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Offspring development and life-history variation in a water flea depends upon clone-specific integration of genetic, non-genetic and environmental cues

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Summary

1. Theory predicts that offspring developmental strategies involve the integration of genetic, non-genetic and environmental 'cues'. But it is unclear how cue integration is achieved during development, and whether this pattern is general or genotype specific.

2. In order to test this, we manipulated the maternal and offspring environments of three genetically distinct clones of the water flea *Daphnia magna* taken from different populations. We then quantified the effect that the genotype, maternal environment and the offspring environment had on the development and life histories of the three different clones.

3. Mothers responded to the same maternal environments in different ways, resulting in clone-specific maternal effects on neonate size. Offspring responses to maternal cues varied according to the trait in question and were also clone specific. The integration of these maternal effects during development was highly context dependent in two clones but more consistent across environments in the third.

4. Genetic, non-genetic and environmental cues contributed to offspring phenotypic variation in all three clones, but there was no general pattern linking traits to specific cues. In fact, two clones used different combinations of cues at different points in development to achieve similar phenotypic outcomes. Reaction norms for the age and size at which maturation was initiated differed among genotypes, between maternal environments and across current environments. Developmental transitions such as the decision to mature may thus play an important role in determining patterns of cue integration.

5. Considering multiple traits during development demonstrated that variation in the integration of genetic, non-genetic and environmental cues was an important determinant of life-history variation among *D. magna* genotypes. This variation is likely to influence phenotypic evolution.

Key-words: cue integration, developmental plasticity, maternal effects, non-genetic inheritance, probabilistic maturation reaction norm, water flea

Introduction

Understanding evolutionary processes requires an understanding of where phenotypic variation comes from and how it is transmitted between generations (West-Eberhard 2003). Environment-induced phenotypic variation is increasingly recognised as an integral part of the evolutionary process because phenotypic plasticity allows populations to persist in the face of rapid environmental change and contributes to the phenotypic variation that selection operates on (Schlichting & Pigliucci 1998; West-Eberhard 2003; Moczek *et al.* 2011). Moreover, non-genetic inheritance mechanisms (see Bonduriansky & Day 2009; Danchin *et al.* 2011; Day & Bonduriansky 2011 for reviews) can transmit phenotypic plasticity and spontaneously generated phenotypic variation across generations. Plasticity and non-genetic inheritance are therefore able to alter the direction and the speed of evolution, generate phenotypic novelty, and potentially decouple phenotypic change from genotypic change altogether (West-Eberhard 2003; Galloway & Etterson 2007; Bonduriansky & Day 2009; Day & Bonduriansky 2011). The incorporation of plasticity and

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non-genetic inheritance into a more inclusive evolutionary synthesis potentially changes our assumptions about how populations evolve and respond to rapid environmental change (Bonduriansky, Crean & Day 2012; Hallsson, Chenoweth & Bonduriansky 2012), and places a much greater emphasis on understanding how genes, non-genetic inheritance and environmental variation combine to shape phenotypic evolution (Day & Bonduriansky 2011; Leimar & McNamara 2015).

The population and quantitative genetic framework that underpins our current evolutionary thinking is useful for understanding how selection and patterns of genetic variation and co-variation shape evolutionary responses (Falconer & Mackay 1996). Within this framework, non-genetically inherited effects (such as maternal effects) are modelled as a static coefficient that translates variation in maternal phenotype into variation in offspring fitness (Kirkpatrick & Lande 1989; Hoyle & Ezard 2012). While quantitative genetics models of non-genetic inheritance are now beginning to incorporate more realism (Kuijper & Hoyle 2015), and empirical studies reveal the importance of non-genetically inherited maternal effects in explaining phenotypic variation (Wilson *et al*. 2005; Nespolo *et al*. 2014), this approach neglects the dynamic nature of such effects. Maternal effects on offspring vary according to a mother's environment (Plaistow *et al*. 2007; Yanagi & Tuda 2010) and her age (Lind *et al*. 2015; Plaistow *et al*. 2015), and offspring responses to these maternal effects may change, or be constrained, in different environments (Czesak & Fox 2003; Plaistow, Lapsley & Benton 2006; Räsänen & Kruuk 2007), resulting in different phenotypic outcomes. Furthermore, the interaction between maternal and offspring environmental effects may differ between genotypes (Hallsson, Chenoweth & Bonduriansky 2012; Plaistow *et al*. 2015; Walsh *et al*. 2015). As a result, we have limited empirical understanding of how genetic, non-genetic and environmental effects interact to shape offspring phenotypes (Day & Bonduriansky 2011; Leimar & McNamara 2015).

A better understanding of how non-genetic inheritance and phenotypic plasticity interact with genetic effects is achieved by studying individual development and developmental causes of phenotypic variation (Atchley & Hall 1991; Cheverud 1996; Schlichting & Pigliucci 1998). Schlichting & Pigliucci (1995) popularised the idea that environmental cues were integrated by developing phenotypes, and in an attempt to unify development and phenotypic evolution, modelling approaches have recently appeared that consider genetic inheritance (e.g. the genotype), non-genetic inheritance (e.g. maternal effects) and environmental variation to all be cues which are integrated by developing phenotypes (Leimar, Hammerstein & Van Dooren 2006; Dall, McNamara & Leimar 2015; Leimar & McNamara 2015; McNamara *et al*. 2016). The concept of cue integration proposed by this body of work is a general one: a cue can be thought of as any signal that may be informative to a developing organism; and if the organism is able to perceive the cue, then it can be integrated into the phenotype during the developmental process. In order to test these models and assess the relative contributions of genetic, non-genetic and environmental cues on phenotypic development, we need studies that go beyond the static quantification of genetic variation in parental effects and consider phenotypic variation during the course of the developmental cycle, as all these different cues are simultaneously incorporated (Wolf *et al*. 2001; Uller 2013; Wang *et al*. 2014). This approach is essential given that non-genetic effects can influence multiple traits during development, both simultaneously and consecutively (Kaplan & Phillips 2006; Bonduriansky & Head 2007; Burgess & Marshall 2011), and potentially in conflicting ways (Marshall & Uller 2007; Cahan, Graves & Brent 2011).

