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A THEORETICAL MODEL OF THE EVOLUTION OF MATERNAL EFFECTS UNDER PARENT–OFFSPRING CONFLICT

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The evolution of maternal effects on offspring phenotype should depend on the extent of parent–offspring conflict and costs and constraints associated with maternal and offspring strategies. Here, we develop a model of maternal effects on offspring dispersal phenotype under parent–offspring conflict to evaluate such dependence. In the absence of evolutionary constraints and costs, offspring evolve dispersal rates from different patch types that reflect their own, rather than the maternal, optima. This result also holds true when offspring are unable to assess their own environment because the maternal phenotype provides an additional source of information. Consequently, maternal effects on offspring diapause, dispersal, and other traits that do not necessarily represent costly resource investment are more likely to maximize offspring than maternal fitness. However, when trait expression was costly, the evolutionarily stable dispersal rates tended to deviate from those under both maternal and offspring control. We use our results to (re)interpret some recent work on maternal effects and their adaptive value and provide suggestions for future work.

KEY WORDS: **Dispersal, maternal effects, parent–offspring conflict, phenotypic plasticity.**

There has been a steady increase in the interest in ecology and evolution of parental effects, that is, a causal effect of the parental phenotype or parental environment on offspring development (Mousseau and Fox 1998; Uller et al. 2009). From being mainly a concern of quantitative genetics to account for their effect on the response to selection (Dickerson 1947; Willham 1972; Cheverud 1984; Riska et al. 1985; Kirkpatrick and Lande 1989; Cheverud and Moore 1994) parental effects are now a major focus for behavioral and evolutionary ecologists (e.g., Mousseau and Fox 1998; Groothuis et al. 2005; Boulinier and Staszewski 2008; Crean and Marshall 2009; Donohue 2009; Duckworth 2009; Russell and Lummaa 2009). The latter commonly interpret parental effects as adaptive outcomes of strategies that enable short-term adjustment of offspring phenotype to match selection in a fluctuating environment (e.g., Fox and Mousseau 1998; Galloway and Etterson 2007; Crean and Marshall 2009) or as byproducts of constraints imposed on parents or offspring (e.g., Cohen 1979; Heath and Blouw 1998; Uller et al. 2004; Brown and Shine 2009). Although both position have their merits, they do not capture the dynamic evolution of parental effects (Badyaev and Uller 2009). For example, divergent selection across generations, constraints on information acquisition or processing, and energetic costs may modify interactions between parents and offspring, but their importance in determining the phenotypic outcomes that comprise parental effects remain poorly understood.

Three components of the evolution of parental (from now on maternal) effects by natural selection are particularly contentious: antagonistic selection on parents and offspring, costs to

parents and offspring, and constraints on information acquisition or the evolution of counter responses to strategies of the other party (reviewed in Uller 2008). First, life-history theory suggests that parent–offspring conflict should be common as a result of a discrepancy between fitness-maximizing strategies for parents versus offspring (Trivers 1974; Godfray 1995; Mock and Parker 1997). Although mothers may be able to impose their own optima on the offspring when the scope for offspring counter responses is limited (Marshall and Uller 2007), the evolutionary outcome when both maternal and offspring strategies are allowed to vary is more difficult to predict. The majority of work to date has focused on the outcome of static conflicts, that is, conflicts that are constant across time and space (reviewed in Clutton-Brock 1991; Mock and Parker 1997; Smiseth et al. 2008). However, parental effects emphasize the environmental contingency of such conflicts (Badyaev and Uller 2009). Second, most theoretical studies have considered costly parental effects, such as resource provisioning to offspring (reviewed in Mock and Parker 1997; Mousseau and Fox 1998). However, maternal effects may also involve offspring responses to variation in parental phenotypes that do not incur costs to the parents or vice versa (e.g., Mousseau and Dingle 1991; Muller et al. 2007; Uller 2008). Finally, there is a general assertion that constraints on detection or responses to maternal or offspring strategies should be important for the evolution of maternal effects (e.g., Groothuis et al. 2005; Groothuis and Schwabl 2008; Uller 2008), but there has been limited development of a formal theory.

Our goal in this article is to contribute some insights regarding the importance of conflicts, costs, and constraints for adaptive evolution of maternal effects in a heterogeneous environment. We therefore aimed for a model that fulfills the following criteria: (1) The optimal offspring phenotype must differ for mothers and offspring, (2) environments must vary in time and/or space, (3) environmental conditions across generations should be predictable, but the predictability of the environment for mothers and offspring can vary, (4) the mechanisms of maternal effects can involve costs in terms of reduced survival or reproductive output for mothers and offspring, (5) offspring can be constrained to be unable to "ignore" maternal transmission of developmental factors. Given the complexity of incorporating all these aspects into a single model, we opted for a well-established system of parent–offspring conflict over dispersal resulting from competition between related offspring (Frank 1986; Motro 1983; Taylor 1988). This allowed us to partly derive the basic results analytically and expand the analyses using individual-based simulation models. We emphasize that our goal is to clarify the most fundamental questions regarding how maternal effects evolve in response to selection on parental and offspring strategies by using a general model with dispersal as the offspring trait, not to specifically address dispersal evolution per se. We use our results to evaluate adaptive scenarios of maternal effects previously proposed in the literature.

The Model

The model consists of a meta-population with M patch types with type i occurring at frequency p_i. Each patch can only hold a small number (N) of individuals that therefore compete for limited resources (i.e., space). Dispersal from one patch to another is associated with a mortality cost incurred by the offspring ($0 \leq c_i \leq 1$; assigned to the patch from which dispersal occurs) that can differ for the patch types. Biologically, site-specific dispersal costs may result from differences in the risk of mortality due to, for example, predation risk associated with movement through different habitats, site-specific benefits of parental care or costs associated with abandoning the social group, or environmental and maternal factors that influence the cost of allocation of resources to dispersal versus other life-history traits (see Clobert et al. 2001, 2004; Ronce 2007 for reviews). It is a well-established result that costs of dispersal under conditions where related individuals compete for limited resources (in this case space within a patch) generate parent–offspring conflict over the optimal dispersal rate (Motro 1983; Frank 1986; Taylor 1988). Patch-specific dispersal costs therefore select for different optimal dispersal rates from each patch type under parental versus offspring control (parental control results in higher dispersal rates; e.g., Motro 1983; Frank 1986; Taylor 1988; Ronce et al. 1998; Starrfeldt and Kokko 2010; see below for the analytical version of the present model).

