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SHORT COMMUNICATION

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When mother knows best: A population genetic model of transgenerational versus intragenerational plasticity

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Abstract

Many organisms exhibit phenotypic plasticity; producing alternate phenotypes depending on the environment. Individuals can be plastic (intragenerational or direct plasticity), wherein individuals of the same genotype produce different phenotypes in response to the environments they experience. Alternatively, an individual's phenotype may be under the control of its parents, usually the mother (transgenerational or indirect plasticity), so that mother's genotype determines the phenotype produced by a given genotype of her offspring. Under what conditions does plasticity evolve to have intragenerational as opposed to transgenerational genetic control? To explore this question, we present a population genetic model for the evolution of transgenerational and intragenerational plasticity. We hypothesize that the capacity for plasticity incurs a fitness cost, which is borne either by the individual developing the plastic phenotype or by its mother. We also hypothesize that individuals are imperfect predictors of future environments and their capacity for plasticity can lead them occasionally to make a low-fitness phenotype for a particular environment. When the cost, benefit and error parameters are equal, we show that there is no evolutionary advantage to intragenerational over transgenerational plasticity, although the rate of evolution of transgenerational plasticity is half the rate for intragenerational plasticity, as predicted by theory on indirect genetic effects. We find that transgenerational plasticity evolves when mothers are better predictors of future environments than offspring or when the fitness cost of the capacity for plasticity is more readily borne by a mother than by her developing offspring. We discuss different natural systems with either direct intragenerational plasticity or indirect transgenerational plasticity and find a pattern qualitatively in accord with the predictions of our model.

KEYWORDS
cost of plasticity, environmental cues, indirect genetic effects, maternal effects, phenotypic plasticity

1 | INTRODUCTION

Different individuals within a population may experience different environmental conditions, but a single phenotypic adaptation is unlikely to be adequate for all local conditions. Many organisms exhibit phenotypic plasticity, the capacity to produce alternative phenotypes depending on the environment. Plasticity is defined as 'the environmentally sensitive production of alternative phenotypes

by given genotypes' (DeWitt & Scheiner, 2004, p. 2). Environmental cues may occur during critical periods of development and be correlated with or predictive of present or future local conditions that affect fitness. In such cases, phenotypic plasticity may permit an adaptive response to present or future environmental variation by developing a phenotype that enhances local fitness (Auge, Leverett, Edwards, & Donohue, 2017; Scheiner, 1993; Via et al., 1995). There are many examples of alternative phenotypes found in nature. These include induced defences in the presence of predators (e.g. DeWitt, 1998; Harvell, 1998; Hazel, Smock, Lively, & Moore, 2004; Lively, 1986; Walsh et al., 2016), alternative male mating strategies (e.g. Aubin-Horth & Dodson, 2004; Cruickshank & Wade, 2012; Moczek, 1998; Shuster & Wade, 1991; Stern & Emlen, 1999), winged and wingless morphs (e.g. Abouheif & Wray, 2002; Roff & Gélinas, 2003; Ueno, de Jong, & Brakefield, 2004), cannibalism morphs in amphibians (Collins, 1979; Collins & Cheek, 1983; Maret & Collins, 1997), facultative paedomorphosis (Whiteman, 1994; Wilbur & Collins, 1973), alternative patterns of cryptic coloration (Hazel, Ante, & Stringfellow, 1998; Hazel, Smock, & Johnson, 1990) and gender differences in plants (Delph & Wolf, 2005; McCauley & Taylor, 1997) and reptiles (Crews, 2003; Freedberg & Wade, 2004). In some cases, like the defensive morphs induced by the presence of predators or the dispersal morphs (winged/wingless) induced by environmental deterioration or stress, alternative plastic phenotypes have been shown to be adaptive, that is to enhance fitness, although the relationship to fitness has not yet been definitively established in other cases but is often presumed to be fitness enhancing. The cost and benefits of plasticity are themselves often quite difficult to measure (Auld, Agrawal, & Relyea, 2010).

Individuals can be plastic (intragenerational or direct plasticity), wherein members of the same genotype produce different phenotypes in response to cues from the environments they experience. This is often the case for predator-induced defences (Agrawal, Laforsch, & Tollrian, 1999). Alternatively, an individual's phenotype may be under the control of its parents, usually the mother (transgenerational or indirect plasticity), so that mother's genotype determines the phenotype produced by a given genotype of her offspring. This is often the case with alternative male mating strategies (e.g. Hunt & Simmons, 2000; Kotiaho, Simmons, Hunt, & Tomkins, 2003) or brood sex ratios (e.g. Cruickshank & Wade, 2012). Under what conditions does plasticity evolve to have intragenerational as opposed to transgenerational genetic control? To address this question, we present a population genetic model for the evolution of transgenerational and intragenerational plasticity in order to evaluate both types of plasticity within the same evolutionary context.

In constructing our genotype-based model, we use principles previously identified as potentially important to the evolution of adaptive plasticity. For example, Auge et al. (2017) identified the relative predictability or accuracy of reading environmental cues as a key determinant in the evolution of intragenerational relative to transgenerational control of plasticity. Clearly, development in many organisms is affected both by the environment they experience directly and by the environment experienced by their parents (e.g. Cavieres, Alruiz, Medina, Bogdanovich, & Bozinovic, 2019). The central theoretical issue is whether and to what degree these different environmental experiences are harnessed during development to enhance offspring fitness, that is, to confer adaptive plasticity.

It is reasonable to assume that cues experienced directly by progeny are better predictors of future selective environments than cues experienced by parents, because the time between cue perception and onset of the selective environment is shorter in the former. Ezard, Prizak, and Hoyle (2014) formalized aspects of this relationship in an additive quantitative genetic model and showed quantitatively how the pattern of environmental variation and the magnitude of the developmental lag between cue perception and onset of the selective environment together affect the evolution of plasticity. Their model results were consistent with this assumption in that slowly changing environments were relatively more favourable for the evolution of transgenerational plasticity, although their model did not incorporate a fitness cost to plasticity. When Auge et al. (2017) reviewed plasticity in plants to test assumption that the shorter relationship between cue perception and selection onset favours direct intragenerational plasticity, they found little support for it, although few studies provide accurate estimates of the variability of the selective environment or the cost of plasticity. Furthermore, there is empirical evidence that the developmental integration of parental and offspring environmental experiences can be complex. In their studies of predator-resistant phenotypes in *Daphnia ambigua*, Walsh, Cooley, Biles, and Munch (2015) found a trade-off in the developmental effects of parent and offspring environmental experience: offspring phenotypic responses to predator cues within and across generations differed in sign. Our model provides a common theoretical framework in which to compare differences in predictability of future environments from present environmental experience as well as differences in the costs and benefits of plasticity in the evolution of direct within-generation control and indirect across-generation control of adaptive plasticity. In our model, we also distinguish the fitness cost of the capacity for plasticity from the error rate of the developmental response to plasticity. For example, in their studies of environmental determination of sex in the turtle *Graptemys ouachitensis*, Freedberg, Ewert, and Nelson (2001) found that, despite a strong environmental cue, some male turtles developed at female-inducing temperatures, and some females developed at male-inducing temperatures. Here, although the environmental cue was strong and reliable, the developmental response was not all or nothing.

