Maternal Effects and Parent-Offspring Conflict

Bram Kuijper[1,3] & Rufus A. Johnstone[2,4]

Abstract

Maternal effects can provide offspring with reliable information about the environment they are likely to experience, but also offer scope for maternal manipulation of young when interests diverge between parents and offspring. To predict the impact and outcome of parent-offspring conflict, we model the evolution of maternal effects on local adaptation of young. We find that parent-offspring conflict strongly influences the stability of maternal effects; moreover, the nature of the disagreement between parents and young predicts how conflict is resolved: when mothers favour less extreme mixtures of phenotypes relative to offspring (i.e., when mothers stand to gain by hedging their bets), mothers win the conflict by providing offspring with only limited amounts of information. When offspring favour overproduction of one and the same phenotype across all environments compared to mothers (e.g., when offspring favour a larger body size), neither side wins the conflict and signaling breaks down. Only when offspring favour less extreme mixtures relative to their mothers (the case we consider least likely), offspring win the conflict and obtain full information about the state of the environment. We conclude that a partial or complete breakdown of informative maternal effects will be the norm rather than the exception in the presence of parent-offspring conflict.

Keywords Nongenetic effects, maternal hormone, transgenerational effect, inheritance, information, epigenetics

Affiliations

1. Environment and Sustainability Institute (ESI), College of Life and Environmental Sciences, University of Exeter, Penryn Campus, Penryn TR10 9EZ, UK
2. Department of Zoology, University of Cambridge, Downing Street, Cambridge CB2 3EJ, UK
3. a.l.w.kuijper@exeter.ac.uk
4. raj1003@hermes.cam.ac.uk

1 Introduction

Maternal effects comprise any causal influence of the environment or phenotype of the mother on the phenotype of her offspring that is not mediated by genetic transmission (Wolf & Wade, 2009; Day & Bonduriansky, 2011; Danchin *et al.*, 2011). Such effects have been identified in many species, and may involve a wide variety of different mechanisms, ranging from hormonal influences (von Engelhardt & Groothuis, 2011), through the transmission of antibodies (e.g., Boulinier & Staszewski, 2008) and maternal provisioning of nutrients (e.g., Wells, 2010), to social learning (Mesoudi *et al.*, 2016) and even active teaching (Rapaport, 2011). It is well established that maternal effects can, at least in principle, strongly influence the course of evolution within a population (Mousseau & Fox, 1998; Räsänen & Kruuk, 2007; Badyaev & Uller, 2009; Hoyle & Ezard, 2012). More recently, there has been much discussion of when and why selection might favour the evolution of such effects themselves (Kuijper *et al.*, 2014; English *et al.*, 2015; Kuijper & Hoyle, 2015; McNamara *et al.*, 2016; Proulx & Teotónio, 2017).

Adaptive explanations of the evolution of maternal effects often suggest that they serve to provide offspring with information about the environment they are likely to encounter (Marshall & Uller, 2007; Shea *et al.*, 2011; Kuijper & Johnstone, 2013; Leimar & McNamara, 2015). This information allows the young to anticipate the challenges they will face and to develop an appropriate phenotypic response (Agrawal *et al.*, 1999; Galloway & Etterson, 2007; McGhee & Bell, 2014; Holeski *et al.*, 2012, but see Uller *et al.*, 2013). For instance, offspring field crickets (*Gryllus pennsylvanicus*) born from mothers that have been exposed to predators exhibit greater antipredator immobility (Storm & Lima, 2010). Other antipredator adaptations have been observed in *Daphnia*, where offspring from mothers that have been exposed to predatory stimuli grow larger defensive helmets (Agrawal *et al.*, 1999). Similar processes also operate in plants, for example in *Campanulastrum americanum*, where offspring phenotypes are dependent on the maternal light environment, and those that experience a light environment that matches that of their mother have a 3.4 times larger fitness in comparison to offspring that develop in different light conditions (Galloway & Etterson, 2007). These examples show that in at least some cases, maternal effects facilitate offspring anticipation of environmental challenges.

Maternal effects, however, do not always operate to the advantage of offspring. In some cases, they appear to benefit the mother at the expense of individual young (Jaenike, 1986; Einum & Fleming, 2000; Mayhew, 2001; Janz *et al.*, 2005; McCormick, 2006, reviewed in Marshall & Uller, 2007). For example, in *Cephaloleia* beetles, maternal survival is increased when ovipositing on novel plant hosts, whereas individual offspring survival was reduced compared to young on native hosts (García-Robledo & Horvitz, 2012). This raises the question whether mothers always stand to gain by supplying information beneficial to their young.