ANALYTICAL MODEL

Here, we first describe details of the analytical model that we used to calculate optimal offspring dispersal rates under offspring or maternal control. Unlike in most of the simulations (see below), we assume that individuals can perfectly assess environmental quality.

Individuals are diploid simultaneous hermaphrodites and generations are nonoverlapping. Offspring disperse if a certain amount of "dispersal substance" x in the offspring's body exceeds a fixed threshold value t_d. The amount of substance is under maternal or offspring control, up to some Gaussian noise ε with mean zero and standard deviation σ_ε. The probability of dispersal as a function of the amount of dispersal substance is then given by

$$y(x) = \frac{1}{2} + \frac{1}{2}\mathrm{erf}\left(\frac{x - t_d}{\sqrt{2}\sigma_\varepsilon}\right). \quad (1)$$

Here $\mathrm{erf}(z) = (2/\sqrt{\pi}) \int_0^z \exp(-\frac{1}{2}x^2/\sigma_\varepsilon^2)\,dx$ is the well-known error function. The resulting function $y(x)$ has an S-shaped or "logistic" form that approaches a step function or switch as σ_ε approaches zero.

We assume that dispersal may be adjusted to patch type i, so we write x_i and $y_i = y(x_i)$ for patch type-specific values of

x and y. Production of dispersal substance may be costly: maternal clutch size and/or offspring survival before reproduction declines exponentially with the amount of dispersal substance produced according to exp(-ax).

There are M patch types with type j occurring at frequency p_j ($M = 2$ in the simulations; see below). We use bars above symbols to indicate mean values in a focal patch and hats ($\hat{}$) to indicate population-wide mean values. The direct fitness of an offspring with amount of dispersal substance x_i in a focal patch of type i is then given by

$$W_i(x_i, y_i, \bar{y}_i) = \exp(-ax_i)\left[\frac{1-y_i}{1-\bar{y}_i+\hat{z}} + y_i(1-c_i)\sum_{j=1}^{M}\frac{p_j}{1-\hat{y}_j+\hat{z}}\right]. \quad (2)$$

Here $\hat{z} = \sum_{k=1}^{M} p_k\hat{y}_k(1-c_k)$ is the mean number of immigrants per patch.

Using the direct fitness approach (Taylor and Frank 1996), selection gradients for the x_i can be calculated as

$$\frac{\mathrm{d}W_i}{\mathrm{d}x_i} = r\left[\frac{\partial W_i}{\partial x_i} + \frac{\partial W_i}{\partial y_i}\frac{\mathrm{d}y_i}{\mathrm{d}x_i}\right] + R\frac{\partial W_i}{\partial \bar{y}_i}\frac{\mathrm{d}\bar{y}_i}{\mathrm{d}x_i}. \quad (3)$$

If the within-patch variation in x_i is small, we can approximate $\bar{y}_i$ by $y(\bar{x}_i)$and the derivatives of y are given by Gaussian functions: $\mathrm{d}y/\mathrm{d}\xi = \exp(-\frac{1}{2}(\xi - t_\mathrm{d})^2/\sigma_\varepsilon^2)(2\pi\sigma_\varepsilon^2)^{-1/2}$,where ξ is either x_i or $\bar{x}_i$.

The coefficients r and R in equation (3) are coefficients of relatedness, the values of which depend on whose genes we give control over the amount of dispersal substance. Following the method of Taylor (1988), they can be expressed in terms of the coefficient of consanguinity g between (singly mated) offspring born in the same patch, and all other model parameters. Specifically, under offspring control of dispersal, r refers to the offspring's relatedness to itself and R its relatedness to a random offspring from the same patch

$$r = 1, \quad R = \frac{4g}{1+3g}. \quad (4)$$

R is the ratio of consanguinity of alleles drawn from two random offspring born on the same patch (equal to g by definition), and the consanguinity of alleles drawn from the same individual, where one allele is drawn from the individual's two alleles at its own gene locus (assumed to be in control of dispersal), and the other from the four alleles at the individual's own locus plus at the same locus from the sperm the individual is carrying. Therefore, with probability $\frac{1}{2}$ an allele is drawn from the individual's own genome twice, in which case the alleles are identical by descent (IBD) with probability $\frac{1}{2} + \frac{1}{2}f$, where f is the inbreeding coefficient. But in equilibrium under random mating f must equal g, hence we can replace f with g. With complementary probability $\frac{1}{2}$an allele from the individual's sperm storage (its random mate) is drawn, which is IBD to the individual's own allele with probability g. Hence R is given by $g/(\frac{1}{2}(\frac{1}{2} + \frac{1}{2}g) + \frac{1}{2}g)$ which is the same as in (4).

Under maternal control r is her own offspring's relatedness to her, taking into account that her offspring carry sperm from random mates, and R that of random patch-offspring to her. These work out to be given by

$$r = \frac{1+7g}{2+6g}, \quad R = \frac{r}{n} + \frac{n-1}{n}\frac{(2+2h)g}{1+3g}. \quad (5)$$

Here n is the number of mothers per patch and h is the probability that two random mothers living in the same patch are both native to the patch, which can be approximated by

$$h = \sum_{i=1}^{M} p_i\left[\frac{1-\hat{y}_i}{1-\hat{y}_i+\hat{z}}\right]^2. \quad (6)$$

Finally, we can set up a recursion for g, which in equilibrium gives the following equation for g:

$$g_{t+1} = \frac{1}{n}\left(\frac{1}{4} + \frac{3}{4}g\right) + \frac{n-1}{n}hg_t. \quad (7)$$

This equation can be solved for g, assuming that g reaches equilibrium. Because patch types and patch compositions fluctuate, this is an approximation at best, but the individual-based simulations described below indicate that it approximates the mean value of g quite well.