2 | THE MODEL

We consider a simplified world composed of two spatial environments, E_1 and E_2, that occur at relative frequencies e_1 and e_2, respectively, such that $e_1 + e_2 = 1$. We assume that any given individual experiences only one of the two environments and that individuals randomly experience one of the two environments, with probability equal to the relative frequency of each environment. Individuals are

sedentary after initial dispersal from their mothers. Furthermore, the phenotype with the highest fitness in E_1 is different from the phenotype with the highest fitness in E_2. As a result, an individual with a 'plastic phenotype' can achieve high fitness by irreversibly developing the adaptive phenotype in each environment (i.e. correctly develop into the phenotype adapted for E_1 in environment E_1, or into the phenotype adapted for E_2 in environment E_2). We hypothesize that there is a fitness cost to this developmental capacity for phenotypic plasticity, which, in some circumstances, may exceed its fitness advantage. We further hypothesize that an individual might err in its reading of environmental cues and, although plastic during their development, might nevertheless produce a suboptimal or environmentally mismatched phenotype in response to these cues—for example individuals could incorrectly develop into the phenotype adapted for E_1 in environment E_2, or into the phenotype adapted for E_2 in environment E_1. In short, we model how errors in the perception of the environment during development and the cost of the capacity for plasticity itself set limits on the evolution of adaptive plasticity in the face of environmental variation.

We postulate an additively acting, diploid, autosomal, 'plasticity' locus, with alternative alleles, B_1 and B_0, in frequency q_{direct} and p_{direct}, respectively. The subscripts 'direct' indicate that individuals with these alleles manifest plasticity that affects their fitness in the face of environmental variation; that is, gene expression is intragenerational. We contrast the evolution of this intragenerational plasticity locus (B_1 at frequency q_{direct}) with the evolution of a similar locus where offspring plasticity is controlled by alternative alleles acting in (i.e. expressed by) the maternal genome. That is, we also investigate the evolution of a 'transgenerational plasticity' locus, wherein an additively acting, diploid and autosomal locus has alternative alleles, A_1 and A_0, in frequencies q_{trans} and p_{trans}, with a maternal effect on offspring developmental plasticity, whereby mothers are fully responsible for the portion of offspring phenotype that is plastic. With transgenerational plasticity, the individual that bears the fitness costs, usually the mother, can be different from the individual that enjoys the fitness advantage, her offspring. Following the classifications of DeWitt, Sih, and Wilson (1998) and Auld et al. (2010), mothers bear the genetic costs of plasticity loci, as well as the maintenance and production costs of alternate phenotypes. In addition, adult perception of environmental conditions can be cheaper (i.e. reduced information-acquisition costs) and in many cases more accurate than embryonic perceptions, so that the error rate may be reduced with transgenerational plasticity.

Individuals bearing B_1 and offspring of mothers bearing A_1 can irreversibly develop into alternative phenotypes. This plasticity comes with an inherent cost c to fitness that affects the bearers of B_1 and A_1 by amounts c_{direct} and c_{trans}, respectively. Plasticity also confers fitness benefits that may offset these costs. When a phenotype matches the environment, organisms receive an incremental fitness advantage a; specifically, they receive a_1 for matching E_1 and a_2 for matching E_2. There is another potential fitness cost beyond the inherent cost of plasticity, that is the cost when an environmental cue is misread and rather than matching its environment, when an organism then produces a phenotype that does not match its environment, it incurs a fitness penalty d. Like the fitness increments for environmental matching, we impose the fitness decrements, d_1 for mismatching E_1 and d_2 for mismatching E_2.

How often does an individual produce a phenotype that matches its environment as opposed to mismatching its environment? This depends both on e_1 and e_2, the frequencies of both environments, and on the error rate in reading and interpreting environmental cues which is r. Note $1-r$ is the probability of a plastic individual correctly reading and interpreting environmental cues and producing the adaptive phenotype. We call the error rate of A_1-bearing mothers r_{trans} and the error rate of B_1-bearing individuals r_{direct}. Both r_{trans} and r_{direct} lie within the bounds $0 \le r \le 0.5$. When $r = 0$, plastic individuals do not make mistakes reading and interpreting cues; they always produce the adaptive phenotype. When $r = 0.5$ the plastic individual is arbitrarily plastic; there is no correspondence between the cues and phenotype produced, and the odds of having a matching phenotype are equal to flipping a coin.

The fitness advantage of matching is thus $(1-r)(a_1e_1 + a_2e_2)$, which equals the product of correctly 'predicting' the environment $(1-r)$ and the advantages weighted by the frequency of each environment $(a_1e_1 + a_2e_2)$. We will call this latter term $\bar{a}$ the mean advantage. Similarly, the fitness disadvantage from mistakenly interpreting the cues is $-r(d_1e_1 + d_2e_2)$, which is the product of the frequency of mistakes r and the environmentally weighted mismatch penalties $(d_1e_1 + d_2e_2)$. We call this term $\bar{d}$ the average mismatch disadvantage. In the terms $\bar{a}$ and $\bar{d}$, environmental frequencies (e_1 and e_2), either the advantage of matching (a_1 and a_2) or disadvantage of mismatching (d_1 and d_2), are taken into account and multiplied by the error rate of plasticity (r) or its inverse, which explicitly links environment frequency and the evolutionary pressure to evolve a better adapted phenotype (Snell-Rood, 2013; Snell-Rood, Van Dyken, Cruickshank, Wade, & Moczek, 2010). The plastic individuals B_0B_1, heterozygotes bearing the new plasticity allele B_1, would have an average fitness of $1 + (1-r)(a_1e_1 + a_2e_2) - r(d_2e_2 + d_1e_1) - c$. Having a_1, a_2, d_1 and d_2 as separate terms allows us to vary fitness advantages and disadvantages to plasticity both independently and in an environment-specific way. When a_1 and a_2 are both greater than 0, plasticity means that it is possible to produce a better more specialized phenotype for each environment, E_1 and E_2, relative to the nonplastic phenotype. Nonplastic individuals with genotype B_0B_0 and offspring of mothers with genotype A_0A_0 all have a fixed phenotype, with a baseline fitness of 1.

This parameterization gives us the flexibility to model a variety of different starting points for the evolution of plasticity. For example, consider a case where, initially, all genotypes are adapted to E_1 and the evolution of plasticity amounts to developing a phenotype better able to exploit (i.e. having a higher fitness in) a novel environment, E_2. If we set a_1 equal to 0 but $a_2 > 0$, this would mean the advantage of plasticity comes from producing a phenotype to match E_2. And, if we set $d_2 = 0$ and $d_1 < 0$, this means that the potential disadvantage of plasticity is producing a poorer phenotype in environment E_1. The error rate determines whether the

TABLE 1 Combinations of maternal and paternal genotypes, their respective frequency, mean fitness, offspring genotypes and family contribution to q'_{trans}

Parental genotypes ♀ × ♂	Family frequency	Family mean fitness	Offspring proportion for each genotype			Contribution to q'_{trans} $\left[\text{frequency} \times \text{fitness} \times \left(\frac{1}{2} A_0A_1\text{proportion} + A_1A_1\text{proportion}\right)\right]$
			A_0A_0	A_0A_1	A_1A_1	
$A_0A_0 \times A_0A_0$	$P \times P$	1	1	–	–	0
$A_0A_0 \times A_0A_1$	$P \times H$	1	1/2	1/2	–	$\frac{1}{4}PH$
$A_0A_0 \times A_1A_1$	$P \times Q$	1	–	1	–	$\frac{1}{2}PQ$
$A_0A_1 \times A_0A_0$	$H \times P$	$1+(1-r)\bar{a}-r\bar{d}-c$	1/2	1/2	–	$\frac{1}{4}HP \times \left[1+(1-r)\bar{a}-r\bar{d}-c\right]$
$A_0A_1 \times A_0A_1$	$H \times H$	$1+(1-r)\bar{a}-r\bar{d}-c$	1/4	1/2	1/4	$\frac{1}{2}HH \times \left[1+(1-r)\bar{a}-r\bar{d}-c\right]$
$A_0A_1 \times A_1A_1$	$H \times Q$	$1+(1-r)\bar{a}-r\bar{d}-c$	–	1/2	1/2	$\frac{3}{4}HQ \times \left[1+(1-r)\bar{a}-r\bar{d}-c\right]$
$A_1A_1 \times A_0A_0$	$Q \times P$	$1+2\left[(1-r)\bar{a}-r\bar{d}-c\right]$	–	1	–	$\frac{1}{2}QP \times \left\{1+2\left[(1-r)\bar{a}+r\bar{d}-c\right]\right\}$
$A_1A_1 \times A_0A_1$	$Q \times H$	$1+2\left[(1-r)\bar{a}-r\bar{d}-c\right]$	–	1/2	1/2	$\frac{3}{4}QH \times \left\{1+2\left[(1-r)\bar{a}+r\bar{d}-c\right]\right\}$
$A_1A_1 \times A_1A_1$	$Q \times Q$	$1+2\left[(1-r)\bar{a}-r\bar{d}-c\right]$	–	–	1	$QQ \times \left\{1+2\left[(1-r)\bar{a}+r\bar{d}-c\right]\right\}$