Parents and offspring often face an evolutionary conflict of interest (Trivers, 1974; Parker & Macnair, 1978; Godfray, 1995; Smiseth *et al.*, 2008; Kilner & Hinde, 2008). This conflict arises because offspring value their own survival more strongly than that of current or potential future siblings, while parents value all of their offspring equally (Trivers, 1974). Behavioural ecologists have focused mostly on conflicts over resource provisioning, in which offspring are selected to demand more resources than parents are selected to provide (Parker & Macnair, 1978; Godfray, 1995; Hinde *et al.*, 2010; Wells, 2007a,b; Kuijper & Johnstone, 2012). However, this conflict may influence information exchange as well. In particular, much attention has been devoted to parents' acquisition of information about offspring need or hunger, and to what extent they can

rely on offspring signals of condition (Godfray, 1991; Godfray & Johnstone, 2000; Royle *et al.*, 2002; Wells, 2003). Here, by contrast, we are concerned with acquisition of information about the environment by offspring from their parents, but similar issues arise within each context of information exchange (Uller & Pen, 2011). When there is parent-offspring conflict over the optimal offspring phenotype, can offspring rely on maternal signals about the environment? Alternatively, might maternal effects provide a means by which mothers can manipulate offspring phenotype and enforce their own optima on their young (Müller *et al.*, 2007; Uller, 2008; Kilner & Hinde, 2008; Tobler & Smith, 2010)?

So far, how parent-offspring conflict affects the evolution of informative maternal effects has seen surprisingly little formal investigation. A single model by Uller & Pen (2011) has considered how parent-offspring conflict over dispersal affects the degree of information contained in maternal effects. Unless offspring are somehow constrained in their response to maternal signals, they find that parent-offspring conflict typically does not affect the evolution of informative maternal effects, so that at evolutionary equilibrium, offspring are able to rely on maternal signals to implement their own optimal strategy. This contrasts markedly with other signalling models that focus on information transfer from offspring to parents, which suggest that conflict leads to the breakdown of informative signalling, unless honesty is maintained by some form of signal cost (Godfray *et al.*, 1991; Johnstone, 1999; Godfray & Johnstone, 2000). Consequently, this raises the question of whether informative signalling by mothers to offspring is indeed a general outcome of parent offspring conflict, as suggested by Uller & Pen (2011), or whether there are contexts in which conflict can lead to a breakdown of informative maternal signals instead.

To assess how parent-offspring conflict affects the evolution of maternal effects, we focus on a scenario of conflict over offspring local adaptation in a spatiotemporally varying environment (Leimar & McNamara, 2015; English *et al.*, 2015; Kuijper & Johnstone, 2016). Fluctuating environments often favour parents that produce a mixture of offspring phenotypes, containing some offspring that are adapted and some offspring that are maladapted to the current state of the local environment (Starrfelt & Kokko, 2012). Producing a mixture of offspring phenotypes ensures that at least some offspring are likely to survive, even if the local environment changes, thus preventing the extinction of the parental gene lineage (Ellner, 1986; McNamara, 1995; Leimar, 2005). In contrast to their parents, however, individual offspring have a higher genetic interest in their own survival than in that of their siblings. Consequently, offspring favour a lower probability of developing a currently maladapted phenotype than do their parents, resulting in parent-offspring conflict over local adaptation (Ellner, 1986).

We explore a situation in which offspring cannot assess the environment they will experience directly for themselves, but must rely on signals from their mother. A key ingredient of our model is that mothers can potentially 'skew' the information they provide, by signalling in a misleading way. The question we then seek to answer is whether reliable maternal signalling is stable, allowing for the persistence of maternal effects, or whether it is vulnerable to disruption by maternal dishonesty.