"Optimal" dispersal rates are then calculated by plugging the equilibrium values of g into (4) or (5), depending who is in control, and then solving for the roots $\hat{\mathbf{x}} = (\hat{x}_1, \hat{x}_2)$of equation (3). Usually multiple roots were found, but in all cases only a single one was convergence stable in the sense that the matrix

$$\begin{pmatrix} \frac{\partial}{\partial \hat{x}_1}\left.\frac{\mathrm{d}W_1}{\mathrm{d}x_1}\right|_{\mathbf{x}=\hat{\mathbf{x}}} & \frac{\partial}{\partial \hat{x}_2}\left.\frac{\mathrm{d}W_1}{\mathrm{d}x_1}\right|_{\mathbf{x}=\hat{\mathbf{x}}} \\ \frac{\partial}{\partial \hat{x}_1}\left.\frac{\mathrm{d}W_2}{\mathrm{d}x_2}\right|_{\mathbf{x}=\hat{\mathbf{x}}} & \frac{\partial}{\partial \hat{x}_2}\left.\frac{\mathrm{d}W_2}{\mathrm{d}x_2}\right|_{\mathbf{x}=\hat{\mathbf{x}}} \end{pmatrix} \quad (8)$$

was negative definite (all eigenvalues having negative real part).The solutions were used to derive the expected optimal dispersal rates under maternal versus offspring control, which were subsequently compared to the outcomes of the simulation models (see below).

SIMULATION MODEL

The life history of our simulation model follows a simple structure (Fig. 1). Individuals are diploid simultaneous hermaphrodites and generations are nonoverlapping. The mother first assesses the patch type she is in with more or less accuracy ($0 \le e_\mathrm{m} \le 0.5$) and transfers an amount m_i of a dispersal-inducing substance to all her offspring according to patch type immediately before oviposition. This transfer is either cost-free or it reduces the mother's expected

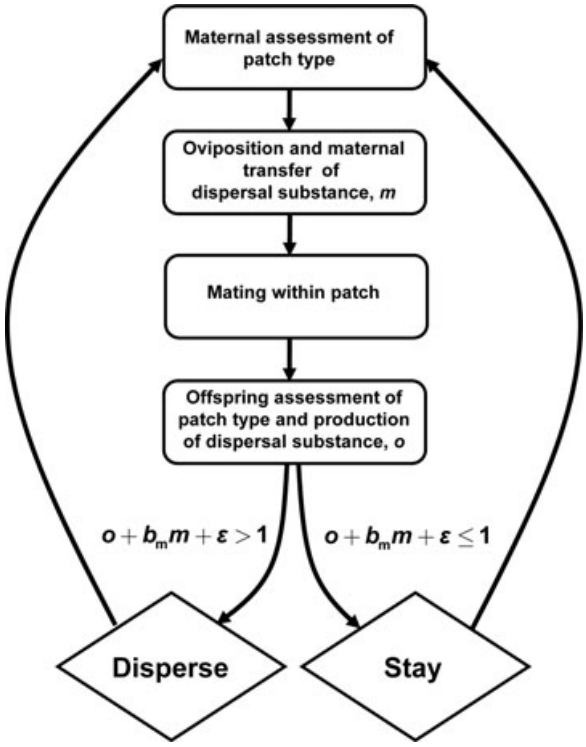

Figure 1. The life history of the simulation model. There are two types of patches and patch type is randomized each round before maternal assessment. See text for details.

clutch size according to $\exp(-am_i)$, which represents the maternal cost involved with maternal "programming" of offspring phenotype (note that this cost is not the same as the cost of dispersal that generate parent–offspring conflict). Offspring can "ignore" their mother's input because they have genes (the b_m locus) that can neutralize the maternal input to some extent. Offspring hatch, assess the patch type with some degree of error ($0 \leq e_o \leq 0.5$) and produce a level o_i of the same dispersal-inducing substance. Either the substance is cost-free or it reduces offspring survival according to $\exp(-ao_i)$, which represents the cost of development of the dispersal strategy to offspring (as with the maternal cost of production of the dispersal substance, this cost is distinct from the cost of dispersal that generates parent–offspring conflict in the first place). Mating occurs at random within patches without reciprocal sperm transfer. If the value of the combination of maternal and offspring production of the substance (i.e., $x_i = o_i + b_m m_i + \varepsilon$; where ε is an error term) exceeds a certain threshold, t_d, offspring will disperse, else they will stay in the same patch. Individuals that disperse are randomly assigned to a different patch and each patch is subsequently filled up to carrying capacity using the combined pool of dispersing and sedentary offspring. Finally, the patch type is randomized to become type j with probability $p_j = 1/M$, where M is the number of patch types ($M = 2$ in the simulations), before the cycle starts anew with maternal assessment of patch type.

The basic model includes five diploid loci (Table 1): two maternal loci, one each for maternal production of the dispersal-inducing substance for the two different patch types (m_1 and m_2), and three offspring loci, one each for offspring production of the dispersal-inducing substance for each patch type (o_1 and o_2), and one that weighs the maternal input (b_m). Because maternal manipulation of offspring phenotype may involve transmission of dishonest signals we also ran simulations in which we allowed the accuracy by which mothers assess their own patch (i.e., e_m) to evolve (see Results). We kept the errors symmetrical, that is, independent of patch type. We also ran some simulations where only mothers on bad patches (i.e., with high dispersal costs) made mistakes, but this did not affect the outcome (results not shown). Separate and unlinked loci for each of the patches mean that the dispersal rates are allowed to evolve to their optimal

Table 1. Variables and parameters of the model.

Variable/parameter	Meaning	Simulation value(s)
n_p	Number of patches	2500
n_a	Number of adults per patch	4
k	Clutch size	10
m_i	Maternal input in patch type i	evolvable ($\geq$0)
o_i	Offspring input in patch type i	evolvable ($\geq$0)
b_m	Offspring weighing of maternal input	evolvable ($\geq$0)
ε	Gaussian noise added to input	N(0, σ_ε)
σ_ε	Standard deviation noise	0.1
e_m	Maternal accuracy of patch assessment	$0 \leq e_m \leq 0.5$
e_o	Offspring accuracy of patch assessment	$0 \leq e_o \leq 0.5$
c_i	Cost of dispersal from patch type i	$c_1 = 0$; $c_2 = 0.4$
a_i	Scales cost of input ($\exp(-a \times \text{input})$)	0 – 0.5
μ	Mutation rate/allele/generation	0.001
σ_μ	SD Gaussian mutation step size	0.01

values and not be constrained by, for example, genetic correlations that could otherwise bias or constrain the evolution of phenotypic plasticity (Pigliucci 2001). This is important because we are concerned with how maternal effects evolve under parent–offspring conflict when both mothers and offspring can achieve their optimal environment-specific strategies in the absence of counter-strategies by the other party. We did not allow b_m to be patch-specific. In biological terms this means that "neutralization" of maternal input is a decision that occurs before offspring are able to assess their own environment.