advantage of matching offsets the disadvantage of mismatching and the inherent cost of plasticity. Plasticity is generally thought to require a genotype-by-environment interaction (G × E) to evolve (e.g. Scheiner, 1993). It is mathematically possible—although unlikely in practice—for plasticity to evolve without G × E *for fitness*. In our model, plasticity evolves without G × E for fitness only when $a_1 = a_2$ and $d_1 = d_2$. In most models, either a_1 or a_2 is 0, because there is only one environment in which a plastic individual can produce a better phenotype. When this is the case in our model, we have G × E for fitness. In the special case when $a_1 = a_2$ and $d_1 = d_2$, our 'plastic' phenotype enjoys equivalent fitness increments (or decrements) in both environments; that is, it is simply a better phenotype everywhere.

We now consider whether or not a gene (maternal or direct) for plasticity invades the nonplastic population. Using the above, we can calculate the mean fitness W of the genotypes with intragenerational plasticity and no plasticity:

$$W_{B_1B_1} = 1+2\left[(1-r)\,\bar{a}_{\text{direct}} - r\bar{d}_{\text{direct}} - c_{\text{direct}}\right] \tag{1a}$$

$$W_{B_0B_1} = 1+(1-r)\,\bar{a}_{\text{direct}} - r\bar{d}_{\text{direct}} - c_{\text{direct}} \tag{1b}$$

$$W_{B_0B_0} = 1+(e_1+e_2) = 1 \tag{1c}$$

By multiplying the fitness of each genotype by its frequency, and simplifying, we obtain the mean fitness of the population:

$$\overline{W} = 1+2q_{\text{d}}\left[(1-r)\,\bar{a}_{\text{direct}} - r\bar{d}_{\text{direct}} - c_{\text{direct}}\right] \tag{2}$$

After selection, q'_{direct}, the new value of frequency of B_1, is the sum of plastic homozygote mean fitness and half of heterozygote mean fitness, divided by mean population fitness. In turn, the change of allele frequency Δq_{direct} can be calculated by subtracting the original frequency of q_{direct} from its new frequency q'_{direct}:

$$\Delta q_{\text{direct}} = \frac{Wq_{\text{d}}q_{\text{d}} + \frac{1}{2}Wq_{\text{d}}p_{\text{d}}}{\overline{W}} - q_{\text{d}} = \frac{(p_{\text{d}}q_{\text{d}})\left[(1-r_{\text{direct}})\,\bar{a}_{\text{direct}} - r\bar{d}_{\text{direct}} - c_{\text{direct}}\right]}{\overline{W}} \tag{3}$$

In the case of transgenerational plasticity, the phenotype—and crucially, the fitness—of an individual depends not on its own genotype but on its mother's genotype. For example, all offspring born to mothers with genotype A_0A_0 will have the same phenotype and fitness of 1, regardless of their own genotype or paternal genotype, whereas all offspring (again regardless of genotype) born to mothers with genotype A_1A_1 either receive a fitness increment, $2a$, for matching or incur a fitness decrement, $2d$, for mismatching.

When calculating the transgenerational plasticity allele's change of allele frequency (Δq_{trans}), the dependence of fitness on maternal genotype makes it necessary to keep track of both parents' genotypes. Paternal genotype, despite having no effect on fitness of individual offspring, provides half of each offspring's genotype. To keep track of genotype frequencies, we use P to

denote the frequency of nonplastic homozygote individuals (with genotype A_0A_0), Q to denote the frequency of plastic homozygote individuals (with genotype A_1A_1) and H to denote the frequency of plastic heterozygote individuals (with genotype A_0A_1). Assuming equal sex ratio and regular Mendelian segregation of alleles, we calculated the contributions to q'_{trans} made by each possible pairing of maternal and paternal genotype (Table 1). For example, families with A_1A_1 mothers and A_0A_0 fathers, occurring at a frequency of $Q \times P$, incur a cost of plasticity $2c$ and produce exclusively heterozygous offspring, each either receiving $2a$ for matching or incurring $2d$ for mismatching. Meanwhile, families with A_0A_0 mothers and A_1A_1 fathers, which occur at the same frequency $P \times Q$, also produce exclusively heterozygous offspring, but these all have a fitness of 1.

The change in frequency of the transgenerational plasticity allele A_1 after one generation (Δq_{trans}) is calculated by summing the contributions to q'_{trans} (listed in Table 1)—which are calculated taking family of origin into account—then dividing q'_{trans} by the mean fitness of the population ($\overline{W}$) and subtracting the original allele frequency (q_{trans}). Mean fitness equals the sum of the products of family frequency and family mean fitness (columns 2 and 3 in Table 1), which is $\left\{1+2q\left[(1-r)\,\bar{a}+r\bar{d}-c\right]\right\}$, the same quantity as mean fitness for the direct-effect model (see Equation (2) above).