Parthenogenetic organisms such as *Daphnia* are ideal models for empirical studies investigating the integration of multiple cues during development because it is easy to separate genetic and non-genetic influences, and large numbers of genetically identical individuals can be assessed simultaneously across different environments in parental and offspring generations. Furthermore, offspring development and life history can be easily assayed (Harney *et al*. 2013; Plaistow & Collin 2014; Plaistow *et al*. 2015) including the ability to partition variation in maturation decisions into genetic, maternal and environmental components (Plaistow *et al*. 2015). In order to investigate how genetic, non-genetic and environmental cues are integrated during offspring development, we manipulated the maternal resource environment of three *Daphnia magna* clones, each from a different population, and reared their offspring across a resource gradient. For the analysis, a multivariate approach is necessary because the integration of multiple traits is itself plastic, and an important component of G × E interactions (Plaistow & Collin 2014). We used a multivariate analyses of variance (MANOVA) to test the effect of genetic (clone), non-genetic (maternal food environment) and environmental (offspring food environment) cues on the development and life history of offspring. Principal component analysis (PCA) and phenotypic change vectors (PCVs: Collyer & Adams 2007; Plaistow & Collin 2014) were then used to visualise and compare changes in the influence of genetic, non-genetic and environmental cues on offspring multivariate phenotypes across the offspring environmental gradient. In a separate analysis, we used a probabilistic maturation reaction norm (PMRN) analysis (Harney *et al*. 2013) to investigate the effects that genetic, non-genetic and environmental cues had on offspring maturation decisions. We used our analyses to test: (i) whether offspring phenotypic development involves the integration of genetic, non-genetic and environmental cues; (ii) whether the relative influence of the different cues differs according the offspring environment or genotype; and finally (iii) whether the integration of genetic, non-genetic and environmental cues could be linked to plastic responses in specific developmental traits.

Materials and methods

EXPERIMENTAL ANIMALS

Our experiments were conducted on three *D. magna* clones (DKN1-3, Ness1, and B5) that originated from different sites, and that had previously displayed different reaction norms for maturation (clone origins and reaction norms are provided in Harney *et al.* 2013). The basic design of our experiment is outlined in Fig. 1. During both the conditioning and experimental periods, *D. magna* were individually reared in glass jars containing 150 mL of hard artificial pond water media (OECD 1984) enriched with a standard organic extract (Baird *et al.* 1989). Media was completely replaced every other day, and jars were housed in incubators maintained at 21 ± 1 °C with a 14 : 10 light : dark photoperiod.

In order to create (non-genetic) maternal effects, individuals from each clone were conditioned in one of two maternal environments for three generations (see Fig. 1). The jars of individuals in the high-food maternal environment (HME) received a daily food ration of 200 cells per μL of *Chlorella vulgaris*, while the jars of individuals in the low-food maternal environment (LME) treatment received a daily food ration of 40 cells per μL *C. vulgaris*. For the first (great-grand-maternal) generation a single individual was maintained in each maternal environment, and a single neonate from the third clutch was used to set up the second (grand-maternal) generation (Fig. 1). Ten neonates from the third clutch of the second generation were used to set up the third (maternal) generation. From this third (maternal) generation, 27–40 neonates (n = 212) from the third clutch of at least five individuals (that had all produced neonates within a 12 h window) were used as experimental animals. Offspring from these five or more mothers were mixed together to prevent systematic bias due to maternal ID, then randomly assigned and maintained individually in one of four offspring environment treatments, receiving food rations of 133, 59, 26 or 12 cells per μL. Each of the 5–10 replicate individuals in each offspring environment treatment were checked every day and photographed after moulting for all instars up to the deposition of eggs in the brood chamber, which was considered to be the point at which they achieved maturity (see Fig. 1).

In *D. magna*, the maturation process lasts three instars, from initial oocyte development (IM-1), through oocyte provisioning (IM-2) to the appearance of eggs in the brood chamber at primiparity (IM-3). The transparent carapace of *Daphnia* allows the assessment of these developmental stages. Body size was defined as the distance from the top of the head to the base of the tail spine and measured in mm from photos, using the image analysis software ImageJ (Rasband 1997). Age at a given developmental stage (IM-1, IM-2 and IM-3) was recorded as the number of whole days to reach that stage, and the number of instars was also an integer, representing the number of prematuration instars required to reach IM-1. Growth rates in mm per day were derived from the slope coefficients of linear regressions fitted to