2 The model

We consider an 'infinite island' model (Wright, 1931; Rousset, 2004; Lehmann & Rousset, 2010) comprising a sexually hermaphroditic metapopulation that is distributed over an infinite number of patches, each of which contains n adult breeders. Generations are discrete and non-overlapping,

and in each generation, every breeder produces, as mother, a large number of offspring, each of which is sired by a random breeder. With probability ℓ, this sire is chosen from the same patch as the mother (including the possibility of self-fertilisation), while with probability $1-\ell$ the sire is chosen from a random remote patch. For the sake of tractability, we assume that the population is haploid, where gametes are produced clonally and pair to form diploid zygotes, which immediately undergo meiosis to form a new generation of haploid offspring (individual-based simulations assuming diploid inheritance and a finite number of patches give similar results, see Figures S6-S8). Upon birth, a fraction $1-d$ of newborn young remain on the natal patch, while the remaining fraction d disperse to a random patch in the metapopulation. After dispersal, offspring on a patch, both native and immigrant, compete for the n breeding vacancies created by the death of the previous generation. Those that fail to obtain a breeding vacancy die, and the life cycle then repeats. Below we provide a verbal summary of the model, while a more extensive description is given in section S2 of the Online Supplement.

Environmental variation Following previous models of maternal influences on offspring phenotype determination that do not consider parent-offspring conflict (e.g., Shea *et al.* 2011; English *et al.* 2015; Leimar & McNamara 2015; Kuijper & Johnstone 2016), we consider a spatiotemporally fluctuating environment in which each patch fluctuates between two environmental states, e_1 and e_2. In each generation, an e_i patch can change to an e_j patch with probability $\sigma_{i\to j}$ ($i \neq j$) while it remains in environmental state e_i with probability $1-\sigma_{i\to j}$. Patches fluctuate independently of one another, so that at any given time a proportion $p_1 = \sigma_{2\to 1}/(\sigma_{1\to 2}+\sigma_{2\to 1})$ of patches is in environmental state e_1, while the remainder $p_2 \equiv 1-p_1$ is in environmental state e_2.

Phenotype determination Upon birth of an offspring, it can adopt one of two phenotypes, z_1 or z_2. Individuals are 'locally adapted' and therefore experience a lower mortality rate when their phenotype z_i is identical to the environment e_i of their patch (Kawecki & Ebert, 2004). Individuals are characterised by the genetically determined strategy f_i which reflects the probability that an offspring develops phenotype z_1 as opposed to phenotype z_2. Importantly, f_i may depend upon an offspring's natal environment e_i, so we consider the evolution of a strategy $\mathbf{f} = \{f_1, f_2\}$ that specifies phenotype determination probabilities for each of the two environments. Our model also accounts for the possibility that offspring of one phenotype are potentially more costly to the mother (i.e. they require more maternal resources) than offspring of the opposite phenotype (e.g., Trivers, 1974; Ellner, 1986; Kuijper & Pen, 2014). Moreover, we allow such maternal production costs to vary dependent on the local environment e_i, so that the parameters β_i and γ_i reflect the maternal cost of producing a z_1 and z_2 offspring respectively when the local environment is in state e_i. Hence, the average investment E_i by a mother living in environment e_i per offspring is proportional to $f_i\beta_i + (1-f_i)\gamma_i$. Following classical life-history models (Smith & Fretwell, 1974; Parker & Macnair, 1978), we assume that the total number of offspring produced is inversely proportional to the average investment per offspring. Consequently, the proportions of z_1 and z_2 offspring produced by a mother living in environment e_i are then given by f_i/E_i and $(1-f_i)/E_i$ respectively. After phenotype determination, offspring either disperse or stay in the local patch, with dispersal occurring prior to environmental change. The survival probability of an offspring with phenotype z_j that ends up competing in a patch that is in environmental state e_j is given by ω_{ij}. Throughout, we assume that offspring with a phenotype that matches the local environment always survive, so that $\omega_{11} = \omega_{22} = 1$, while z_1 offspring in an e_2 environment survive with probability $\omega_{12} = 1-c_2$

and z_2 offspring in an e_1 environment survive with probability $\omega_{21} = 1-c_1$. All surviving offspring in a patch, both immigrant and philopatric, then compete for the n adult breeding positions that are locally available. The resulting fitness equations, which describe the number of successfully established offspring born from adults living in each environment are set out in section S2.1 of the Online Supplement.