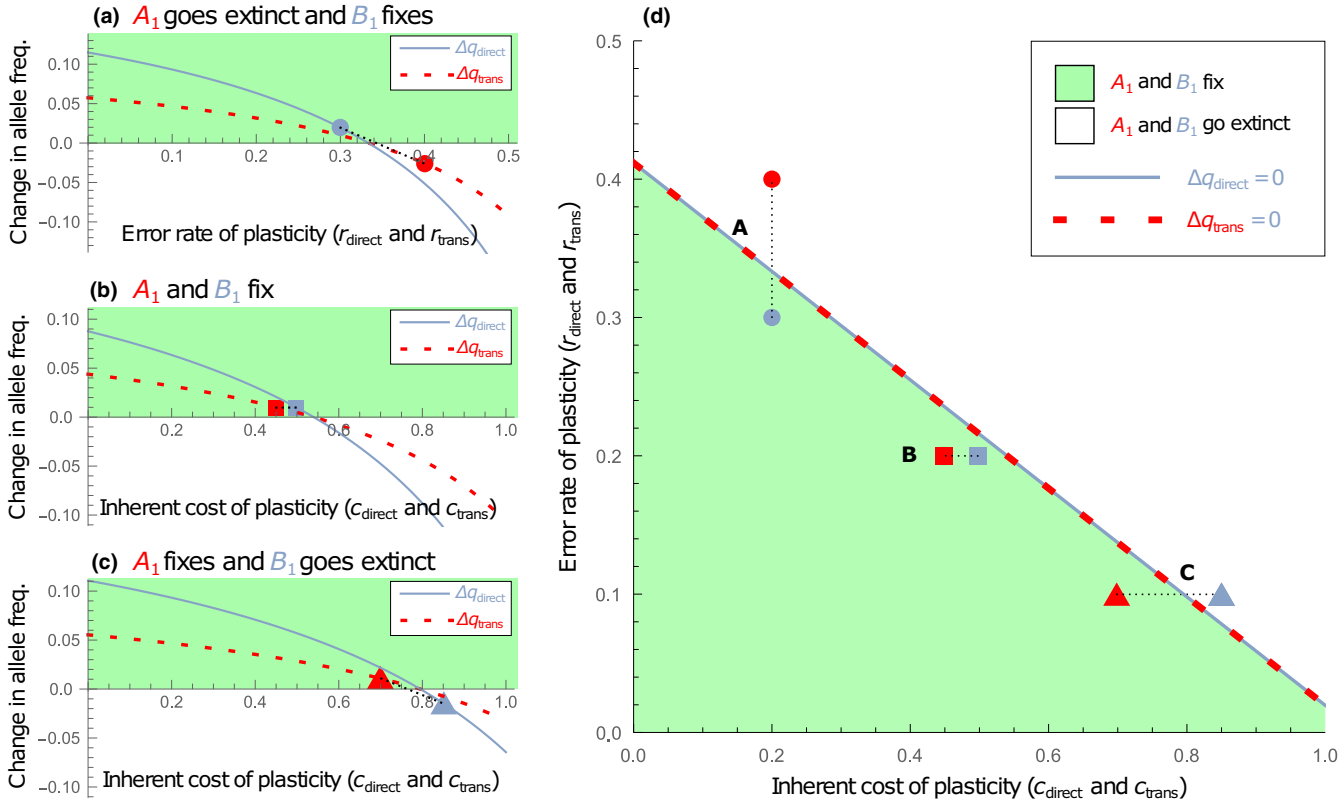

FIGURE 1 Persistence or extinction of plasticity depends on differences in error rate (r) and inherent cost (c). Panel a depicts a scenario where the accuracy of correctly predicting the future environment is lower for transgenerational plasticity than it is for intragenerational plasticity ($r_{direct} < r_{trans}$), and the inherent cost of plasticity is the same for both alleles ($c_{direct} = c_{trans}$). Circles represent the values of Δq resulting from the following parameter values: $r_{direct} = 0.3$, $c_{direct} = 0.2$, $r_{trans} = 0.4$, $c_{trans} = 0.2$; the difference in error rate of correctly predicting the future environment is enough alone to result in a positive value of Δq_{direct} (blue circle) and to make intragenerational plasticity (allele B_1) advantageous, and to result in a negative value of Δq_{trans} (red circle) and make transgenerational plasticity (allele A_1) disadvantageous. Panel b depicts a scenario where both plasticity alleles (B_1 and A_1) evolve at essentially the same rate because $2\left\{(1-r)\,\bar{a}-r\bar{d}-c\right\}_{trans}$ equals $\left\{(1-r)\bar{a}-r\bar{d}-c\right\}_{direct}$, despite differing error rates, benefits and costs of plasticity. Squares represent the values of Δq resulting from the following parameter values: $r_{direct} = 0.2$, $c_{direct} \approx 0.50$, $r_{trans} = 0.2$, $c_{trans} = 0.45$; these result in positive values of both Δq_{direct} (blue square) and Δq_{trans} (red square), with both intra- and transgenerational plasticity going to fixation. Panel c depicts a scenario where the cost of plasticity is lower for A_1 than for B_1 ($c_{trans} < c_{direct}$), but the error rate of correctly predicting the future environment is the same for both ($r_{direct} = r_{trans}$). Triangles represent values of Δq resulting from the following parameter values: $r_{direct} = 0.1$, $c_{direct} = 0.85$, $r_{trans} = 0.1$, $c_{trans} = 0.7$; the sole difference in inherent cost of plasticity is enough to make intragenerational plasticity (allele B_1) advantageous, and to result in a positive value of Δq_{trans} (red triangle), while making transgenerational plasticity (allele A_1) disadvantageous, and give a negative value of Δq_{direct} (blue triangle). Panel d, two-dimensional bifurcation plot for outcomes of the introduction of alleles allowing for either intragenerational plasticity (B_1 at frequency q_{direct}) or transgenerational plasticity (A_1 at frequency q_{trans}), depending on the error rate of plasticity (r)—rate of generating a mismatched phenotype and receiving a fitness penalty rather than an advantage—and inherent cost of plasticity (c), with all other parameters being equal ($e_1 = 0.5$; $e_2 = 0.5$; $a_1 = 1.05$; $a_2 = 1.05$; $d_1 = 1.5$; and $d_2 = 1.5$). Scenarios A, B and C, which represent different outcomes with given values of r_{direct} & r_{trans} and c_{direct} & c_{trans}, are also depicted in panels a, b and c and Figure 2

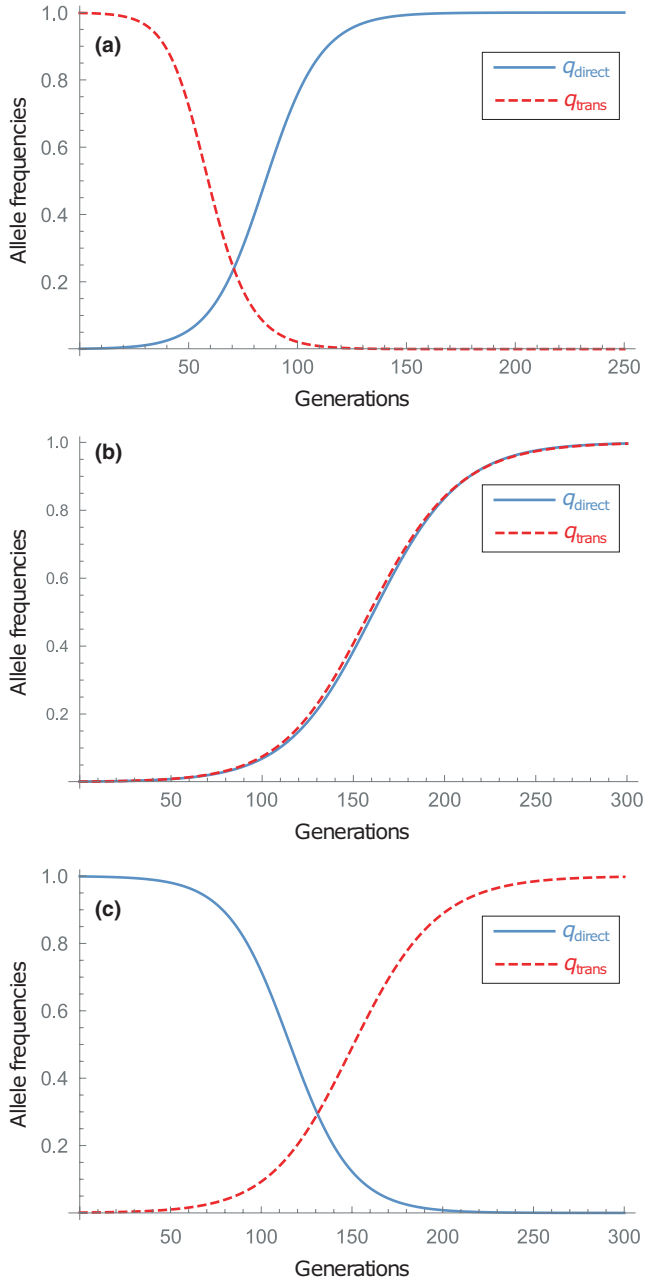

FIGURE 2 Evolution of intragenerational plasticity and transgenerational plasticity. Temporal changes in frequencies of alleles for intragenerational plasticity (B_1 at frequency q_{direct}, solid blue line) and of alleles for transgenerational plasticity (A_1 at frequency q_{trans}, dashed red line) with given error rates of plasticity (r)—rate of generating a mismatched phenotype—and inherent cost of plasticity (c), with all other parameters equal ($e_1 = 0.5$; $e_2 = 0.5$; $a_1 = 1.05$; $a_2 = 1.05$; $d_1 = 1.5$; and $d_2 = 1.5$). Starting frequencies are 0.999 if Δq is negative and 0.001 if Δq is positive. Scenarios A, B and C represent different outcomes with given values. Scenario A: $r_{direct} = 0.3$, $c_{direct} = 0.2$, $r_{trans} = 0.4$, $c_{trans} = 0.2$, intragenerational plasticity goes to fixation, whereas transgenerational plasticity goes extinct; Scenario B: $r_{direct} = 0.2$, $c_{direct} \approx 0.50$, $r_{trans} = 0.2$, $c_{trans} = 0.45$, both intra- and transgenerational plasticity go to fixation; Scenario C: $r_{direct} = 0.1$, $c_{direct} = 0.85$, $r_{trans} = 0.1$, $c_{trans} = 0.7$, intragenerational plasticity goes extinct, whereas transgenerational plasticity goes to fixation. The same three scenarios are depicted in Figure 1

$$\Delta q_{trans} = \frac{\left(\frac{1}{4}HP + \frac{1}{4}HQ + PQ\right)\left[(1-r)\,\bar{a}_{trans} - r\bar{d}_{trans}c_{trans}\right]}{\overline{W}} \quad (4)$$