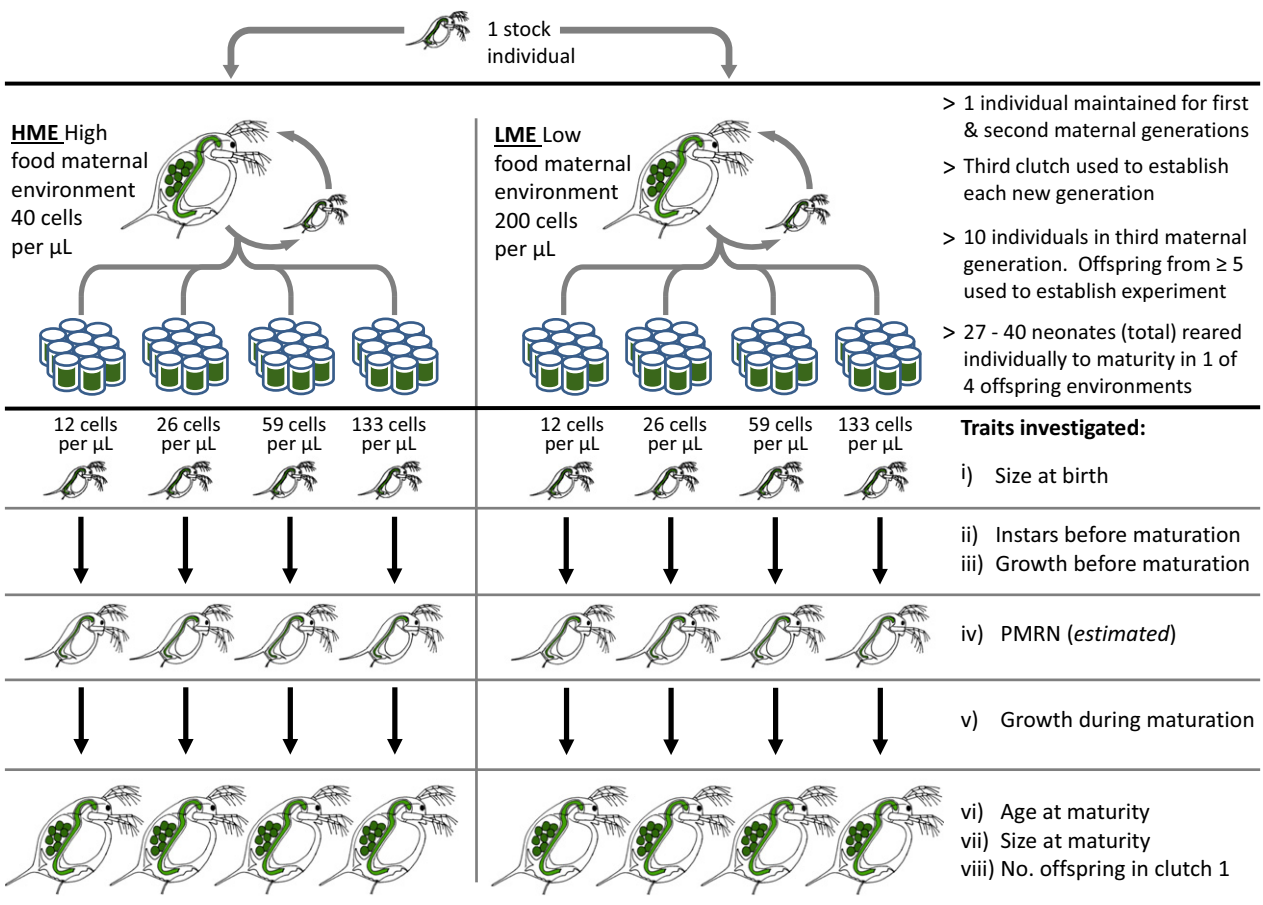

Fig. 1. Within-clone experimental design. For each of the three clones, maternal effects were generated by maintaining individuals in high or low food conditions (concentration shown is *Chlorella vulgaris* cells per μL) for three generations. In the fourth generation, 27–40 individuals from both maternal environments were randomly allocated to one of four offspring food environments: 133, 59, 26 or 12 *C. vulgaris* cells per μL). Eight traits were investigated: seven traits were directly measured, and PMRNs were estimated.

untransformed size and age data for each individual. Because maturation processes in *D. magna* may vary between clones (Harney *et al.* 2013), and because allocation of resources between competing demands of growth and maturation are likely to be influenced by both genetic and environmental cues (Glazier & Calow 1992; Hart & Bychek 2010), two separate growth rates were calculated. Early growth considered age and size values up to and including IM-1; and late growth considered age and size values inclusively between IM-1 and IM-3. In total, we directly measured seven offspring traits: (i) neonate size; (ii) early growth; (iii) the number of prematuration instars; (iv) late growth; (v) age at maturation; (vi) size at maturation; and (vii) number of first clutch offspring (Fig. 1). The probabilistic maturation reaction norms (PMRNs) were estimated, using information about age and size in the instar prior to IM-1, together with age and size at IM-1.

STATISTICAL ANALYSES I: ANOVAS AND MANOVA

Because neonate size could only have been influenced by clone (B5, DKN1-3, Ness1) and maternal environment (HME, LME), it was analysed independently using analysis of variance (ANOVA) with neonate size as a response variable and clone and maternal environment as factors (fixed effects). For the six remaining directly measured traits (number of prematuration instars, early growth, late growth, age at maturity, size at maturity and number of first clutch offspring), clone (B5, DKN1-3, Ness1), maternal environment (HME, LME) and offspring food environment (133, 59, 26 and 12 cells per μL) were considered as fixed effects and their significance was tested for using multivariate analysis of variance (MANOVA). The number of prematuration instars was rank transformed, while early growth, late growth, age at maturity and number of first clutch offspring were log-transformed prior to analysis in order to ensure normality. To further investigate interactions between fixed effects, ANOVAs and generalised linear models were used as appropriate to investigate effects of these factors at individual trait level. For each ANOVA/GLM model, selection based on Akaike's Information Criterion (AIC) was used to remove non-significant interactions and effects. In cases where interactions with maternal environment effects were maintained, false discovery rate (FDR) corrected post hoc tests were then carried out with the fixed effects clone and/or offspring environment held constant (ANOVA, GLM and post hoc test results are presented in the Supporting Information). ANOVA, MANOVA and GLM analysis were performed, using the STATS package of the R language for statistical computing (R Development Core Team 2014) with the packages MASS (Venables & Ripley 2002) and HMISC (Harrell 2013) loaded, and post hoc tests were carried out with the *testInteractions* function in the PHIA package (De Rosario-Martinez 2015) with the package CAR loaded (Fox & Weisberg 2011).

STATISTICAL ANALYSES II: PRINCIPAL COMPONENT ANALYSIS AND PHENOTYPIC CHANGE VECTORS

A 'two-state multivariate' analysis similar to that of Plaistow & Collin (2014) was used to visualise and compare changes in the influence of maternal environment, offspring environment and clone cues on offspring phenotypes. This approach consisted of carrying out: (i) principal component analysis (PCA) to identify patterns of covariation between traits, and visualise the effect of clone, maternal environments and offspring environments on offspring phenotypes, and (ii) phenotypic change vector (PCV) analysis (Collyer & Adams 2007), to compare the effect that maternal environment had on offspring phenotypes for each clone in each offspring environment. PCA and PCVs were calculated from all directly measured offspring traits using data scaled to unit variance (estimates of PMRNs are unsuitable for incorporation into multivariate analyses).

Following PCA, co-variation among traits was identified from high loadings onto the same principal component. Multivariate clone × maternal environment × offspring environment means were projected onto a plot of the first two principal components in order to aid interpretation of the statistical results. Maternal environment PCVs were defined as multivariate mean vectors: $\Delta\bar{Y} = \Delta\bar{Y}_{ij} - \Delta\bar{Y}_{ik}$ for treatment group i (maternal environment) in environments j and k (e.g. offspring food environments of 133 and 59 cells per μL for a given clone). The magnitude of the vector is calculated as the Euclidean distance: $D_E = ||\Delta\bar{Y}_i|| = (\Delta\bar{Y}_i\Delta\bar{Y}_i^T)^{1/2}$, where T represents a matrix transpose. To test for significant differences in the magnitude of maternal environment effects in different environments, the test statistic: $|D_{E1} - D_{E2}|$ is calculated, i.e. the difference in the lengths of PCVs (Adams & Rohlf 2000). To test for significant differences in the nature of maternal environment effects, differences in the angle of PCVs were calculated. For pairs of PCVs their correlation is calculated as the inner product of the two vectors scaled to unit length (Schluter 1996; Bégin & Roff 2003):

$$\mathrm{VC} = \left(\frac{\Delta\bar{Y}_1}{D_{E1}} \cdot \frac{\Delta\bar{Y}_2^T}{D_{E2}}\right)$$

The arccosine of this value is the angle between the two vectors: θ, which describes the similarity of their direction. The significance of these two test statistics (ΔD for magnitude and θ for direction) was then calculated by comparing them to distributions of random vector pairs generated, using a permutation procedure. A comparison of the difference in length (ΔD) and/or angle (θ) of maternal environment PCVs between any two clones for a given offspring environment was used to statistically test for clonal variation in the effect of maternal environment, and a comparison of ΔD and/or θ for the same clone in any two offspring environments was used to statistically test whether maternal effects were context dependent or not. A full description of PCVs can be found in Collyer & Adams (2007), and the application of this approach to *Daphnia* life-history data is described in Plaistow & Collin (2014). The STATS package of R was used for both analyses (R Development Core Team 2014), and PCA was carried out, using the FACTOMINER package (Lê, Josse & Husson 2008).