2.1 Mapping the battleground

The question now arises to what extent the evolutionary interests of parents and offspring diverge when it comes to the decision of developing phenotype z_1 versus z_2. To resolve this issue, we compare the evolutionarily stable values of f_1 and f_2 under maternal and under offspring control (the divergence between these outcomes defining the 'battleground' within which parent-offspring conflict will be played out, Godfray, 1995). To determine the equilibrium probabilities of producing a z_1 phenotype under either maternal or offspring control, we adopt an adaptive dynamics approach (Geritz *et al.*, 1998; Rousset, 2004; Dercole & Rinaldi, 2008). This assumes that evolution proceeds by the successive substitution of mutations of small effect, with a clear separation of time scales between demographic and evolutionary processes (Otto & Day, 2007). We use a direct fitness (also called neighbour-modulated fitness) approach (Taylor & Frank, 1996; Taylor *et al.*, 2007) to derive the selection gradient $\mathcal{F}_i$ that determines the evolutionary change in the probability f_i of producing a z_1 offspring in environment e_i (see eq. [S5]). By numerically iterating the selection gradients until they vanish, we are able to solve numerically for the equilibrium probabilities f_1^*, f_2^* of producing a z_1 phenotype in each of the two environments.

2.2 Resolving the conflict

If the interests of mothers and offspring diverge, how then might maternal-offspring conflict be resolved? If offspring must rely on mothers for information about the state of the local environment, could this enable mothers to manipulate the behaviour of their young in each of the two contexts considered? To evaluate this possibility, we suppose that mothers can assess the state of the local environment, while offspring cannot (see also Uller & Pen, 2011). Mothers may choose to give or to withhold a signal from each of their young, with probabilities of giving the signal dependent on the state of the local environment. Offspring may then choose to develop phenotype z_1 or z_2, with probabilities dependent on whether or not they have received a signal from their mother.

The maternal signalling strategy $\mathbf{s} \equiv (s_1, s_2)$ thus specifies the probabilities of giving (rather than withholding) the signal in each type of patch, while the offspring phenotype determination strategy $\mathbf{q} = (q_S,\ q_{NS})$ specifies the probabilities of developing a z_1 phenotype when a signal is or is not received. It is the combination of these two strategies that determines the fraction of young f_i that develop as z_1 in each environment e_i:

$$f_i(s_i, q_S, q_{NS}) = s_i q_S + (1-s_i)\, q_{NS}, \tag{1}$$

so that with probability s_i a mother living in environment e_i provides her offspring with a signal, who will therefore develop as a z_1 offspring with probability q_S (and as a z_2 offspring with probability $1-q_S$). By contrast, with probability $1-s_i$, the mother withholds the signal, in which case offspring develops as a z_1 or z_2 offspring with probabilities q_{NS} and $1-q_{NS}$ respectively. Associated fitness expressions for the maternal signaling probabilities and offspring phenotype determination

strategies are given in eqns. (S17 - S20) in the Online supplement. We again assume that evolution proceeds by the successive substitution of mutations of small effect, with a clear separation of time scales between demographic and evolutionary processes (Otto & Day, 2007). This allows us to use a direct fitness approach to derive the selection gradients $\mathcal{S}_i$ and $\mathcal{Q}_j$ that determine the rates of evolutionary change in the probability s_i of providing offspring a signal in each environment and the probability $q_j \in \{q_S, q_{NS}\}$ of producing a z_1 offspring in the presence or absence of a signal (see eqns. [S21,S22]).

To solve the conflict resolution model, we seek to identify equilibrium strategy pairs for which all selection gradients (for both strategies) are simultaneously equal to zero. To do so, we choose initial conditions such that the signal is highly informative (e.g., we might choose $s_1 = 0.9$ and $s_2 = 0.1$) and offspring highly responsive (e.g., $f_S = 0.9$ and $f_{NS} = 0.1$), and iteratively update the signalling and phenotype determination probabilities by adding to each the value of the relevant selection gradient (given the current strategies), bounding the updated values between 0 and 1. This procedure is repeated until all strategies converge to stable values. The solutions obtained in this way are robust to changes in the precise starting conditions chosen, and convergence stable by construction. Note, however, that two mirror-image signalling equilibria are possible in any particular case – one in which the signal is given more often in environment e_1 and withheld more often in environment e_2, and one in which the signal is given more often in environment e_2 and withheld more often in environment e_1. These provide offspring with equal information, and thus have identical consequences in terms of the phenotype determination rates out of each patch type. For ease of interpretation, however, we consistently choose starting conditions in which the signal is given more often in environment e_1. Individual-based simulations, which assume a continuous distribution of mutations and no necessary separation of timescales, yield very similar results to the analytical model (see Figures S6-S8).