This Δq_{trans} is expressed in terms of genotype frequencies rather than in terms of allele frequencies like Δq_{direct}, which hinders direct comparison. We can reduce the first term in the numerator to $(1/2)(Pq_t + Qp_t)$. Then, we assume that, with weak selection, $P \sim p_t^2$ and $Q \sim q_t^2$ so that we can reduce further and find that $(1/2)(p_t q_t)(p_t + q_t) = (p_t q_t/2)$. Now Equation (4) equals (1/2) times Equation (3): $\Delta q_{trans} = {}^{1}\!/_{2}\,\Delta q_{direct}$. The rate of evolution of transgenerational plasticity is therefore half the rate of evolution of intragenerational plasticity. In the theory of indirect genetic effects, all else being equal (i.e. gene frequencies, selection coefficients, mean fitness and random mating), a maternal-effect gene evolves at half the rate of a direct-effect gene (Barker, Demuth, & Wade, 2005; Cruickshank & Wade, 2008; Demuth & Wade, 2007; Wade, 1998; Wolf & Wade, 2009). When an individual dies because of its own gene, that death removes a copy of the gene from the population. However, when an individual dies because of a gene in its mother's genome, that death removes, on average, half of one copy of the gene because the regression of offspring genotype on mother's genotype is 1/2. In our model, with transgenerational plasticity, mother's genotype determines the plastic phenotype of an offspring, whereas, with intragenerational plasticity, the offspring genotype determines its plastic phenotype. Thus, the slower rate of transgenerational plasticity is consistent with the expectations of the theory of indirect genetic effects.

Because the rate of evolution of intragenerational plasticity is twice the rate of evolution of transgenerational plasticity, in situations where all other parameters are equal (i.e. when $\bar{a}_{direct} = \bar{a}_{trans}$; $c_{direct} = c_{trans}$; and $\bar{d}_{direct} = \bar{d}_{trans}$), intragenerational and transgenerational plasticity alleles will either be both advantageous or be both disadvantageous (note, in Figure 1, the completely overlapping regions in which intragenerational and transgenerational plasticity alleles become either fixed or lost). However, when there are differences in the inherent costs of plasticity or in the developmental error rates, transgenerational plasticity may be favoured over intragenerational plasticity (e.g. scenarios A, B and C, with parameter values chosen to illustrate different outcomes in Figures 1 and 2).

In scenario A (Figures 1 and 2a), we model the case wherein the accuracy of correctly predicting the future environment is lower for transgenerational plasticity than it is for intragenerational plasticity ($r_{direct} < r_{trans}$), and the inherent cost of plasticity is the same for both alleles ($c_{direct} = c_{trans}$). With the specific parameter values modelled, the difference in error rate of correctly predicting the future environment is enough alone to make intragenerational plasticity (allele B_1) advantageous, while making transgenerational plasticity (allele A_1) disadvantageous.

Any combination of differing error rates, benefits and costs of plasticity, such that $2\left\{(1-r)\,\bar{a} - r\bar{d} - c\right\}_{trans}$ equals $\left\{(1-r)\,\bar{a} - r\bar{d} - c\right\}_{direct}$,

results in both plasticity alleles (B_1 and A_1) evolving at essentially the same rate. For example, in scenario B (Figures 1 and 2b), r_{direct} is larger than r_{trans} but c_{direct} is smaller than c_{trans}, but the reverse could also lead to the same changes in allele frequency. In scenario C (Figures 1 and 2c), the cost of plasticity is lower for A_1 than for B_1 ($c_{trans} < c_{direct}$), but the error rate of correctly predicting the future environment is the same for both ($r_{direct} = r_{trans}$). With the specific parameter values modelled, the sole difference in inherent cost of plasticity is enough to make intragenerational plasticity (allele B_1) advantageous, while making transgenerational plasticity (allele A_1) disadvantageous. These three scenarios are by no means an exhaustive list of parameter combinations; they represent parameter values chosen to depict three of four possible outcomes. The fourth possible outcome is not depicted and would be a case where both types of plasticity go extinct. Thus, our scenarios illustrate that it is the differences in either the fitness cost of the capability of plasticity, the error rate of correctly predicting the future environment, or the fitness benefits and costs of matching or mismatching that lead to different outcomes.

3 | DISCUSSION

We find that both types of plasticity are favoured in the same parameter spaces, because the change of allele frequency for transgenerational plasticity is half that for intragenerational plasticity ($\Delta q_{trans} = {}^1\!/_2\, \Delta q_{direct}$), that is, both go to fixation or extinction in the same regions of parameter space (Figure 1). As far as we are aware, this difference in the evolution of intra- and transgenerational plasticity has not been considered in prior models. These results support a verbal model by Marshall and Uller (2007), where they proposed that the evolution of adaptive transgenerational phenotypic plasticity, where parents determine the phenotype of their offspring, should be broadly similar to the evolution of intragenerational phenotypic plasticity. Plasticity of either type is favoured when (1) the environment is heterogeneous; (2) the environment provides clues to reliably predict future conditions (i.e. when the error rate of plasticity and the fitness consequence of mismatching are low); and (3) the inherent cost of the capability of plasticity is low. Moreover, one type of plasticity may be favoured over the other when there are different costs and error rates for transgenerational compared to intragenerational plasticity (e.g. scenarios A, B and C in Figures 1 and 2). The results of our model are complementary to those of Kuijper and Hoyle (2015), they also ask when and whether intra- or transgenerational plasticity should be favoured, but contrary to our model, they consider transgenerational plasticity (their maternal-effect coefficient m_t) is controlled by the offspring, as in cases when offspring sensitivity evolves in response to signals in the parental phenotype.

Nature provides numerous examples of both transgenerational and intragenerational plasticity. The easiest examples to categorize are those where the control of plasticity can only be maternal and transgenerational, and those where the control of plasticity can only be intragenerational. In Figure 1, these types of plasticity straddle the diagonal line indicating parameter combinations where $\Delta q = 0$; one type of plasticity goes to fixation, whereas the other type is lost. We begin our discussion with examples of intragenerational plasticity.

3.1 | Plasticity where individuals themselves can best influence their own phenotype

Environmental cues useful to determining which phenotype is advantageous may not be available to parents if they are highly dependent on the individual's local context or condition. This is depicted by scenario A in Figure 1: the error rate of intragenerational plasticity is low enough for intragenerational plasticity to evolve, whereas the error rate of transgenerational plasticity is too high for transgenerational plasticity to evolve or be maintained. This is the case for intragenerational sex plasticity, which is characteristic of some fishes when local conditions *after maturation* determine whether it might be advantageous to be male or female. They may go from female to male (these are protogynous hermaphrodites) or from male to female (these are protandrous hermaphrodites). Determining when it is most advantageous to change sex is usually best done using cues available to individuals themselves rather than cues available to their parents. For example, orange clownfish (*Amphiprion percula* (Lacepède, 1802)) live in groups within the protective tentacles of sea anemones. In each group, the largest individual is female, the second-largest is male, and the following individuals (up to four) are nonbreeders. If the female dies, the male, now the largest individual, changes sex and becomes female, and the largest nonbreeder, now second-largest individual, becomes male (Buston, 2004). This type of extremely local environmental cue, occurring during adult life, permits intragenerational sex plasticity and probably prohibits transgenerational plasticity.