STATISTICAL ANALYSES III: PROBABILISTIC MATURATION REACTION NORMS

The effect of clone, maternal environment and offspring environment on maturation decisions was tested for using a PMRN approach (Van Dooren, Tully & Ferrière 2005), which explicitly incorporates the fact that maturation decisions are probabilistic rather than deterministic. A thorough explanation of the methodology and its application to *Daphnia* can be found in Harney *et al.* (2013), but briefly: PMRNs were modelled by means of logit-link binomial generalised linear models (Lindsey & Ryan 1998; Collett 2003). These models can include the logarithm of the interval duration as an offset to account for variation in observation intervals, as well as the age and size covariates and categorical variables. Data from all offspring environments was included in PMRN analysis, although offspring environment was not considered as a variable per se, as variation in this factor is necessary to produce the diverse growth trajectories that enable PMRN estimation. As in Harney *et al.* (2013), the importance of different offsets was considered (age, size and none); backwards stepwise term deletion was used to test the importance of interactions between factors (clone and maternal environment) and covariates (age and size); and age and size were modelled as either interval start-,

mid-, or end-points using log-transformed or untransformed values. Likelihood ratio tests (LRTs) were used to compare nested models, and AIC or likelihood comparisons were used to compare non-nested models. PMRNs were visualised by simulating growth curves and calculating probabilities per growth curve based on the best-fitting generalised linear model. Interpolated 25th, 50th and 75th percentiles were then superimposed onto real growth data (Van Dooren, Tully & Ferrière 2005). By overlaying different maternal environment PMRNs (HME, LME) onto the same plot, the effect of the maternal environment can be visualised. When PMRNs of the two maternal environments do not overlap, maturation decisions are occurring at different ages and sizes. PMRN analyses were carried out in R (R Development Core Team 2014), and the packages MASS (Venables & Ripley 2002) and NLME (Pinheiro *et al.* 2016) were used to prepare plots.

Results

MATERNAL EFFECTS ON NEONATE SIZE

Neonate size was influenced by the maternal environment in a clone-dependent manner (ANOVA: maternal environment × clone interaction: $F = 89{\cdot}43$, d.f. = 2, $P < 0{\cdot}001$; see Table 1, Fig. 2). Post hoc tests (see Table S1, Supporting Information) revealed that neonates of clone Ness1 and DKN1-3 born to mothers from the low food maternal environment (LME) were significantly larger than those born to mothers from the high food maternal environment (HME). However, in clone B5, the maternal environment did not significantly influence neonate size.

Table 1. Results of univariate analysis showing the effects of clone, maternal environment (ME) and their interaction on neonate size in *Daphnia magna*

Factors	d.f.	SS	*F*-value	*P*-value	% variance
Clone	2	0·076	67·95	<0·0001	21·45
ME	1	0·064	114·84	<0·0001	18·12
Clone × ME	2	0·098	88·43	<0·0001	27·91
Residual	206	0·115			32·51

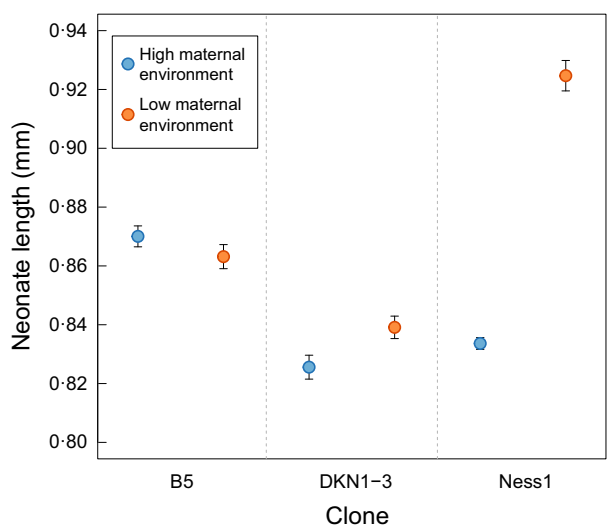

Fig. 2. Mean neonate size of clones B5, DKN1-3 and Ness1 from mothers reared in either high or low food maternal environments. Error bars are standard errors.

INTERACTIONS OF ENVIRONMENTAL, NON-GENETIC AND GENETIC CUES

Variation in offspring phenotypes was dependent on the three-way interaction between the fixed effects clone, maternal environment, and offspring environment, (MANOVA results, Table 2). Even when considered as individual traits, the three-way interaction between maternal environment, clone and offspring environment remained significant for all traits (ANOVA and GLM results, Table S2). Thus, for the three clones considered in this study, maternal effects interacted with the offspring environment to shape the developing phenotype, and this interaction differed between each of the clones. Plots of univariate reaction norms across the offspring food environmental gradient are provided for all traits after neonate size in the supplementary material (Figs S1–S6), as are post hoc tests of the significance of maternal environment effects for given combinations of clone and offspring environment (Table S3). The context dependence and clone-specific nature of maternal environment effects can be seen in key examples where reaction norms cross over for size or age at maturity. For example, in clone B5, LME progeny are significantly larger than HME progeny in high offspring food (133 cells per μL), but significantly smaller in lower offspring foods (26 and 12 cells per μL, Fig. S5A; Table S3); and in DKN1-3, LME progeny are significantly older at maturity than HME progeny in higher offspring foods (133 and 59 cells per μL), but significantly younger at maturity in low offspring food (12) (see Fig. S4B; Table S3).

CORRELATIONS AMONG DEVELOPMENTAL TRAITS

All traits correlated strongly with one another in the PCA, with the exception of neonate size (Table 3; Fig. 3a). Individuals with high PC1 scores had higher early and late

Table 2. MANOVA summary statistics showing the effect of clone, maternal environment (ME) and current food environment (CE) on trait variation in the traits neonate size, number of instars before the maturation decision, growth before and after the maturation decision, age and size at maturity and the number of offspring in the first clutch

Factors	d.f.	Wilks λ	*F*	*P*
Clone	2	0·09	69·44	<0·0001
ME	1	0·41	44·07	<0·0001
Food	3	0·04	60·42	<0·0001
Clone × ME	2	0·27	28·41	<0·0001
Clone × Food	6	0·32	6·54	<0·0001
ME × Food	3	0·64	4·93	<0·0001
Clone × ME × Food	6	0·25	8·37	<0·0001
Residuals	188			

Table 3. Factor loadings for seven life-history traits following principal component analysis of three *Daphnia magna* clones in two maternal food environments and four current food environments. Only the first five principal components (PCs) are shown. For PCs 1 and 2 loadings with values >0·65 are in bold to emphasize strong correlations between PCs and traits