3 Results

3.1 The battleground

Figure 1 illustrates the ways in which the interests of mothers and offspring diverge. The graphs show the stable fraction of z_1 offspring produced in environment e_1 (blue) and in environment e_2 (red), under maternal control (dotted lines) versus under offspring control (solid lines), as a function of c_2, the cost of maladaptation in environment e_2 (while holding c_1, the cost of maladaptation in environment e_1, constant at 0.8). In general, both mothers and offspring favour higher proportions of z_1 offspring when the cost of maladaptation in environment e_2 is low (at the left-hand side of each graph), and lower proportions of z_1 offspring when the cost of maladaptation in environment e_2 is high (at the right-hand side of each graph). However, stable outcomes under maternal versus offspring control rarely agree precisely.

The three panels of the figure show results for three different sets of parameter values, which we have chosen to illustrate three possible kinds of 'disagreement' between mother and young (see Supplementary Figure S1 for a more extensive overview of model results).

Scenario 1 (panel A): offspring favour production of more of the locally adapted phenotype in each environment (i.e. more of phenotype z_1 in environment e_1, and more of phenotype z_2 in environment e_2); in terms of the graph, the red and blue solid lines for equilibria in the case of offspring control lie 'outside' the corresponding dotted lines for equilibria in the case of maternal control. In

this scenario, z_1 and z_2 offspring are equally costly to produce. Under these circumstances, mothers do best (in either environment) to hedge their bets by producing a certain fraction of young with a currently (locally) maladapted phenotype, to ensure survival of at least some of their brood in case the environment changes. Since offspring, by contrast, have a greater evolutionary interest in their own survival than in that of the brood as a whole, they favour a higher probability of developing the currently well-adapted phenotype.

Scenario 2 (panel B): Offspring favour production of more of phenotype z_1 across both environments; in terms of the graph, the red and blue solid lines for equilibria in the case of offspring control lie above the corresponding dotted lines for equilibria in the case of maternal control. In this case, z_2 offspring are twice as costly for mothers to produce as are z_1 offspring. Under these circumstances, mothers favour mixtures of offspring phenotypes that are more biased towards the cheaper z_1 phenotype across both environments, because producing a larger fraction of costly young reduces their overall fecundity. By comparison, offspring are less concerned with maternal fecundity relative to their own survival, and so favour mixtures of phenotypes that are more biased towards the expensive z_2 phenotype, across both environments.

Scenario 3 (panel C): Offspring favour production of more of the locally maladapted phenotype in each environment (i.e. more of phenotype z_2 in environment e_1, and more of phenotype z_1 in environment e_2); in terms of the graph, the red and blue solid lines for equilibria in the case of offspring control lie 'inside' the corresponding dotted lines for equilibria in the case of maternal control. In this case, maternal costs of producing one phenotype versus the other are assumed to depend on the local environment: specifically, we assume that a z_2 young is twice as costly to produce as a z_1 young in environment e_1, while z_1 young are twice as costly to produce than z_2 young in environment e_2. In this case, mothers favour more extreme mixtures that are biased towards the the locally-adapted phenotype that is the cheapest to produce in that particular environment, while offspring favour less extreme mixtures that feature more of the locally costly phenotype.

We have chosen parameter values to highlight the different kinds of conflict that can arise between mothers and young, because the nature of the 'disagreement' turns out to affect the resolution of the conflict, as detailed below.

3.2 Resolution of the conflict

How is parent-offspring conflict resolved when offspring control the determination of their phenotype, but must rely on maternal signals about the state of the local environment? We can categorise outcomes of the model according to the extent of information supplied by mothers to their young - offspring may obtain (i) full information about the environment (because the presence or absence of the maternal signal is perfectly correlated with the state of the environment), (ii) partial information (because the signal is given more commonly in one environment than in the other, but the correlation is imperfect) or (iii) no information (because the presence or absence of the signal is uncorrelated with the environment).

Alternatively, taking into account both the probabilities of the signal being given or withheld, and the response of offspring in each case, we can categorise outcomes according to the degree to which the realised probabilities of producing each phenotype match the values favoured by mothers versus young - (i) parents may win (i.e. the outcome matches what evolves under maternal control), (ii) neither 'side' may win (i.e. the outcome diverges from what evolves under either maternal or offspring control), or (iii) offspring may win (i.e. the outcome matches what evolves

under offspring control). Figure 2 shows the regions of parameter space in which the model predicts different levels of information transfer, while Figure 2 shows the regions in which mothers or offspring (or neither) are predicted to win (with equivalent results for additional regions of parameter space shown in Supplementary Figures S3 and S4).