Plants are plastic in many ways that are best controlled through intragenerational plasticity rather than through transgenerational plasticity. Heterophylly is a striking example of plasticity where plants can produce different types of leaves below and above water; their aerial leaves are thicker, cutinized and have stomata, whereas their aquatic leaves are thinner and lack both cuticle and stomata (Wells & Pigliucci, 2000). Intragenerational plasticity related to seedling growth and resource acquisition permits reproductive success of individual plants across diverse microenvironments, including those created by intraspecific and interspecific crowding (Sultan, 2000). Inducible defences are another example of plasticity, in the case of invertebrates and vertebrates, these are defensive shifts in behaviour, morphology or life history induced by predators, pathogens or parasites. In the case of plants, they are defensive shifts in chemistry or morphology, induced by herbivores, pathogens or parasites (Harvell & Tollrian, 1999). The induction of these defences is often better done by individuals themselves rather than their parents, but in some cases, parents do indeed participate in defence induction (Agrawal et al., 1999).

3.2 | Plasticity influenced by both parents and individuals themselves

Plasticity can be under both intra- and transgenerational control. In Figure 1, these cases are akin to scenario B, where alleles for both types of plasticity are favoured. The dual control of plasticity can allow for a stronger adaptive response (e.g. in a model; Ezard et al., 2014). Hormone deposition in avian eggs can have lasting effects on offspring phenotype, effects that can enhance existing intragenerational plasticity (Groothuis, Müller, von Engelhardt, Carere, & Eising, 2005). In plants, the combination of intra- and transgenerational plasticity in the adaptive response of redshank (*Persicaria maculosa* Gray) seedlings to drought was earlier and more pronounced than from intragenerational plasticity alone (Herman & Sultan, 2011).

3.3 | Plasticity where only mothers can influence offspring phenotype

In some cases, only mothers can influence plasticity. In Figure 1, these cases are akin to scenario C in that transgenerational plasticity is advantageous but differ in that intragenerational plasticity would simply be impossible. For example, when female seed beetles (*Stator limbatus* (Horn, 1873)) discover the large and thick-coated seeds of the blue palo verde (*Parkinsonia florida* (Benth. ex A. Gray) S. Watson), the beetles lay fewer, larger eggs, so that hatching larvae are sufficiently large to penetrate the seed coat and begin feeding. However, when females find the small thin-coated seeds of the catclaw acacia (*Senegalia greggii* (A. Gray) Britton & Rose), they lay more and smaller eggs, because even small larvae can penetrate the seed coat and begin feeding (Fox, Thakar, & Mousseau, 1997). Another example is found in the predatory stink bug *Podisus maculiventris* (Say, 1832), where females lay dark eggs on the upper surface of leaves and lighter eggs on the undersides of leaves (Abram et al., 2015). These are cases where the environmental cues associated with laying site choice are available to the mother but not to the egg. That is, this adaptive type of transgenerational plasticity of eggs is likely the only possible route to plasticity.

It can be advantageous for mothers to manipulate the sex ratio of offspring for a variety of reasons including local mate competition, local resource competition and local resource enhancement (West, Shuker, Sheldon, & Mueller, 2005). In species with chromosomal or genotypic sex determination (e.g. the broad-horned flour beetle, *Gnathocerus cornutus* (Fabricius, 1798); Cruickshank & Wade, 2012), sex ratio is exclusively manipulated by parents, generally mothers. Cruickshank and Wade (2012) showed, for example, that nutritionally stressed females produced more offspring and more female-biased offspring than females raised in nutritionally good environments, but observed no effects on either family size or brood sex ratio as a result of male nutritional stress. Mothers in haplodiploid fig wasps adjust the sex of their offspring; early on, they lay only male eggs, which receive a competitive advantage from being first to mature, and later, mothers lay mostly female eggs (Raja, Suleman, Compton, & Moore, 2008). Species with temperature-dependent sex determination (e.g. many crocodilians, Lang & Andrews, 1994) are cases of transgenerational plasticity because mothers control the sex of their offspring by choosing where to lay their brood.

3.4 | Plasticity where mothers can best influence offspring phenotype

When transgenerational plasticity is favoured over intragenerational plasticity, either the error rate in plasticity is smaller for parents, the cost is lower for parents, or both, making transgenerational plasticity advantageous, but intragenerational plasticity too costly to evolve or maintain; this is scenario C in Figure 1. Transgenerational plasticity in seed coat traits, which can affect vulnerability to predation, dispersal and germination time, is almost always less costly to the maternal plant (Roach & Wulff, 1987). In many insects, transgenerational plasticity is common when a female's experience of photoperiod or temperature affects the likelihood of diapause in her offspring (Mousseau & Fox, 1998). Mothers can also use biotic cues. For example, female aphids are more likely to produce winged offspring when conditions are crowded and when predators are present (Mehrparvar, Zytynska, & Weisser, 2013).

Transgenerational plasticity may have an additional advantage over intragenerational plasticity that we have not explored here. 'Inbreeding makes maternal genetic effects more available for an evolutionary response to selection, with the potential to double their contribution to adaptation when a population is fully inbred' (Wolf & Wade, 2016, p. 837). Additionally, when confronted with novel environments, parents can increase their offspring's genetic variation for survival (Chirgwin, Marshall, Sgrò, & Monro, 2018). Moreover, increased heritable variation is maintained at mutation–selection equilibrium for an indirect maternal genetic trait compared to a direct individual trait with the same fitness costs and benefits (Barker et al., 2005; Cruickshank & Wade, 2008; Demuth & Wade, 2007; Wade, 1998). The greater standing genetic variation, all else being equal, may permit a greater response to selection in the face of increased environmental variation when the phenotypic plasticity is transgenerational rather than intragenerational.

3.5 | Conclusion and implications

In addition to a fitness cost to the capacity for adaptive plasticity, recent evolutionary models have highlighted the importance of the predictability of environmental cues, the accuracy of reading and interpreting those cues, as well as the time between cue perception and onset of the selective environment. Our model builds on these earlier models and, in particular, examines the cost of plasticity and the fitness cost of errors in reading or interpreting environmental cues in the same framework. In addition, our model allows us to contrast whether mothers (transgenerational plasticity) or their offspring (intragenerational plasticity) have the capacity to sense environmental cues, and bear the fitness cost of plasticity within the

same population genetic framework. Two major findings emerge: (i) transgenerational plasticity evolves when mothers are better predictors of future environments than offspring and when the fitness cost of the capacity for plasticity is more readily borne by mothers than by offspring, and (ii) the rate of evolution of transgenerational plasticity is half the rate for intragenerational plasticity, consistent with the theory of indirect genetic effects. To better understand the evolution of plasticity, it will be key to continue performing studies that include multiple generations. In those studies, we will need to better identify which individual, mother or offspring reads and interprets environmental cues and which bears the multiple costs of plasticity. Finally, theory of indirect genetic effects (Wolf & Wade, 2016) has shown that inbreeding reduces the difference in rate of evolution between direct and maternal effects because it increases the regression between offspring and mother. Although we did not directly examine inbreeding here, we predict that transgenerational plasticity should be more common in species with high levels of inbreeding than it is in species with low levels of inbreeding or random mating.

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AUTHOR CONTRIBUTIONS

GJD and MJW jointly designed the model and wrote the manuscript. GJD performed the calculations and generated the figures. MJW provided advice and guidance.

