, 0.223; Fig. 3a). None of our other moderators were important for explaining heterogeneity, although it is notable that there were stronger (albeit nonsignificant) positive effects of matching in animals than in plants (HPD animal: −0.064, 0.777) and in physiological and morphological traits compared with other trait types (Fig. 3a). There was low heterogeneity explained by the random terms considered ($I^2_{[total]}$: 7.45%, Table 2), with most of the variance being accounted for by variance within traits ($I^2_{[residual]}$: 87.19%).

We found little evidence for carry-over effects of poor maternal environments (β_{mean} = 0.023, HPD: −0.084, 0.237, Fig. 3b) in the subset of studies where the directional difference in environment quality was clear. The lack of an overall effect is quite robust, with a weaker effect when the offspring environment was also poor (HPD: −0.171, −0.023) and a marginal negative effect in adults compared with embryos and juveniles and in traits related to survival (Fig. 3b). In contrast to the matching Hedge's *d* effect sizes, there was high heterogeneity explained by the random terms ($I^2_{[total]}$: 74.15%, Table 2), which was relatively evenly accounted for by variance among studies, among traits within studies and within traits (Table 2).

In general, we did not find any evidence of publication bias as evident in the funnel plots (Fig. 4a–c). Results from the Egger's regression on both effect size models supported this finding: the value for the intercept b_0 intersected zero for both models (matching b_0: 0.073, HPD: −0.262, 0.385; carry-over b_0: 0.026, HPD: −0.198, 0.210).

Discussion

Our quantitative analyses of the empirical support for anticipatory parental effects revealed a trend for higher offspring performance when the offspring environment was matched to the maternal environment that supposedly provided the correct cue about the offspring environment. However, the effect was small and showed rather large uncertainty (i.e. wide credible interval). The overwhelming effect of offspring environment on trait expression suggests that most studies find scenarios equivalent to Fig. 1b(i) or 1b(iii) rather than Fig. 1b(ii) and 1b(iv) and that parental effects in these studies are generally small compared with the direct effects of offspring environment. Furthermore, because a positive effect of matching was stronger in high-quality environments, our results suggest that the overall experimental evidence for anticipatory parental effects (as defined in Fig. 1) is weak. Thus, despite some undeniable empirical examples (e.g. Gustafsson *et al.*, 2005; Galloway & Etterson, 2007; Fig. S1, S2), the quantitative evidence from this meta-analysis suggests that this form of adaptive plasticity may be quite rare in natural systems, that the effect is much more subtle than typically assumed or that it is common yet existing studies have failed to demonstrate its existence. Below, we discuss how well our analyses support these different scenarios and the implications this has for the evolution of parental effects.

A minimum requirement for the adaptive evolution of transgenerational plasticity is that the parental phenotype provides a source of information about future selective regimes (Shea *et al.*, 2011). It is unclear how common this situation is, although it certainly applies in environments that exhibit seasonal (e.g. winter follows autumn; Tauber *et al.*, 1986), more or less stochastic temporal (e.g. light conditions stay the same for multiple generations; Galloway, 2005) and different forms of spatial (e.g. local differences in predation and limited juvenile dispersal; Alekseev & Lampert, 2001) variation. Nevertheless, one potential reason for a failure to establish the adaptive nature of transgenerational plasticity is that studies may not have correctly identified (and manipulated) the relevant environments. Although we did not find evidence for a stronger effect in studies with quantitative data on environmental heterogeneity than those relying on verbal argument only, it is only for a fraction of study systems that a selective advantage of parental cues has been confirmed. Of the 58 studies included in this paper, only seven provided data or cited papers with data that demonstrated environmental autocorrelation between generations. Thus, the extent to which the parental phenotype is likely to provide a cue is surprisingly poorly understood in the maternal effects literature, which substantially weakens inference of adaptive function. In this context, it is interesting that the positive effect of matching was