In summary, this model therefore involves parent–offspring conflict over offspring phenotype [condition (1)], a variable environment [condition (2)], predictability of environmental conditions across generations, which itself can be varied [condition (3)], a potential cost of maternal manipulation of offspring phenotype and a potential cost of offspring development of the same phenotype [condition (4)], and a means by which offspring can be constrained to respond to maternal allocation [condition (5)]. Thus, this model allows us to assess whether costs or constraints involved in maternal and offspring strategies modify the evolution of maternal effects and to what extent the outcome reflects the optimal dispersal rate that evolves in the absence of the evolution of counter-strategies by the other party, which were verified analytically (see above).

SIMULATION SETTINGS

All simulations in the present article are based on a model with two patch types. We fixed the threshold at 1, without loss of generality; dispersal rates therefore evolve through the production of dispersal factor and offspring weighing of the maternal input. Offspring disperse from patch type i if

$$o_i + b_m m_i + \varepsilon > 1 \quad (9)$$

and remain in their natal patch otherwise, where ε is a Gaussian error term with mean zero and standard deviation $\sigma_\varepsilon = 0.1$.

We initially ran simulations in which the costs of dispersal was set to $c_1 = 0$ and $c_2 = 0.4$ for patch type one and two, respectively, as this generated relatively large differences in optimal dispersal under parental versus offspring control, which enabled high resolution for assessing whether changes in the model affected the dispersal rates toward maternal or offspring optima. In addition, the model outcome is somewhat sensitive to the number of individuals per patch relative to clutch size as this will affect the strength of kin competition and hence the extent to which optimal dispersal rates differs for mothers and offspring (e.g., Frank 1986). However, those aspects all refer to the strength of conflict that, although interesting, is something that represents a basic feature of our model rather than a target for theoretical analysis (see Motro 1983, Frank 1986; Taylor 1988 for details). Throughout all simulations, we fixed the carrying capacity of each patch to 4 and clutch size to 10. Mutation rates for all loci were set to $\mu = 0.001$ per allele per generation and mutational step sizes were drawn from a normal distribution with mean zero and standard deviation $\sigma_\mu = 0.01$. A summary of the parameter settings can be found in Table 1. We verified the generality of our results by varying the cost of dispersal (and therefore optimal dispersal rates for mothers and offspring) but here report only results from our original settings.

Simulation Results

BASIC MODEL

When mothers were in full control of dispersal (m-alleles were allowed to mutate but evolution of o-alleles and b_m-alleles was "switched off"), the dispersal rates of our simulations coincided with analytical results (see above) for the maternal dispersal optima (Fig. 2). These results also showed stabilizing selection on m_1 and m_2, that is, they were consistent with the analytical results of a single optimal dispersal rate from each patch type. Conversely, with fixed maternal production of the dispersal factor and evolvable offspring production of dispersal factor, the dispersal rates rapidly converged on the analytically predicted offspring optima.

More interestingly, allowing both maternal and offspring alleles to evolve always resulted in dispersal rates close to the offspring, not the maternal, optima for both patch types (Fig. 2). As evident from the example in Figure 2B, this result arises through a combination of a devaluation of the maternal factor via evolution of b_m and evolution of o_1 and o_2. The maternal production of dispersal factor goes up over time, but is countered by offspring devaluation (b_m) and fine-tuning by offspring production of dispersal factor (o_1 and o_2; Fig. 2). The distributions of the alleles for each locus showed that all loci were subject to both directional and stabilizing selection as a result of coevolution of maternal and offspring strategies.

CONSTRAINTS

Two types of "constraints" can be included in the model: offspring inability to ignore maternal transfer of the dispersal factor or insufficient information to assess patch type for mothers, offspring, or both. We first constrained offspring to respond to maternal strategies (i.e., fixing b_m at some level between 0 and 1). This resulted in dispersal rates at the maternal optima for both patches. The reason that it does not lead to a continuous arms race between maternal and offspring alleles (as occurs when offspring are unconstrained; see above) is that the maternal optimal dispersal rates from both patch types always are higher than for the offspring. Selection therefore favors zero production of dispersal factors by the offspring, because any additional factor would lead to a dispersal rate that would be even larger than the maternal optima. Thus, as long as maternal production does not impose a cost on

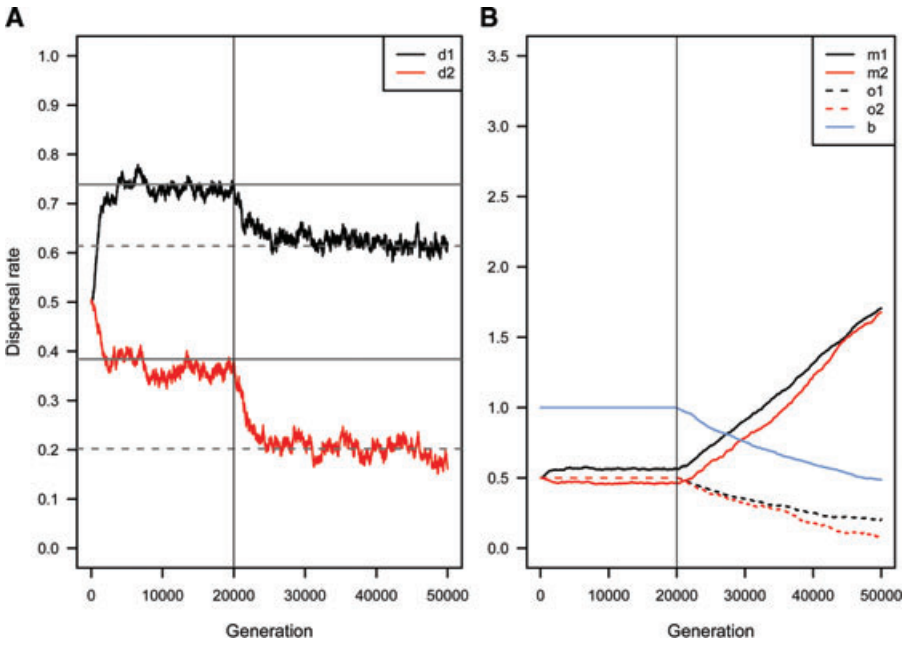

Figure 2. **A representative example of evolution of patch-specific dispersal in the absence of costs and constraints. (A) Dispersal rates from the two different patch types. For the first 20,000 generations only evolution of maternal loci (m_1, m_2) is allowed. Evolution of offspring loci (b_m, o_1, o_2) is enabled at generation 20,000, whereafter dispersal rates rapidly converge on the offspring optima. Solid lines indicate the optimal maternal dispersal rates for the two patch types and dashed lines indicate the optimal dispersal rates for offspring (from analytical solution). (B) Evolution of the five loci in the simulation generating the dispersal rates in (A).**

mothers (see below), fixing b_m at different levels does not affect the outcome.