As detailed below, comparison of Figures 2 and 3 with Figure 1 reveals that there is not necessarily a strict relationship between the nature of the parent/offspring battleground and the outcome (in terms of either information conveyed or who wins the battle). At the same time, however, there is a strong correlation, such that each of the three battleground scenarios we list above is typically associated with a different kind of outcome.

3.2.1 Scenario 1: when offspring favour more extreme mixtures than their mothers, mothers typically win the conflict by providing partial information

When offspring favour more extreme mixtures of phenotypes relative to their mothers (as in Figure 1A), maternal signals often evolve to be partially informative to offspring, particularly when survival costs of a maladapted offspring (c_1, c_2) are large in both environments (Figure 2A, right corner). In this case, offspring are selected to rely on maternal information, as the alternative results in substantial costs due to local maladaptation. However, by limiting the amount of information about the local environment, mothers force offspring to increase their level of bet-hedging, thus resulting in a less extreme mixture of offspring phenotypes that coincides with the maternal optimum. (see Figure 3A). An example of such a partially informative signaling strategy is given in Figures 4A,B (see Figure S6 for a corresponding individual-based simulation).

Note, however, that coevolution between maternal signals and offspring responsiveness can also lead to alternative outcomes: when the cost of maladaptation is large in one environment, but small in the other, maternal signals evolve to become uninformative (white regions in Figure 2A), as mothers favour the exclusive production of a single offspring phenotype (the one having the highest costs of maladaptation) across the two environments. Conversely, when costs of maladaptation are modest and of similar magnitude in both environments, parental and offspring optima align, leading parents to evolve signals that are fully informative to offspring (black region in Figure 2A).

3.2.2 Scenario 2: when offspring favour more of one phenotype, then typically neither side wins, and signalling often breaks down

As described above, when one phenotype is more costly to produce than the other, offspring favour mixtures in both environments that are more biased towards the more expensive phenotype (here z_2) than do mothers (see the battleground in Figure 1B). By far the commonest outcome for this type of battleground is that signaling breaks down (light grey areas in Figure 2B), thus resulting in unconditional offspring phenotype determination strategies. Who wins the conflict now starts to depend on the relative costs of maladaptation (Figure 3B): when survival costs of maladaptation are high in environment e_1, yet very low in environment e_2 (white regions in Figure 3B), both parents and offspring favour the production of a single phenotype (z_1; which matches the most severe environment) across both environments, so conflict is absent. When costs of maladaptation in environment e_2 are slightly larger, however, offspring born in environment e_2 favour the production of costly z_2 offspring, while mothers still favour the production of z_1 offspring in both environments. However, in the presence of an uninformative signal, offspring are forced to play an unconditional

strategy which results in the exclusive production of z_1 offspring in both environments, so that mothers can be said to win the conflict (light grey area in Figure 3B).
For even higher costs of maladaptation in environment e_2 in Figure 3B, mothers again favour the exclusive production of z_1 offspring across both environments (see Figure 4C, D for a detailed example). However, offspring now favour the production of a mixture of both z_1 and z_2 offspring in the absence of any maternal information, so that the resolution is one in which neither parent nor offspring wins the conflict (black region in Figure 3B). Finally, when costs of maladaptation are high in environment e_2, but not in environment e_1 (right part in Figure 2B), mothers too now start to favour the production of some costly z_2 offspring in environment e_2 (see Figure 4E, F for a detailed example). However, as offspring favour a much larger proportion of z_2 offspring in environment e_2 than mothers do, mothers only provide a partially informative signal to offspring (dark grey area in Figure 2B). The resulting uncertainty leads to less extreme proportion of z_2 offspring in environment e_2, but also leads to the production of some z_2 offspring in environment e_1. Consequently, again neither parents or offspring can be said to win the conflict (see Figure 4E).