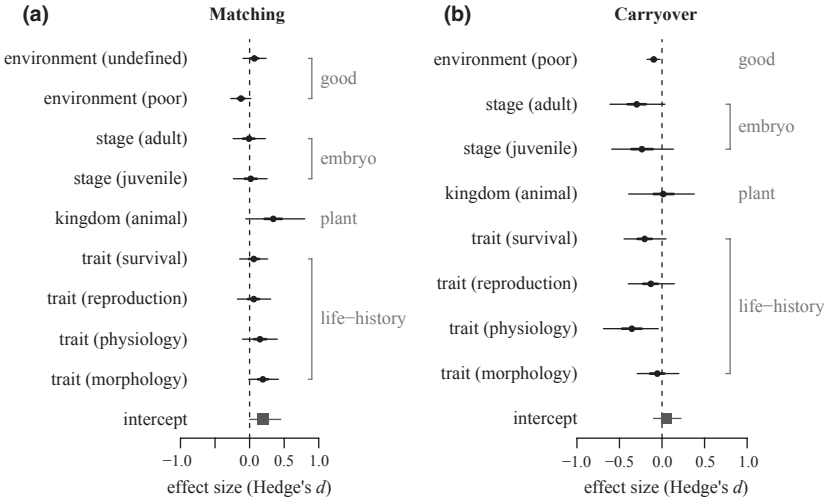

Fig. 3 Forest plot showing effect size, standard deviation (thick line) and 95% credible interval (thin line) for moderators in the models on (a) matching Hedge's *d* and (b) carry-over Hedge's *d*. The effect size and confidence interval for the intercept-only models are also provided (in grey) to indicate whether there was an overall effect. Baseline levels for each factor to which others are compared are indicated in grey on the right.

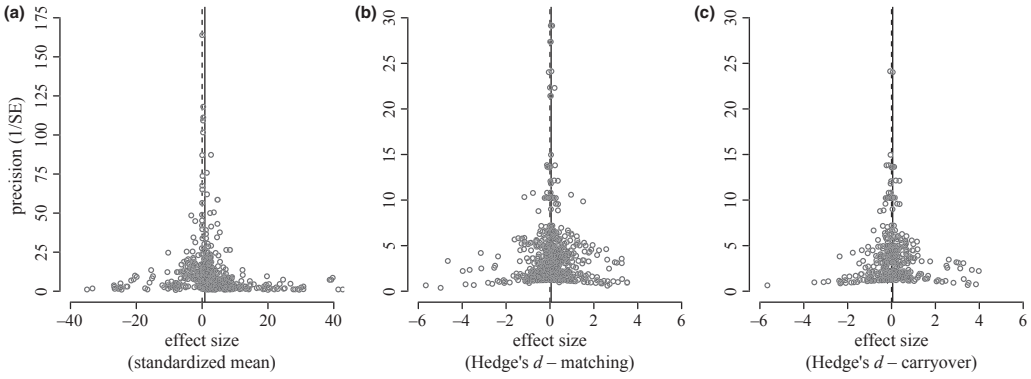

Fig. 4 Funnel plots showing effect sizes against their precision (1/s.e.) for all three analyses: (a) standardized means, (b) matching Hedge's *d* and (c) carry-over Hedge's *d*. The dashed line indicates zero effect size, and the solid line represents the overall meta-analytic mean.

generally weaker in plants than in animals, the opposite to expectations based on generalization of the spatial and temporal heterogeneity of selection across generations (largely driven by lack of mobility in plants, e.g. Herman & Sultan, 2011). Thus, we emphasize that studies that set out to test adaptive plasticity should take care to ensure that their environmental treatments reflect ecologically realistic patterns of environmental heterogeneity (as in the study of *Campanulastrum americanum,* Galloway, 2005; Galloway & Etterson, 2007; see also Doughty & Reznick, 2004). In addition, the prediction from theory is an interaction effect of parental and offspring environment on offspring phenotype, which means that it is important to ensure that sample sizes are large enough to pick up interaction effects (especially if there are large main effects).

How parents respond to environmental variation can also have substantial effects on the opportunity for anticipatory parental effects. Whereas many studies provide some general estimate of parental effects (e.g. egg size), the way in which the parental environment affects the parental phenotype is typically poorly understood. Such lack of understanding can lead to erroneous predictions. For example, if parents compensate for stressful environments, the information about offspring environment carried by the parental phenotypes may be low (e.g. Sultan, 1996; Uller *et al.*, 2011), which should limit the potential for adaptive adjustment of offspring phenotype. Instead, in noisy environments, parents may eliminate irrelevant environmental variation and magnify important cues that enable offspring to assess fitness-relevant features of their environment

(Wells, 2010). One reason we have failed to detect strong overall support for adaptive transgenerational plasticity therefore is that our data set may include systems in which parents buffer against or compensate for negative environmental conditions. A better understanding of the mechanisms underlying treatment effects on parental phenotype and subsequent offspring response would have allowed us to distinguish among these possibilities.