We subsequently addressed to what extent the accuracy by which offspring and mothers can assess their environment affect the observed outcome. Not surprisingly did impaired accuracy in the assessment of patch type reduce the difference in optimal dispersal rates between the patch types (Fig. 3). However, the level of maternal transfer of the dispersal factor provides an alternative source of information about patch type. Offspring dispersal rates therefore closely tracked their theoretical optima even if offspring were themselves completely ignorant of their own environment (Fig. 3). Thus, maternal control of offspring phenotype in this model is not a stable outcome under conflicting selection even when offspring have limited or no ability to assess their own environment directly.

Introducing maternal error into the model affects the dispersal rates from both patches (Fig. 3). However, when allowed to evolve, maternal error rapidly evolved toward zero regardless of the initial model settings (Fig. S1).

COSTS

The previous models all assume that production and transfer of the dispersal factor comes without a cost to mothers or offspring. However, the evolutionary dynamics for maternal effects that involve costs associated with maternal transmission or offspring development may be different (Uller 2008). We therefore expanded our original model to include a cost, so that offspring survival or maternal clutch size declined exponentially with the amount of dispersal substance produced.

Costs associated with maternal transmission and offspring production of dispersal factor modifies the dispersal rates from both patches (Figs. 4 and S2). For "bad" patches with high dispersal mortality, the dispersal rate always evolves to zero except when costs are very small. When the cost to offspring is small relative to the cost to mothers, both m_1 and m_2 go toward zero, whereas o_1 and o_2 remain positive. The dispersal rates in the simulation are therefore close to the offspring optima derived analytically (Fig. 4). As the cost to offspring increases, o_1 and o_2 decline, whereas m_1 and b_m are subject to stabilizing selection at nonzero values (Fig. S2). The resulting dispersal rates are typically lower than both maternal and offspring optima (Fig.4). Consequently, the maternal effects on dispersal, and to what extent they correspond to maternal and offspring optima in the absence of evolution of counter-strategies by the other party, will depend on the magnitude of costs involved with maternal allocation and development of offspring phenotype.

Discussion

We have analyzed a model of the evolution of maternal effects on offspring dispersal in a heterogeneous environment with patches of low carrying capacity, where the cost of dispersal to the

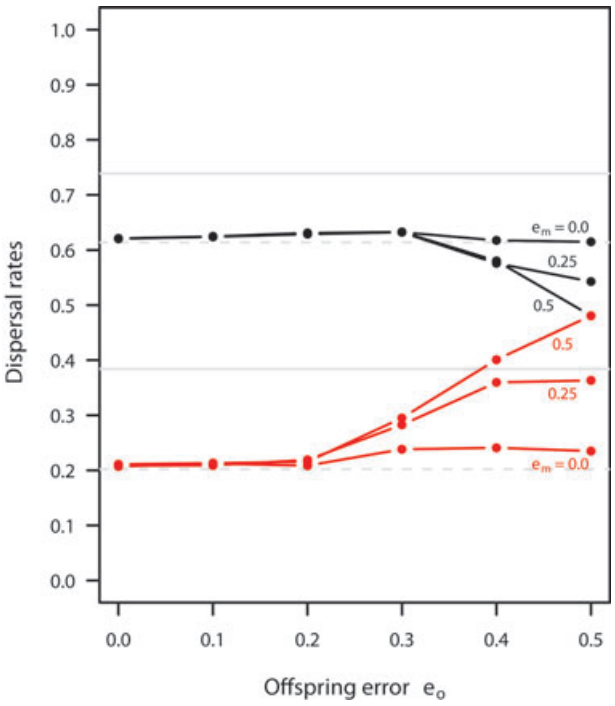

Figure 3. Equilibrium dispersal rates from the two different patch types (black and red lines) for different information constraints. Solid gray lines indicate the optimal maternal dispersal rates for the two patch types and dashed gray lines indicate the optimal dispersal rates for offspring under complete information (from analytical model). An error of zero means that mothers or offspring always assess the patch type accurately. An error of 0.5 represents a complete lack of direct information about patch type. Note that if both mother and offspring have no information about patch type, then equilibrium dispersal rates from both patch types are identical. Simulations were highly consistent in their outcome and confidence intervals therefore too small to be included.

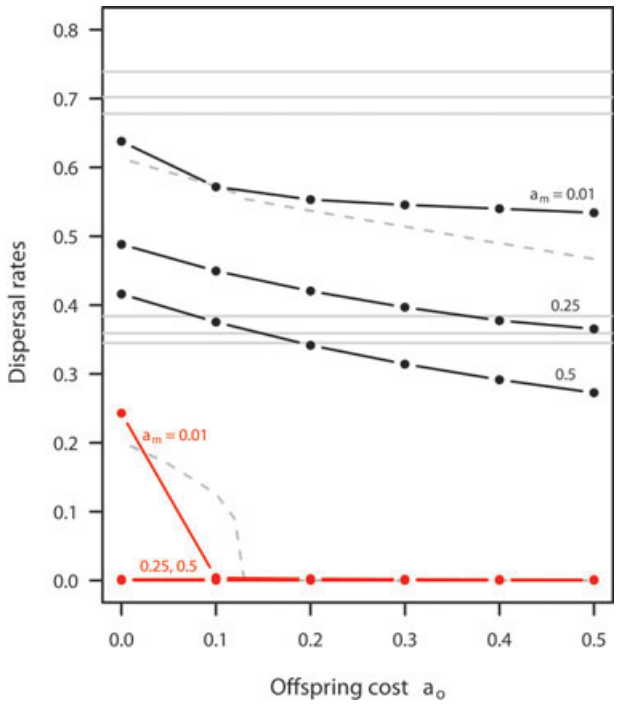

Figure 4. Equilibrium dispersal rates form the two different patch types (black and red lines) for different levels of cost of maternal or offspring production of dispersal substance. Solid gray lines indicate the optimal maternal dispersal rates for the two patch types for three cost levels (from top to bottom a_m = 0.01, 0.25, 0.5) and dashed gray lines indicate the optimal dispersal rates for offspring (from analytical model). Costs modify the optimal dispersal of both mothers and offspring. Whether the evolved outcome is closer to cost-specific dispersal rates for mothers or offspring depend on the magnitude of both maternal and offspring costs (see text for details).