	Loadings				
Variables	PC1 63·56%	PC2 18·46%	PC3 9·17%	PC4 5·61%	PC5 1·61%
Neonate size	0·360	**−0·762**	0·525	−0·095	0·065
Number of instars	**−0·828**	0·461	0·234	0·097	0·140
Prematuration growth	**0·821**	0·367	−0·081	−0·397	0·111
During maturation growth	**0·857**	−0·120	−0·182	0·439	0·134
Age at maturity	**−0·911**	0·208	0·299	0·087	−0·014
Size at maturity	**0·806**	0·443	0·340	0·056	0·087
Offspring in clutch 1	**0·864**	0·334	0·261	0·114	−0·226

growth rates, larger size at maturity and more offspring in their first clutches (all PC1 loadings >0·8) and typically matured with fewer instars and at younger ages (PC1 loadings <−0·8, see Table 3, Fig. 3a). The variation in PC1 scores was primarily explained by the effect of offspring food environment and clonal variation in offspring traits. PC1 scores increased as offspring food levels increased (see Fig. 3b,d). Clone DKN1-3 typically had low PC1 scores (Fig. 3c), clone B5 had intermediate scores (Fig. 3b) and clone Ness had high PC1 scores (Fig. 3d). PC1 explained more than 63% of offspring phenotypic variation, while PC2 explained just 18%, and only one trait (neonate size) correlated with it strongly (loading = −0·762). PC2 related to maternal environment effects, especially in clone Ness 1 (Fig. 3d). LME offspring were typically larger neonates, and weak correlations with number of instars prior to maturation and body size at maturity (PC2 loadings >0·4), suggested that LME offspring were, to some extent, initiating maturation after fewer instars and reaching a smaller size at maturity. However, as demonstrated by the differences in neonate size (Fig. 2) and the PCV analysis carried out below (Fig. 3), the overall picture of maternal environment effects is that they were clone-specific and context dependent.

VARIATION IN CUE INTEGRATION AMONG CLONES

Comparison of the length of PCVs for different pairs of clones demonstrated that the magnitude of maternal environment effects was significantly larger in clone Ness1 than the other two clones in all offspring environments (Table 4, Fig. 3). In contrast, maternal environment effects in clones B5 and DKN1-3 were of similar magnitude, except when offspring food was 59 cells per μL (Table 4, Fig. 3). Comparison of angles of PCVs for different pairs of clones demonstrated that the nature of the maternal environment effect in clone Ness1 was significantly different from both of the other clones in high offspring food environments (133 and 59 cells per μL) and different from clone B5 in low offspring food environments (26 and 12 cells per μL; Table 4, Fig. 3). The nature of the maternal environment effect was statistically indistinguishable for clones B5 and DKN1-3 in high offspring food environments (133 and 59 cells per μL; Table 4), where HME offspring had similar or higher PC1 scores and lower PC2 scores than LME offspring. In contrast, in low offspring food environments (26 and 12 cells per μL), there was a significant difference in the angle of PCVs between clone B5 and clone DKN1-3 (Table 4). At these lower offspring food levels, PC1 scores in clone B5 were higher in HME offspring than in LME offspring, while in clone DKN1-3, PC1 scores were higher for LME offspring than HME offspring.

VARIATION IN CUE INTEGRATION ACROSS OFFSPRING ENVIRONMENTS

The integration of maternal environmental cues by the developing phenotype was dependent not only on clonal differences but also offspring environmental differences (Table 5). Indeed, in all three clones, the PCVs for at 133 cells per μL were significantly larger in magnitude than the PCVs at 26 cells per μL; and in clone B5, PCVs at 133 cells per μL were larger than the PCVs for all other foods (Fig. 3b). This suggests that maternal effects tended to be stronger when the offspring food level was the highest, particularly in clone B5. While all clones showed some context dependence in the magnitude of maternal environment PCVs, not all clones showed context dependence in their direction. In clone Ness1, the direction of maternal environment PCVs was consistent across offspring environments (Table 5). As can be seen in Fig. 3d, for a given offspring food environment, PC1 scores did not vary much between maternal environments, and PC2 scores were always lower in LME offspring than HME offspring, reflecting the strong effect that maternal food environment had on neonate size in this clone (Fig. 2). In contrast, maternal environment effects on neonate size were much weaker in clones B5 and DKN1-3 and resulted in maternal environment effects that were more context dependent (Table 5). These directional differences can be visualised via the variation in PC1 scores. In clone B5, the difference between PC1 values for HME and LME offspring became more marked as offspring food declined (Fig. 3b) with lower values in LME offspring indicative of older age and smaller size at maturity (Figs S4A and S5A). Conversely, PC1 values in clone DKN1-3 covered a wider range in HME offspring compared to LME offspring (Fig. 3c): HME offspring had higher PC1 values in when they experienced high food availability (133 and 59 cells per μL), and lower PC1 values when they experienced low food availability (26 and 12 cells per μL), suggesting HME