DATA AVAILABILITY STATEMENT

Data used in this population genetic model can be regenerated from the equations and parameter values provided in figure captions.

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REFERENCES

Abouheif, E., & Wray, G. A. (2002). Evolution of the gene network underlying wing polyphenism in ants. *Science*, *297*(5579), 249–252. https://doi.org/10.1126/science.1071468

Abram, P. K., Guerra-Grenier, E., Després-Einspenner, M.-L., Ito, S., Wakamatsu, K., Boivin, G., & Brodeur, J. (2015). An insect with selective control of egg coloration. *Current Biology*, *25*(15), 2007–2011. https://doi.org/10.1016/j.cub.2015.06.010

Agrawal, A. A., Laforsch, C., & Tollrian, R. (1999). Transgenerational induction of defences in animals and plants. *Nature*, *401*(6748), 60–63. https://doi.org/10.1038/43425

Aubin-Horth, N., & Dodson, J. J. (2004). Influence of individual body size and variable thresholds on the incidence of a sneaker male reproductive tactic in Atlantic salmon. *Evolution*, *58*(1), 136–144. https://doi.org/10.1111/j.0014-3820.2004.tb01580.x

Auge, G. A., Leverett, L. D., Edwards, B. R., & Donohue, K. (2017). Adjusting phenotypes via within- and across-generational plasticity. *New Phytologist*, *216*(2), 343–349, https://doi.org/10.1111/nph.14495

Auld, J. R., Agrawal, A. A., & Relyea, R. A. (2010). Re-evaluating the costs and limits of adaptive phenotypic plasticity. *Proceedings of the Royal Society of London B: Biological Sciences*, *277*(1681), 503–511. https://doi.org/10.1098/rspb.2009.1355

Barker, M. S., Demuth, J. P., & Wade, M. J. (2005). Maternal expression relaxes constraint on innovation of the anterior determinant, *bicoid*. *PLOS Genetics*, *1*(5), e57. https://doi.org/10.1371/journal.pgen.0010057

Buston, P. M. (2004). Territory inheritance in clownfish. *Proceedings of the Royal Society B: Biological Sciences*, *271*(Suppl 4), S252–S254.

Cavieres, G., Alruiz, J. M., Medina, N. R., Bogdanovich, J. M., & Bozinovic, F. (2019). Transgenerational and within-generation plasticity shape thermal performance curves. *Ecology and Evolution*, *9*(4), 2072–2082. https://doi.org/10.1002/ece3.4900

Chirgwin, E., Marshall, D. J., Sgrò, C. M., & Monro, K. (2018). How does parental environment influence the potential for adaptation to global change? *Proceedings of the Royal Society B: Biological Sciences*, *285*(1886), 20181374. https://doi.org/10.1098/rspb.2018.1374

Collins, J. P. (1979). Sexually mature larvae of the salamanders *Ambystoma rosaceum* and *A. tigrinum velasci* from Chihuahua, Mexico: Taxonomic and ecologic notes. *Journal of Herpetology*, *13*(3), 351–354. https://doi.org/10.2307/1563331

Collins, J. P., & Cheek, J. E. (1983). Effect of food and density on development of typical and cannibalistic salamander larvae in *Ambystoma tigrinum nebulosum*. *Integrative and Comparative Biology*, *23*(1), 77–84. https://doi.org/10.1093/icb/23.1.77

Crews, D. (2003). Sex determination: Where environment and genetics meet. *Evolution & Development*, *5*(1), 50–55. https://doi.org/10.1046/j.1525-142X.2003.03008.x

Cruickshank, T., & Wade, M. J. (2008). Microevolutionary support for a developmental hourglass: Gene expression patterns shape sequence variation and divergence in *Drosophila*. *Evolution & Development*, *10*(5), 583–590. https://doi.org/10.1111/j.1525-142X.2008.00273.x

Cruickshank, T., & Wade, M. J. (2012). Maternal adjustment of the sex ratio in broods of the broad-horned flour beetle, *Gnathocerus cornutus*. *Integrative and Comparative Biology*, *52*(1), 100–107. https://doi.org/10.1093/icb/ics056

Delph, L. F., & Wolf, D. E. (2005). Evolutionary consequences of gender plasticity in genetically dimorphic breeding systems. *New Phytologist*, *166*(1), 119–128. https://doi.org/10.1111/j.1469-8137.2005.01339.x

Demuth, J. P., & Wade, M. J. (2007). Maternal expression increases the rate of *bicoid* evolution by relaxing selective constraint. *Genetica*, *129*(1), 37–43. https://doi.org/10.1007/s10709-006-0031-4

DeWitt, T. J. (1998). Costs and limits of phenotypic plasticity: Tests with predator-induced morphology and life history in a freshwater snail.

Journal of Evolutionary Biology, *11*(4), 465–480. https://doi.org/10.1046/j.1420-9101.1998.11040465.x

DeWitt, T. J., & Scheiner, S. M. (2004). *Phenotypic plasticity: Functional and conceptual approaches*. Oxford; Toronto: Oxford University Press.

DeWitt, T. J., Sih, A., & Wilson, D. S. (1998). Costs and limits of phenotypic plasticity. *Trends in Ecology & Evolution*, *13*(2), 77–81. https://doi.org/10.1016/S0169-5347(97)01274-3

Ezard, T. H. G., Prizak, R., & Hoyle, R. B. (2014). The fitness costs of adaptation via phenotypic plasticity and maternal effects. *Functional Ecology*, *28*(3), 693–701. https://doi.org/10.1111/1365-2435.12207

Fox, C. W., Thakar, M. S., & Mousseau, T. A. (1997). Egg size plasticity in a seed beetle: An adaptive maternal effect. *The American Naturalist*, *149*(1), 149–163. https://doi.org/10.1086/285983

Freedberg, S., Ewert, M. A., & Nelson, C. E. (2001). Environmental effects on fitness and consequences for sex allocation in a reptile with environmental sex determination. *Evolutionary Ecology Research*, *3*(8), 953–967.

Freedberg, S., & Wade, M. (2004). Male combat favours female-biased sex ratios under environmental sex determination. *Animal Behaviour*, *67*(1), 177–181. https://doi.org/10.1016/j.anbehav.2003.01.004

Groothuis, T. G. G., Müller, W., von Engelhardt, N., Carere, C., & Eising, C. (2005). Maternal hormones as a tool to adjust offspring phenotype in avian species. *Neuroscience & Biobehavioral Reviews*, *29*(2), 329–352. https://doi.org/10.1016/j.neubiorev.2004.12.002

Harvell, C. D. (1998). Genetic variation and polymorphism in the inducible spines of a marine bryozoan. *Evolution*, *52*(1), 80–86. https://doi.org/10.1111/j.1558-5646.1998.tb05140.x

Harvell, C. D., & Tollrian, R. (1999). Why inducible defenses? In C. D. Harvell & R. Tollrian (Eds.), *The ecology and evolution of inducible defenses* (pp. 3–9). Princeton, NJ, USA: Princeton University Press.