offspring could be patch specific. A low carrying capacity of each patch results in competition between kin, which results in higher optimal rates of offspring dispersal from the maternal perspective than from the offspring perspective (e.g., Motro 1983; Frank 1986; Taylor 1988). Thus, this basic model framework allowed us to address how a focal trait in offspring (i.e., dispersal), which potentially could be subject to maternal effects, evolves under maternal–offspring conflict. Furthermore, we could assess to what extent the evolution of dispersal rates were affected by constraints on evolutionary counter-responses and costs associated with maternal manipulation and offspring counter-responses.

Our simulation and analytical models provided four principal results for the evolution of maternal effects under mother–offspring conflict. First, if offspring can freely respond to maternal strategies, the maternal effect (i.e., the effect that the maternal environment has on offspring phenotype) will reflect the offspring optimum. Second, this applies even when offspring are unable to assess their own environment. This, perhaps counter-intuitive, result occurs because the maternal allocation strategy naturally correlates with the environment and thus provides an alternative source of information for offspring. Third, if offspring are constrained to respond to maternal strategies, the maternal effect evolves to reflect the maternal optimum. Fourth, costs associated with maternal and offspring strategies generally results in maternal effects that do not correspond to the theoretical optimal maternal effect for either mothers or offspring. Furthermore, the deviation from maternal and offspring optima depends on the absolute and relative magnitude of the costs.

Despite the simplicity of our model framework, the results clarify several contentious issues in maternal effects research. For example, it is often asserted that conflicting selection on mothers and offspring can lead to maternal effects that reflect the mother's optimal strategy, the offspring's optimal strategy, or neither optimum (e.g., Schwabl et al. 1997; Marshall and Uller 2007; Groothuis et al. 2005; Russell and Lummaa 2009). Yet, the conditions that promote one or the other outcomes are poorly

understood (Muller et al. 2007; Uller 2008; Hinde et al. 2010), despite that this is fundamental to interpretation of maternal effects in, for example, behavioral ecology. Our model of the evolution of maternal effects on dispersal under parent–offspring conflict in a heterogeneous, yet predictable, environment unambiguously shows that (given the assumptions of our model) maternal effects will reflect the offspring's optima in the absence of costs and constraints on offspring strategies (see also Motro 1983; Frank 1986; Ronce et al. 1998). Importantly, the dispersal rates reflected the offspring optima in our model even when offspring were unable to assess their own environment. This is because maternal allocation of dispersal factor to offspring provides an alternative source of information regarding the environment that enables coevolution of maternal and offspring phenotypes. Consequently, offspring inability to assess their environment or to distinguish between maternal allocation and their production of the same substance (e.g., a hormone) may be unlikely to allow maternal manipulation of offspring phenotype using cost-free signals (see also Haig 1996 for a model where hormone signaling is costly).

These results have consequences for the interpretation of maternal effects involving cost-free signals. For example, contrary to what is frequently assumed or concluded (e.g., Schwabl 1993; Schwabl et al. 1997; Love and Williams 2008; Russell and Lummaa 2009), maternal effects on offspring dispersal (Mousseau and Dingle 1991; Simpson and Miller 2007), diapause (Danks 1987; Huestis and Marshall 2006), and other behaviors (Groothuis et al. 2005) that involve signals that incur a minor cost on the mother (e.g., hormones or other factors that may modify DNA expression via epigenetic mechanisms) are unlikely to reflect the maternal optima (unless, of course, there is no parent–offspring conflict; see also Muller et al. 2007). Nevertheless, it is possible that more complex scenarios that relax our assumptions may generate alternative results. For example, in many birds with hatching asynchrony, maternal yolk hormone levels differ both between eggs within broods and among broods and this has significant effects on offspring development, growth, and begging behavior (reviewed in Groothuis et al. 2005; Carere and Balthazart 2007). It has been suggested that the within-brood variation of yolk hormones reflects the maternal optima and allows mothers to 'handicap' the last-hatching chick, thereby facilitating brood reduction (e.g., Schwabl et al. 1997; Love and Williams 2008). Although our results suggest this is unlikely to be evolutionarily stable, the added level of complexity that may arise from variation in hormone levels among broods could potentially prevent offspring from evolving efficient counter-responses to maternal manipulation. More likely, however, is that offspring under most conditions will evolve to adjust their phenotype according to their own optima or simply become insensitive to variation in maternal phenotype. Although this can lead to loss of environment-specific maternal strategies, this need not be the case. In fact, there is an infinite number of strategy sets (combination of allele values) resulting in the same dispersal rates in our model. Consequently, patterns of, for example, maternal allocation of hormones and other substances to eggs or offspring across populations or species (e.g., Gil et al. 2007; Martin and Schwabl 2008) are not necessarily informative with respect to the evolution of maternal effects when maternal and offspring optima differ. Experimental methods that target both maternal and offspring responses will often be necessary to address whether a particular maternal effect represent adaptive modification of offspring phenotype and, more generally, to dissect the evolutionary and ecological implications of maternal effects.