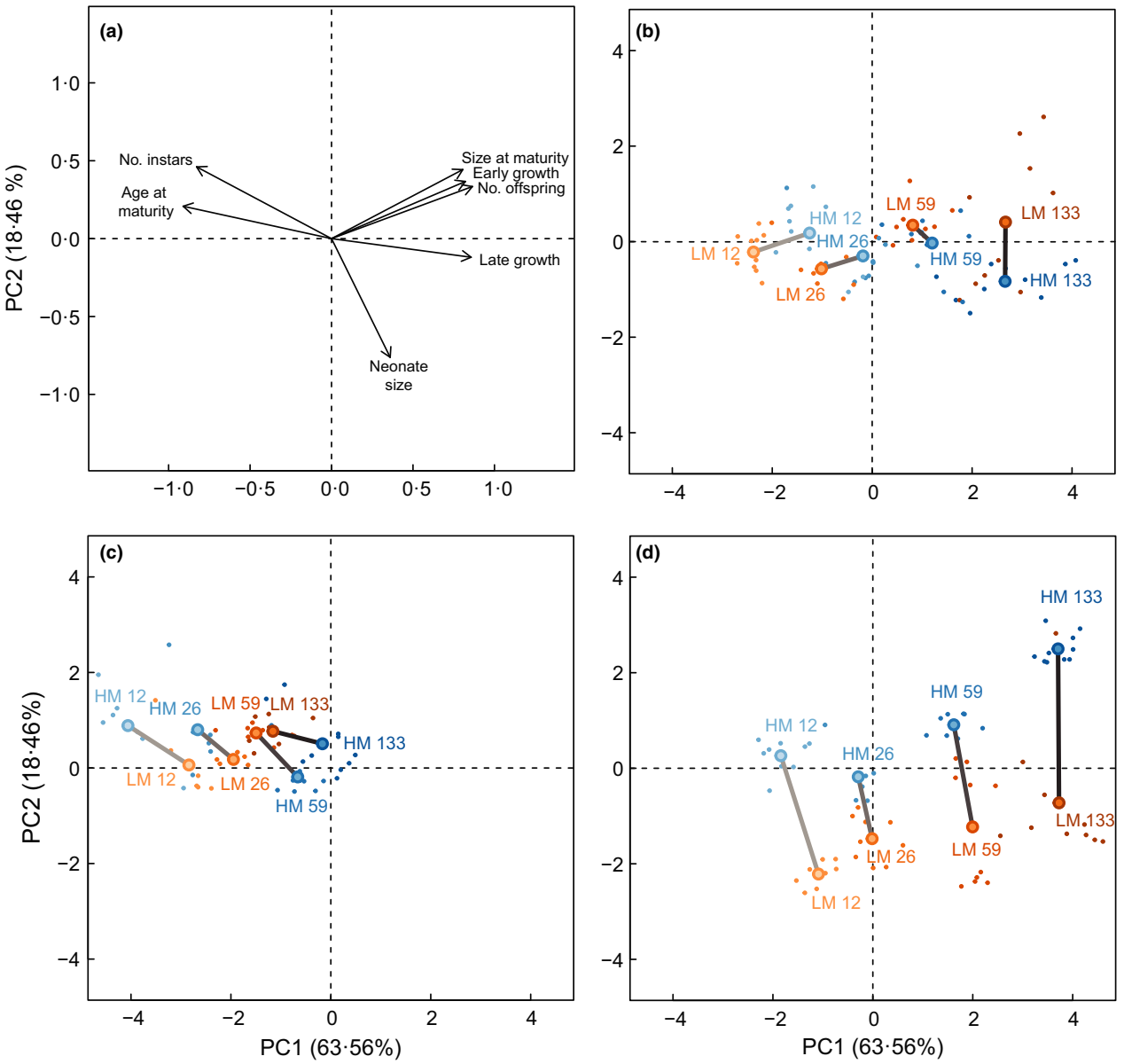

Fig. 3. Principal component analysis results and visualisation of phenotypic change vectors (PCVs). (a) Vector plot showing trait loadings for principal components 1 and 2. Negatively correlated vectors point in opposite directions; uncorrelated vectors are perpendicular; and arrow lengths indicate the amount of variation associated with a vector. Loadings are expressed as values between −1 and 1. Individual and mean co-ordinate values of PC1 and PC2 for each maternal and offspring environment combination are shown for B5 (b), DKN1-3 (c) and Ness1 (d). Values range from −5 to 5. Maternal environment is designated as either HM (high food) or LM (low food) and offspring environments range from 133 (highest food) to 12 (lowest food). Grey lines join multivariate means of individuals from the same offspring environment, but different maternal environments. These lines provide a two-dimensional projection of the phenotypic change vectors.

offspring achieved more rapid maturity when food was abundant, but suffered from slower maturation when food was scarce (Fig. S4B).

MATURATION DECISIONS

Probabilistic maturation reaction norm (PMRN) analysis revealed that the maternal environment influenced the decision to mature via an interaction with size (size × maternal environment interaction: LRT = 20·608, d.f. = 1, $P < 0·001$), and that these maternal effects were clone-dependent (clone × maternal environment interaction: LRT = 20·873, d.f. = 2, $P < 0·001$). The maternal environment influenced the decision to mature by changing the size at which the maturation decision occurred in clone B5 (Fig. 4a) and Ness1 (Fig. 4c). In clone B5, this effect was context dependent: in low offspring food, LME progeny initiated maturation at smaller sizes and older ages than HME progeny, but in high offspring food, the PMRNs overlapped, suggesting that maturation decisions occurred at similar ages and sizes. In Ness1, the effect was not context dependent, and LME progeny always initiated maturation

Table 4. Pairwise phenotypic change vectors between clonal genotypes for the effect of maternal environment in *Daphnia magna*, with current environment held constant

Current food environment	Phenotypic change vector comparison	Euclidian distance $D_{HME,\ LME}$	$D_{P\text{-value}}$	Angle $\Theta_{HME,LME}$	$\Theta_{P\text{-value}}$
133 cells per µL	B5 – DKN1-3	0·001	0·999	44·35	0·430
	B5 – Ness1	1·350	0·002**	118·54	<0·001***
	DKN1-3 – Ness1	1·351	0·002**	88·86	0·013*
59 cells per µL	B5 – DKN1-3	1·243	0·006**	37·37	0·597
	B5 – Ness1	2·244	<0·001***	115·00	0·001***
	DKN1-3 – Ness1	1·002	0·026*	90·61	0·015*
26 cells per µL	B5 – DKN1-3	0·188	0·691	123·79	<0·001***
	B5 – Ness1	1·447	0·002**	103·35	0·002**
	DKN1-3 – Ness1	1·259	0·012*	57·82	0·297
12 cells per µL	B5 – DKN1-3	0·391	0·389	124·66	<0·001***
	B5 – Ness1	1·746	<0·001***	89·66	0·010*
	DKN1-3 – Ness1	1·355	0·004**	64·78	0·157

Asterisks denote significant differences $*P < 0{\cdot}05$, $**P < 0{\cdot}01$, $***P < 0{\cdot}001$.

Table 5. Pairwise phenotypic change vectors between current environments (cells per µL) for the effect of maternal environment in *Daphnia magna*, with clonal genotype held constant

Clone	Phenotypic change vector comparison	Euclidian distance $D_{HME,\ LME}$	$D_{P\text{-value}}$	Angle $\Theta_{HME,LME}$	$\Theta_{P\text{-value}}$
B5	133 – 59	1·570	<0·001***	53·63	0·225
	133 – 26	1·175	0·005**	112·24	0·001***
	133 – 12	0·848	0·039*	90·14	0·008**
	59 – 26	0·395	0·345	66·73	0·086
	59 – 12	0·722	0·079	61·68	0·124
	26 – 12	0·327	0·427	35·08	0·624
DKN1-3	133 – 59	0·326	0·462	25·28	0·880
	133 – 26	0·986	0·040*	83·68	0·050
	133 – 12	0·456	0·325	124·74	<0·001***
	59 – 26	0·659	0·176	100·01	0·012*
	59 – 12	0·130	0·777	139·36	<0·001***
	26 – 12	0·530	0·285	50·34	0·442
Ness1	133 – 59	0·676	0·107	44·58	0·419
	133 – 26	1·077	0·014*	71·89	0·069
	133 – 12	0·452	0·285	53·32	0·255
	59 – 26	0·402	0·357	36·85	0·624
	59 – 12	0·224	0·602	35·13	0·649
	26 – 12	0·625	0·152	30·77	0·763

Asterisks denote significant differences $*P < 0{\cdot}05$, $**P < 0{\cdot}01$, $***P < 0{\cdot}001$.