The evolution of adaptive responses to parental cues will also be affected by the costs of plasticity for parents and offspring, constraints on information acquisition, processing and transmission (e.g. Dewitt *et al.*, 1998) and evolutionary conflicts between parents and between parents and offspring (e.g. Uller & Pen, 2011). Anticipatory parental effects are perhaps particularly likely if direct selection on parental plasticity is strong and transmission of information to offspring does not incur additional costs. This scenario may apply to transgenerational induction of defences, which are among the more convincing case studies of adaptive maternal effects (e.g. Agrawal *et al.*, 1999). There are, however, few studies that estimate costs of parental and offspring plasticity. Furthermore, the scarcity of theoretical models that identify the conditions favouring the use of parental phenotypes rather than relying on, for example, direct environmental cues or genetically determined strategies is a limitation for identifying suitable empirical systems (including environmental contexts and traits). For example, it is possible that adaptive scenarios that involve single traits are too simplistic (Sultan, 1996; Badyaev & Oh, 2008) and that adaptive offspring plasticity may involve modulation of trade-offs, such as that between growth and reproduction (Doughty & Reznick, 2004). Studies that only measure one of those traits – or measure and interpret traits in isolation – would thus fail to establish higher fitness of matched offspring. Indeed, even for one of the most complete studies of adaptive transgenerational plasticity in plants (Galloway & Etterson, 2007), most traits did not show significant changes in the predicted direction. It was only when fitness was estimated as projected population growth rate that the adaptive significance of maternal effects was well supported. We therefore expected studies that used close proxies to fitness (e.g. survival) to show stronger effect sizes. This was not the case, however. We also failed to support the prediction that adaptive responses are more likely to involve adjustment of offspring traits early in development when offspring (e.g. embryos) should have limited ability to access cues about their external environment directly. Thus, there is no evidence that the weak overall effect is due to adaptive responses being restricted to some taxa, environments or traits.

In addition to limitations of the empirical data on which we based our analysis, there are also some important limitations of our meta-analytical approach. The calculation of effect sizes relies on accurate presentation of data, which results in omission of some studies. Unfortunately, some of the strongest candidates for adaptive transgenerational plasticity, such as maternally induced diapause in seasonal environments (Tauber *et al.*, 1986; Mousseau & Dingle, 1991) and transmission of immunity (Hasselquist & Nilsson, 2009), rarely used a fully factorial design. This lack of data for some obvious candidate systems suggests that our results may be somewhat biased towards negative conclusions regarding the strength of the overall effect. Our results provide no evidence for publication bias, however. Furthermore, our estimates of heterogeneity (I^2) suggest relatively little variation among studies in terms of the effect of matching offspring and parental environment. This implies that the effects are subtle rather than rare. In comparison, when we compared offspring from parents in good conditions with offspring from parents in poor conditions (i.e. carry-over Hedge's d), heterogeneity (I^2) was very high among traits and studies. This is expected as the studies in our data set were typically designed as specific tests of anticipatory parental effects rather than general positive or negative carry-over effects. However, the analyses of studies that contrasted good vs. bad environments only generated limited support that positive or negative carry-over effects obscure adaptive transfer of information. Thus, the available evidence is that studies commonly fail to find strong support for adaptive matching of offspring phenotype to local conditions in many of the systems where such matching has been predicted to occur. To what extent this conclusion reflects the problems outlined above remains to be seen, but we suggest that offspring responses to parental phenotype may often be more parsimoniously described as adaptive responses to parental resource allocation rather than to parental cues about future selective regimes. For example, parents may allocate more resources in environments where those resources have a substantial positive effect on offspring survival. In incomplete experimental designs, this can give the impression that offspring fitness is maximized when parental and offspring environments are matched (Marshall & Uller, 2007).

In summary, our meta-analysis suggests that the experimental evidence for anticipatory parental effects (or adaptive transgenerational plasticity) is weak. In addition to the possibility that this form of adaptive plasticity simply is rare, we identified several alternative partial explanations for the weak support within and among studies. Our results emphasize the need to verify selective regimes using data on environmental heterogeneity and the information content of parental phenotypes, the use of accurate estimates of fitness and the importance of developing theoretical models to identify robust predictions. These problems make it difficult to assess whether transgenerational plasticity is a common adaptation to heterogeneous environments.

However, the direct environmental effect on offspring phenotype was generally substantially stronger than the effect of the environment experienced by parents. We therefore tentatively suggest that the weak empirical support that parental phenotypes are used as cues in adaptive plasticity reflects that this does not occur, or because the responses are more subtle than expected, in most of the systems studied in detail. This implies that despite the attention that anticipatory parental effects have received, the current experimental evidence does not support that it is a widespread strategy in nature.

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Supporting information

Additional Supporting Information may be found in the online version of this article:

Data S1 Further details on moderator traits measured in each study.

Figure S1 Standardized mean trait values for four studies: (a) Galloway & Etterson (2007); (b) Sultan *et al.* (2009); (c) Bonduriansky & Head. (2007) and (d) Gustafsson *et al.* (2005).

Figure S2 Funnel plot indicating position of the above four studies in the context of all studies included in the meta-analysis.

Table S1 Data for all studies included in the meta-analysis.

Data deposited at Dryad: doi:10.5061/dryad.pd5ss

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