Although the dispersal rates from both patch types evolved to the offspring's optima even when offspring were unable to assess the patch type themselves, it evolved to the maternal optima when offspring were constrained to respond to the maternal strategy. This supports that developmental and physiological constraints on counter responses are important for understanding the evolution of maternal effects (e.g., Haig 1996; Groothuis et al. 2005; Uller 2008; Hinde et al. 2010). However, it is important to emphasize that this outcome depends on the specifics of our model; no offspring counter-strategies that would increase offspring fitness given the maternal optima were possible via evolution of allelic values. In other scenarios, a constraint on offspring to respond to developmental factors allocated by mothers may lead to alternative counter-strategies and therefore ultimately environment-specific phenotypes at the offspring optima. The underlying mechanisms are therefore crucial determinants of the outcome of the joint evolution of maternal and offspring strategies that comprise maternal effects by dictating the opportunity for counter responses to maternal or offspring strategies.

Several authors have suggested that the extent to which maternal effects incur costs may affect their evolutionary dynamics (reviewed in Müller et al. 2007; Uller 2008). The majority of the literature on parent–offspring conflict deals with parental investment in terms of food provisioning in a single environment (e.g., Godfray 1995; Haig 1996; Mock and Parker 1997; Kilner and Hinde 2008). Our model does not directly address this scenario, because parental transfer of resources is not necessary for offspring development (i.e., contrary to food provisioning, offspring can develop a fully functional phenotype even in the absence of maternal transmission of the dispersal substance). However, we included costs of maternal effects in terms of fecundity (for mothers) or survival (for offspring) and, thus, directly addressed the importance of costs for the evolving patterns of maternal effects across environments. The results showed that whereas dispersal rates always evolved to the offspring optima when maternal and offspring production of dispersal substance were cost free, they deviated more or less from both maternal and offspring optima when they were costly. Furthermore, the magnitude and

direction of the deviation depended on the magnitude of the cost and whether it was incurred by mothers, offspring, or both. As expected, relatively high maternal costs resulted in reduced maternal transmission of dispersal factor and, accordingly, dispersal rates close to the offspring optima. However, when both maternal and offspring costs are incurred, phenotypes can deviate substantially from both the maternal and offspring optima (i.e., the dispersal rate when the other party is unable to evolve counter-strategies). These results indicate that costs associated with maternal and offspring strategies will be very important for the evolution of maternal effects. We therefore suggest that variation in patterns of maternal effects among populations may often be a result of variation in the costs involved. Consequently, documenting costs (and benefits) of maternal and offspring strategies are important to interpret the adaptive significance of maternal effects (Groothuis et al. 2005; Groothuis and Schwabl 2008).

In summary, our model of the evolution of maternal effects in a heterogeneous environment under parent–offspring conflict shows that costs and constraints on counter-strategies or information processing can have important effects on the observed outcome. Maternal manipulation of offspring phenotype was not evolutionarily stable, suggesting that maternal effects on dispersal, diapause and other similar phenotypes generally should reflect the offspring optima if there is parent–offspring conflict. Costs involved with maternal or offspring strategies frequently resulted in dispersal rates that did not correspond to either optima (i.e., the phenotype evolving in the absence of counter-strategies), suggesting that interpretation of whether maternal effects maximize the fitness of offspring, parents, or neither will depend on whether they also are costly.

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LITERATURE CITED

Badyaev, A. V., and T. Uller. 2009. Parental effects in ecology and evolution: mechanisms, processes and implications. Philos. Trans. R. Soc. Lond. B 364:1169–1177.

Boulinier, T., and V. Staszewski. 2008. Maternal transfer of antibodies: raising immuno-ecology issues. Trends Ecol. Evol. 23:282–288.

Brown, G. P., and R. Shine. 2009. Beyond size-number trade-offs: clutch size as a maternal effect. Philos. Trans. Roy. Soc. B 364:1097–1106.

Carere, C., and J. Balthazart. 2007. Sexual versus individual differentiation: the controversial role of avian maternal hormones. Trends Endocrinol. Metabol. 18:73–80.

Cheverud, J. M. 1984. Evolution by kin selection—a quantitative genetic model illustrated by maternal performance in mice. Evolution 38:766–777.

Cheverud, J. M., and A. J. Moore. 1994. Quantitative genetics and the role of the environment provided by relatives in behavioral evolution. Pp. 67–100 *in* C. Boake, eds. Quantitative genetic studies of behavioral evolution. Univ. of Chicago Press, Chicago.

Clobert, J., E. Danchin, A. A. Dhont, and J. D. Nichols. 2001. Dispersal. Oxford Univ. Press, New York, NY.

Clobert, J., R. A. Ims, and F. Rousset. 2004. Causes, mechanisms and consequences of dispersal. Pp. 307–335 *in* I. Hanski and O. E. Gaggiotti, eds. Ecology, genetics, and evolution of meta-populations. Elsevier Academic Press, Burlington, MA.

Clutton-Brock, T. 1991. The evolution of parental care. Princeton Univ. Press, Princeton, NJ.

Cohen, J. 1979. Maternal constraints on development. Pp. 1–8 *in* D. R. Newth and M. Balls, eds. Maternal effects in development. Cambridge Univ. Press, Cambridge, UK.

Crean, A. J., and D. J. Marshall. 2009. Coping with environmental uncertainty: dynamic bet hedging as a maternal effect. Philos. Trans. R. Soc. Lond. B 364:1087–1096.

Danks, H. V. 1987. Insect dormancy: an ecological perspective. Biol. Surv. Canada (Terrestrial Arthropods), Ottawa, Canada.

Dickerson, G. E. 1947. Composition of hog carcasses as influenced by heritable differences in rate and economy of gain. Res. Bull. Iowa. Agric. Exp. Stat. 354:489–524.

Donohue, K. 2009. Completing the cycle: maternal effects as the missing link in plant life histories. Philos. Trans. R. Soc. Lond. B 364:1059–1074.

Duckworth, R. A. 2009. Maternal effects and range expansion: a key factor in a dynamic process? Philos. Trans. R. Soc. Lond. B 364:1075–1086.

Fox, C. W., and T. A. Mousseau. 1998. Maternal effects as adaptations for transgenerational phenotypic plasticity in insects. Pp. 159–177 *in* T. A. Mousseau, and C. W. Fox, eds. Maternal effects as adaptations. Oxford Univ. Press, New York.

Frank, S. A. 1986. Dispersal polymorphisms in subdivided populations. J. Theor. Biol. 122:303–309.

Galloway, L. F., and J. R. Etterson. 2007. Transgenerational plasticity is adaptive in the wild. Science 318:1134–1136.