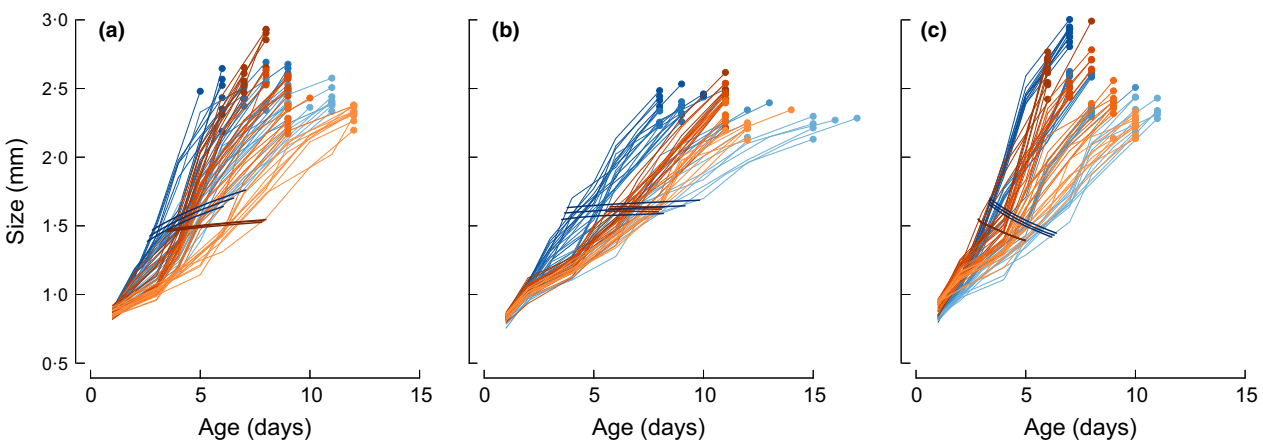

Fig. 4. Growth trajectories (thin lines) and probabilistic maturation reaction norms (PMRNs; thicker lines) showing effects of maternal environment on the maturation decisions of clones B5 (a); DKN1-3 (b); and Ness1 (c). HME growth trajectories and PMRNs are shown in shades of blue, LME growth trajectories and PMRNs are shown in shades of orange. The highest offspring food (133 cells per µL) growth trajectories are signified by the darkest points and lines. For each PMRN, the three dark horizontal lines represent the 25, 50 and 75% PMRNs. In some cases, low variation in PMRNs causes them to overlap one another.

at a smaller size than HME progeny. In clone DKN1-3, PMRN percentiles for HME and LME progeny always overlapped, and maternal environment did not influence the size at which the maturation decision occurred (Fig. 4b). However, in all three clones, reaction norms percentiles for LME progeny were closer together, indicating that there was less variation in the age and size at which maturation decisions occurred. The two-way interaction between clone and age was also required to explain variation in PMRNs (LRT = 32·782, d.f. = 2, $P < 0{\cdot}001$), suggesting that the main difference among clones arose due to the age at which maturation decisions occurred. A comparison of different models for estimating the PMRN is provided in the supplementary material (Table S4).

Discussion

These results demonstrate that development in *D. magna* integrates information from genetic, maternal environmental and offspring environmental cues and that the maternal environment contributes significantly to phenotypic variation at all stages of ontogeny. The environmental cues experienced by the maternal generations led to very different patterns of neonate size among clones, which in turn resulted in highly variable effects of maternal environment later in offspring development. In some cases, these non-genetic maternal effects were consistent across offspring environments, but they were also frequently context dependent, i.e. they influenced development in a way that depended on the environment being experienced by the offspring (sensu Plaistow, Lapsley & Benton 2006). Genotypic variation in the integration of these cues suggests that these developmental strategies may be able to evolve.

Neonate size was dependent on whether maternal generations were reared in high or low resource environments. Maternal effects on neonate size can be considered as a form of plasticity in maternal provisioning of offspring (Bernardo 1996; Mousseau & Fox 1998), and these results confirm the findings of Glazier (1992), showing that the maternal environment influences offspring size in *Daphnia*, and that these effects vary among genotypes. For clones DKN1-3 and Ness1, neonates born to mothers reared in the low food maternal environment (LME) were significantly larger than neonates born to mothers reared in the high food maternal environment (HME). This effect was particularly marked in Ness1, where LME offspring were more than 10% larger than HME offspring. On the other hand, neonate size in clone B5 did not differ significantly between maternal treatments. Genotypic variation in the environmental sensitivity of neonate size, together with context-dependent shifts in how mothers provision their offspring (Plaistow *et al.* 2007; Yanagi & Tuda 2010), highlight how dynamic this trait can be. Methods for modelling the evolution of maternal effects do not currently consider the effects of plasticity in maternal provisioning of offspring (Kirkpatrick & Lande 1989; Falconer & Mackay 1996; Hoyle & Ezard 2012), despite the fact that evidence for such plasticity is widespread (Marshall & Uller 2007; Räsänen & Kruuk 2007) and may be integral to understanding the evolution and adaptive significance of maternal effects, and more generally, non-genetic inheritance (Plaistow *et al.* 2007).

Maternal effects continued to play an important role in shaping phenotypes throughout development. Even when differences in initial size were not significant (clone B5), the environmental conditions experienced by the offspring interacted with maternal environmental cues, leading to context-dependent expression of maternal effects throughout ontogeny. Growth rate was frequently lower in LME progeny (e.g. Fig. S2), perhaps reflecting macronutrient limitation (Frost *et al.* 2010), although in clone DKN1-3, LME progeny experiencing low food (26 and 12 cells per μL) were able to grow faster during maturation than HME progeny in these environments (Fig. S3B). This result fits with the idea that matching between maternal and offspring environments may provide a fitness advantage to the offspring (Monaghan 2008; Bateson, Gluckman & Hanson 2014), even when the environment being matched is stressful (Salinas & Munch 2012). Our results confirm that maternal effects are able to influence maturation thresholds (Plaistow *et al.* 2015) and age at maturity (Walsh *et al.* 2015) in daphniids. Taken with evidence from other species where developmental transitions are known to respond to non-genetically inherited effects (Michimae *et al.* 2009; Harvey & Orbidans 2011), these results suggest that developmental decisions could be an important mechanism controlling the integration of genetic and environmental cues during development.