Hazel, W. N., Ante, S., & Stringfellow, B. (1998). The evolution of environmentally-cued pupal colour in swallowtail butterflies: Natural selection for pupation site and pupal colour. *Ecological Entomology*, *23*(1), 41–44. https://doi.org/10.1046/j.1365-2311.1998.00092.x

Hazel, W. N., Smock, R., & Johnson, M. D. (1990). A polygenic model for the evolution and maintenance of conditional strategies. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, *242*(1305), 181–187. https://doi.org/10.1098/rspb.1990.0122

Hazel, W. N., Smock, R., Lively, C. M., & Moore, A. E. A. J. (2004). The ecological genetics of conditional strategies. *The American Naturalist*, *163*(6), 888–900. https://doi.org/10.1086/386313

Herman, J. J., & Sultan, S. E. (2011). Adaptive transgenerational plasticity in plants: Case studies, mechanisms, and implications for natural populations. *Frontiers in Plant Science*, *2*, https://doi.org/10.3389/fpls.2011.00102

Hunt, J., & Simmons, L. W. (2000). Maternal and paternal effects on offspring phenotype in the dung beetle *Onthophagus taurus*. *Evolution*, *54*(3), 936–941. https://doi.org/10.1111/j.0014-3820.2000.tb00093.x

Kotiaho, J. S., Simmons, L. W., Hunt, J., & Tomkins, J. L. (2003). Males influence maternal effects that promote sexual selection: A quantitative genetic experiment with dung beetles *Onthophagus taurus*. *The American Naturalist*, *161*(6), 852–859. https://doi.org/10.1086/375173

Kuijper, B., & Hoyle, R. B. (2015). When to rely on maternal effects and when on phenotypic plasticity? *Evolution*, *69*(4), 950–968. https://doi.org/10.1111/evo.12635

Lang, J. W., & Andrews, H. V. (1994). Temperature-dependent sex determination in crocodilians. *Journal of Experimental Zoology*, *270*(1), 28–44. https://doi.org/10.1002/jez.1402700105

Lively, C. M. (1986). Predator-induced shell dimorphism in the acorn barnacle *Chthamalus anisopoma*. *Evolution*, *40*(2), 232–242. https://doi.org/10.2307/2408804

Maret, T. J., & Collins, J. P. (1997). Ecological origin of morphological diversity: A study of alternative trophic phenotypes in larval salamanders. *Evolution*, *51*(3), 898–905. https://doi.org/10.1111/j.1558-5646.1997.tb03671.x

Marshall, D. J., & Uller, T. (2007). When is a maternal effect adaptive? *Oikos*, *116*(12), 1957–1963. https://doi.org/10.1111/j.2007.0030-1299.16203.x

McCauley, D. E., & Taylor, D. R. (1997). Local population structure and sex ratio: Evolution in gynodioecious plants. *The American Naturalist*, *150*(3), 406–419. https://doi.org/10.1086/286072

Mehrparvar, M., Zytynska, S. E., & Weisser, W. W. (2013). Multiple cues for winged morph production in an aphid metacommunity. *PLoS ONE*, *8*(3), e58323. https://doi.org/10.1371/journal.pone.0058323

Moczek, A. P. (1998). Horn polyphenism in the beetle *Onthophagus taurus*: Larval diet quality and plasticity in parental investment determine adult body size and male horn morphology. *Behavioral Ecology*, *9*(6), 636–641. https://doi.org/10.1093/beheco/9.6.636

Mousseau, T. A., & Fox, C. W. (1998). The adaptive significance of maternal effects. *Trends in Ecology & Evolution*, *13*(10), 403–407. https://doi.org/10.1016/S0169-5347(98)01472-4

Raja, S., Suleman, N., Compton, S. G., & Moore, J. C. (2008). The mechanism of sex ratio adjustment in a pollinating fig wasp. *Proceedings of the Royal Society of London B: Biological Sciences*, *275*(1643), 1603–1610. https://doi.org/10.1098/rspb.2008.0136

Roach, D. A., & Wulff, R. D. (1987). Maternal effects in plants. *Annual Review of Ecology and Systematics*, *18*(1), 209–235. https://doi.org/10.1146/annurev.es.18.110187.001233

Roff, D. A., & Gélinas, M. B. (2003). Phenotypic plasticity and the evolution of trade-offs: The quantitative genetics of resource allocation in the wing dimorphic cricket, *Gryllus firmus*. *Journal of Evolutionary Biology*, *16*(1), 55–63. https://doi.org/10.1046/j.1420-9101.2003.00480.x

Scheiner, S. M. (1993). Genetics and evolution of phenotypic plasticity. *Annual Review of Ecology and Systematics*, *24*(1), 35–68. https://doi.org/10.1146/annurev.es.24.110193.000343

Shuster, S. M., & Wade, M. J. (1991). Equal mating success among male reproductive strategies in a marine isopod. *Nature*, *350*(6319), 608–610. https://doi.org/10.1038/350608a0

Snell-Rood, E. C. (2013). An overview of the evolutionary causes and consequences of behavioural plasticity. *Animal Behaviour*, *85*(5), 1004–1011. https://doi.org/10.1016/j.anbehav.2012.12.031

Snell-Rood, E. C., Van Dyken, J. D., Cruickshank, T., Wade, M. J., & Moczek, A. P. (2010). Toward a population genetic framework of developmental evolution: The costs, limits, and consequences of phenotypic plasticity. *BioEssays*, *32*(1), 71–81. https://doi.org/10.1002/bies.200900132

Stern, D. L., & Emlen, D. J. (1999). The developmental basis for allometry in insects. *Development*, *126*(6), 1091–1101.

Sultan, S. E. (2000). Phenotypic plasticity for plant development, function and life history. *Trends in Plant Science*, *5*(12), 537–542. https://doi.org/10.1016/S1360-1385(00)01797-0

Ueno, H., de Jong, P. W., & Brakefield, P. M. (2004). Genetic basis and fitness consequences of winglessness in the two-spot ladybird beetle, *Adalia bipunctata*. *Heredity*, *93*(3), 283–289. https://doi.org/10.1038/sj.hdy.6800502

Via, S., Gomulkiewicz, R., Jong, G. D., Scheiner, S. M., Schlichting, C. D., & Tienderen, P. H. V. (1995). Adaptive phenotypic plasticity: Consensus and controversy. *Trends in Ecology & Evolution*, *10*(5), 212–217. https://doi.org/10.1016/S0169-5347(00)89061-8

Wade, M. J. (1998). The evolutionary genetics of maternal effects. In T. A. Mousseau & C. W. Fox (Eds.), *Maternal effects as adaptations* (pp. 5–21). Oxford, UK: Oxford University Press.

Walsh, M. R., Castoe, T., Holmes, J., Packer, M., Biles, K., Walsh, M., … Post, D. M. (2016). Local adaptation in transgenerational responses to predators. *Proceedings of the Royal Society B: Biological Sciences*, *283*(1823), 20152271. https://doi.org/10.1098/rspb.2015.2271

Walsh, M. R., Cooley, F., Biles, K., & Munch, S. B. (2015). Predator-induced phenotypic plasticity within- and across-generations: A challenge for theory? *Proceedings of the Royal Society B: Biological*

Sciences, *282*(1798), 20142205. https://doi.org/10.1098/rspb.2014.2205

Wells, C. L., & Pigliucci, M. (2000). Adaptive phenotypic plasticity: The case of heterophylly in aquatic plants. *Perspectives in Plant Ecology, Evolution and Systematics*, *3*(1), 1–18. https://doi.org/10.1078/1433-8319-00001

West, S. A., Shuker, D. M., Sheldon, B. C., & Mueller, U. (2005). Sex-ratio adjustment when relatives interact: A test of constraints on adaptation. *Evolution*, *59*(6), 1211–1228. https://doi.org/10.1554/04-158

Whiteman, H. H. (1994). Evolution of facultative paedomorphosis in salamanders. *The Quarterly Review of Biology*, *69*(2), 205–221. https://doi.org/10.1086/418540

Wilbur, H. M., & Collins, J. P. (1973). Ecological aspects of amphibian metamorphosis: Nonnormal distributions of competitive ability reflect selection for facultative metamorphosis. *Science*, *182*(4119), 1305–1314. https://doi.org/10.1126/science.182.4119.1305

Wolf, J. B., & Wade, M. J. (2009). What are maternal effects (and what are they not)? *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, *364*(1520), 1107–1115. https://doi.org/10.1098/rstb.2008.0238

Wolf, J. B., & Wade, M. J. (2016). Evolutionary genetics of maternal effects. *Evolution*, *70*(4), 827–839. https://doi.org/10.1111/evo.12905

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