Gil, D., C. Biard, A. Lacroix, C. N. Spottiswoode, N. Saino, M. Puerta, and A. P. Moller. 2007. Evolution of yolk androgens in birds: development, coloniality, and sexual dichromatism. Am. Nat. 169:802–819.

Godfray, H. C. J. 1995. Evolutionary theory of parent-offspring conflict Nature 376:133–138.

Groothuis, T. G. G., and H. Schwabl. 2008. Review. hormone-mediated maternal effects in birds: mechanisms matter but what do we know of them? Philos. Trans. R. Soc. Lond. B 363:1647–1661.

Groothuis, T. G. G., W. Muller, N. von Engelhardt, C. Carere, and C. Eising. 2005. Maternal hormones as a tool to adjust offspring phenotype in avian species. Neurosci. Biobehav. Rev. 29:329–352.

Haig, D. 1996. Placental hormones, genomic imprinting, and maternal-fetal communication. J. Evol. Biol. 9:357–380.

Heath, D. D., and D. M. Blouw. 1998. Are maternal effects in fish adaptive or merely physiological side effects? Pp. 178–201 *in* T. A. Mousseau and C. W. Fox, eds. Maternal effects as adaptations. Oxford Univ. Press, New York, NY.

Hinde, C. A., R. A. Johnstone, and R. M. Kilner. 2010. Parent-offspring conflict and coadaptation. Science 327:1373–1376.

Huestis, D.L., and J. L. Marshall. 2006. Interaction between maternal effects and temperature affects diapause occurrence in the cricket *Allonemobius socius*. Oecologia 146:513–520.

Kilner, R. M., and C. A. Hinde. 2008. Information warfare and parent-offspring conflict. Adv. Study Behav. 38:283–336.

Kirkpatrick, M., and R. Lande. 1989. The evolution of maternal characters. Evolution 43:485–503.

Love, O. P., and T. D. Williams. 2008. The adaptive value of stress-induced phenotypes: effects of maternally derived corticosterone on sex-biased investment, cost of reproduction, and maternal fitness. Am. Nat. 172:E135–E149.

Marshall, D. J., and T. Uller. 2007. When is a maternal effect adaptive? Oikos 116:1957–1963.

Martin, T. E., and H. Schwabl. 2008. Variation in maternal effects and embryonic development rates among passerine species. Philos. Trans. R. Soc. Lond. B 363:1663–1674.

Mock, D. W., and G. A. Parker. 1997. The evolution of sibling rivalry. Univ. of Oxford Press, New York.

Motro, U. 1983. Optimal rates of dispersal. 3. Parent offspring conflict. Theor. Pop. Biol. 23:159–168.

Mousseau, T., and C. Fox. 1998. Maternal effects as adaptations. Oxford Univ. Press, New York.

Mousseau, T. A., and H. Dingle. 1991. Maternal effects in insect life histories. Annu. Rev. Entomol. 36:511–534.

Muller, W., C. M. Lessells, P. Korsten, and N. von Engelhardt. 2007. Manipulative signals in family conflict? On the function of maternal yolk hormones in birds. Am. Nat. 169:E84–E96.

Pigliucci, M. 2001. Phenotypic plasticity. Beyond nature and nurture. John Hopkins Univ. Press, Baltimore, MD.

Riska, B., J. J. Rutledge, and W. R. Atchley. 1985. Covariance between direct and maternal genetic effects in mice, with a model of persistent environmental influences. Genet. Res. 45:287–297.

Ronce, O. 2007. How does it feel to be like a rolling stone? Ten questions about dispersal evolution. Annu. Rev. Ecol. Evol. Syst. 38:231–253.

Ronce, O., J. Clobert, and M. Massot. 1998. Natal dispersal and senescence. Proc. Natl. Acad. Sci. USA 95:600–605.

Russell, A. F., and V. Lummaa. 2009. Maternal effects in cooperative breeders: from hymenopterans to humans. Philos. Trans. R. Soc. Lond. B 364:1143–1167.

Schwabl, H. 1993. Yolk is a source of maternal testosterone for developing birds. Proc. Natl. Acad. Sci. USA 90:11446–11450.

Schwabl, H., D. W. Mock, and J. A. Gieg. 1997. A hormonal mechanism for parental favouritism. Nature 386:231–231.

Simpson, S. J., and Miller, G. A. 2007. Maternal effects on phase characteristics in the desert locust, *Schistocerca gregaria*: a review of current understanding. J. Insect. Physiol. 53:869–876.

Smiseth, P. T., J. Wright, and M. Kolliker. 2008. Parent-offspring conflict and co-adaptation: behavioural ecology meets quantitative genetics. Proc. R. Soc. Lond. B 1823–1830.

Starrfelt, J., and H. Kokko. 2010. Parent-offspring conflict and the evolution of dispersal distance. Am. Nat. 175:38–49.

Taylor, P. D. 1988. An inclusive fitness model for dispersal of offspring. J. Theor Biol. 130:363–378.

Taylor, P. D., and S. A. Frank. 1996. How to make a kin selection model. J. Theor. Biol. 180:27–37.

Trivers, R. L. 1974. Parent-offspring conflict Am. Zool. 14:249–264.

Uller, T. 2008. Developmental plasticity and the evolution of parental effects. Trends Ecol. Evol. 23:432–438.

Uller, T., M. Massot, M. Richard, J. Lecomte, and J. Clobert. 2004. Long-lasting fitness consequences of prenatal sex ratio in a viviparous lizard. Evolution 58:2511–2516.

Uller, T., W. Wapstra, and A. V. Badyaev. 2009. Evolution of parental effects: conceptual issues and empirical patterns. Philos. Trans. R. Soc. Lond. B 364:1033–1177.

Willham, R. L. 1972. Role of maternal effects in animal breeding. 3. Biometrical aspects of maternal effects in animals. J. Anim. Sci. 35:1288–1293.

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Supporting Information

The following supporting information is available for this article:

Figure S1. A representative example of the evolution of patch-specific dispersal with the same settings as in (Fig. 2) except that maternal accuracy of patch assessment (e_m) was also allowed to evolve.

Figure S2. A representative example of the evolution of the five loci when (A) offspring costs (a_o) are zero and maternal costs are intermediate or high (here $a_m = 0.25$).

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

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