The interaction between maternal and offspring environmental effects resulted in considerable variation in age and size at maturity among the three *D. magna* clones. In some cases, this seemed to be adaptive, but in other cases the role of maternal effects was unclear. There was evidence for adaptive maternal effects in clone DKN1-3, where age at maturity was reduced when maternal and offspring environments matched (Fig. S4B); yet antagonistic effects were observed in clone B5, where size at maturity increased when maternal and offspring environments mismatched (Fig. S5A), and maternal effects canalised offspring age and size at maturity in clone Ness1 irrespective of the offspring environment (Figs S4C and S5C). Thus, cue integration is not only dependent on environmental influences but is also genetically variable (this study; Vijendravarma & Kawecki 2015). Variation in cue integration suggests that the reliability of genetic, environmental and maternal environmental signals with respect to food availability differs between the clones, and is likely to reflect the environments in which these clones evolved. In zooplankton such as *D. magna*, developmental plasticity of age, size and growth traits in response to variation in food quantity and quality (which would be suggestive of variation in cue integration) is frequently observed (Hart & Bychek 2010). Among different clones of *D. magna*, such variation could relate to how clones exploit food resources: for example,

power-efficiency trade-offs (Tessier, Leibold & Tsao 2000; Hall *et al*. 2012) may favour larger or faster growing genotypes when resources are abundant, and smaller or slower growing individuals when resources are scarce. Such trade-offs may reflect genetic variation (e.g. Weider 1985; Nix & Jenkins 2000; De Bie *et al*. 2012), but also non-genetically inherited differences: maternal effects may allow *D. magna* that hatch from ephippial resting eggs to grow faster and exploit the abundant food resource of algal spring blooms better than females born through parthenogenesis (Arbačiauskas & Lampert 2003); conversely, different maternal environmental cues associated with higher population density are thought to confer starvation resistance to parthenogenetic females (Cleuvers, Goser & Ratte 1997). Other than location, we have no information about the habitats that our three different clones were sampled from, and since we only used a single clone from each of the three populations, any comment about whether cue integration is locally adapted is premature. However, Walsh *et al*. (2016) demonstrated that studies which consider populations with clearly defined ecological differences (and multiple genotypes within each of these populations) can reveal local adaptation in transgenerational responses.

The complexity of cue integration during development may help to explain why evidence for adaptive maternal effects remains weak (Uller, Nakagawa & English 2013). While matching environments across generations can produce adaptive or anticipatory maternal effects (Galloway & Etterson 2007; Sultan, Barton & Wilczek 2009), maternal effects are frequently subtle, either providing no direct benefit to the offspring (Hafer *et al*. 2011), or resulting in a mixture of seemingly adaptive and maladaptive effects (Vijendravarma, Narasimha & Kawecki 2010). Their adaptive potential may remain elusive because their expression is dependent on both the environments in which they evolved and in which they are expressed. To test the adaptive nature of maternal effects, such effects should be further studied in their ecological context. Leimar & McNamara (2015) propose that non-genetic cues such as maternal effects will be stronger than within-generation phenotypic plasticity when mothers can predict the environment that their offspring will encounter. The findings of Walsh *et al*. (2016) support this prediction, as *Daphnia ambigua* clones from lakes with constant predation pressure displayed stronger maternal effects in response to predator cues than those from lakes where predation was seasonal. *Daphnia ambigua* in lakes with seasonal predation instead showed a greater degree of within-generation phenotypic plasticity in response to predator cues.

In our study, LME offspring of clone DKN1-3 raised under conditions of low food availability increased their growth rate during maturation (Fig. S3B) to reduce age at maturity relative to HME offspring (Fig. S4B). On the other hand, LME offspring of clone Ness1 raised under conditions of high food availability initiated maturation at a smaller size (Fig. 4c) to achieve a similar effect (reduced age at maturity; Fig. S4C). This suggests that potentially adaptive patterns of cue integration in *D. magna* are not linked to a particular developmental mechanism, and emphasises the importance of investigating integration over the course of ontogeny. Thus, dynamic cue integration during development allows phenotypes that are facing similar problems (such as minimising age at maturity) to develop multiple solutions (either increasing growth rate or reducing the size at which maturation is initiated). This finding supports the ideas that phenotypes are evolving in a rugged adaptive landscape, rather than converging on a single 'adaptive peak' (West-Eberhard 2003; Pfennig *et al*. 2010; Vijendravarma & Kawecki 2015). Variation in cue integration during ontogeny is likely to be facilitated by the modular nature of development (West-Eberhard 2003), especially if different developmental or life stages face dissimilar selective pressures. Developmental transitions therefore represent key periods in ontogeny, compartmentalising the phenotype into these modules which may respond to contrasting selective pressures by integrating genetic, non-genetic and offspring environmental cues in different ways (Schlichting & Pigliucci 1995; West-Eberhard 2003). Increasing evidence suggests that developmental transitions themselves are sensitive to non-genetic effects (Schwander *et al*. 2008; Michimae *et al*. 2009; Harvey & Orbidans 2011). Non-genetic effects in the timing of these transitions could represent a form of 'developmental niche construction', whereby parents alter the environment that their offspring experiences and the phenotype it expresses (Donohue 2014). Although maturation decisions did not respond to non-genetic effects in all clones, our identification of genotypic variation in the sensitivity of these transitions to maternal and environmental cues does suggest that they could play an important role in the evolution of phenotypic diversity (Badyaev 2008), and lead to the evolution of adaptive transgenerational responses (Galloway & Etterson 2007; Herman & Sultan 2011). Overall, our results highlight the value of taking a holistic view of ontogeny, showing that the diversity of interactions between genetic, non-genetic and environmental cues is essential for explaining adult phenotypes and is likely to be an important underlying factor in phenotypic evolution.

Authors' contributions

All authors were involved in the design of the experiments. E.H. collected the data, and E.H. and S.J.P. carried out the data analysis. E.H. wrote the first draft of the paper, and all authors contributed to subsequent drafts and revisions.

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Data accessibility

Data available from the Dryad Digital Repository: https://doi.org/10.5061/dryad.mn7k4 (Harney, Paterson & Plaistow 2017).

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Supporting Information

Details of electronic Supporting Information are provided below.

Fig. S1. The number of instars occurring before maturation in three clones of *Daphnia magna*.
Fig. S2. Average growth rate before maturation in three clones of *Daphnia magna*.
Fig. S3. Average growth rate during maturation in three clones of *Daphnia magna*.
Fig. S4. Age at maturity in three clones of *Daphnia magna*.
Fig. S5. Size at maturity in three clones of *Daphnia magna*.
Fig. S6. Number of offspring in the first clutch for three clones of *Daphnia magna*.
Table S1. Post hoc interaction analysis of maternal effects on neonate size.
Table S2. Results of univariate analysis showing the effects of clone, maternal environment and current food environment on six developmental traits in *Daphnia magna*.
Table S3. Post hoc interaction analysis of maternal effects on six developmental traits.
Table S4. Binomial GLMs for PMRN analysis.