3.2.3 Result 3: when offspring favour less extreme mixtures than their mothers, offspring typically win the conflict, with mothers providing full information

When phenotype z_2 is more costly to produce in environment e_1, while phenotype z_1 is more costly to produce in environment e_2, mothers favour mixtures that are more extreme than offspring do (see the corresponding battleground in Figure 1C). Regarding the resolution of the conflict, Figure 2C shows that maternal signals either evolve to be fully informative, or that offspring evolve to be unresponsive to maternal signals (barring narrow regions in which signals are partially informative). In addition, Figure 3C shows that, for this configuration of maternal production costs, there is a substantial region where conflict is absent. However, when conflict occurs, offspring win the conflict as a result of these fully informative signals.

A more detailed example is shown in Figures 4G, H: to avoid the production of offspring that are more costly in terms of maternal resources, mothers favour extreme mixtures consisting only of z_1 offspring in environment e_1 and only of z_2 offspring in environment e_2 (blue dotted line in Figure 4C). Offspring, however, favour a less extreme mixture of phenotypes (red solid line). Mothers are then selected to provide offspring with the maximum amount of information, as this yields mixtures of phenotypes that are closest to what is favoured by the mother. By contrast, would mothers reduce the information content of the maternal signal, they would only select offspring to produce even less extreme mixtures that are even further away from the maternal optima. Hence, provided with complete environmental information, offspring can attain their respective optima in each environment (black dotted and red lines overlap in Figure 4C).

4 Discussion

While our model is general in formulation, it is applicable to many concrete types of maternal effect, whenever mothers can influence the cues available to offspring in a way that potentially reflects the local environment. A biological example of such a mechanism is the provision of different concentrations of a maternal hormone or small RNAs to young in different environments (Groothuis & Schwabl, 2008; Meylan *et al.*, 2012; Liebers *et al.*, 2014). In this kind of situation, mothers can provide offspring with an informative signal by varying hormone or small RNA concentrations markedly across environments, or by contrast, withold information by providing more similar concentrations of the same hormone across environments. The same reasoning applies to heritable epimutations (Heard & Martienssen, 2014), where strong vs weak differences in DNA methylation of gametes between environments reflect a strongly vs weakly informative maternal signal.

Our main conclusion is that parent-offspring conflict can have a significant impact on the evolution of informative maternal effects, even when offspring are unconstrained in their responses. The key feature of our model that leads to this outcome is that parents are allowed to adopt an imperfectly informative signalling strategy, and to 'skew' offspring responses towards their preferred outcome by independently adjusting the probabilities with which they give or withold signals in each environment. When mothers can potentially manipulate offspring in this way, we find that parent-offspring conflict often leads to a partial or even a complete breakdown in information transfer at equilibrium (just as it can do in models of signalling of need by offspring to their parents, Johnstone & Godfray, 2002). Consider, for instance, the case in which parents favour a higher proportion of a cheaper phenotype among their young, compared to that favoured by their offspring, and in which they do so regardless of the local environment. Under these conditions, it is hard for informative maternal signals to persist. If offspring take advantage of this information by responding to such a signal, an individual mother can always 'push' her young closer towards her own optimum by misrepresenting the state of the environment, and signalling in a way typical of local conditions that elicit a higher proportion of the cheaper phenotype. Consequently, we conclude that parent-offspring conflict may provide a powerful explanation for the apparent weakness of transgenerational plasticity in nature (for reviews see Uller *et al.*, 2013; Heard & Martienssen, 2014)

The possibility in our model for parents to independently adjust the probabilities with which they give or withhold signals in each environment explains the contrast between our results and those of Uller & Pen (2011). In their pioneering study of the impact of parent-offspring conflict on the evolution of maternal signals, Uller & Pen (2011) found that offspring typically evolve to be highly sensitive to maternal information about the state of the environment, regardless of any discrepancy between maternal and offspring optima. Their main model, however, assumes that offspring are provided with a highly discrete signal m_i that is tied to a particular patch type e_i. Consequently, even the slightest divergence between maternal signals in each environment (i.e., $m_i \neq m_j$) provides offspring with perfect information about the environment. In other words, mothers are only able to withhold information to offspring when they are able to hold the signal m_i exactly equal to m_j, which would require considerable canalization in the face of mutation and drift in either signal.

In a supplementary model, Uller & Pen (2011) also briefly analyzed the evolution of maternal assessment errors, where making an error in environment e_i implies that offspring are provided

with signal m_j rather than signal m_i. However, they found that these maternal errors do not evolve. A key assumption of this extended model is, however, that the error is constrained to be identical across both environments: hence, a nonzero error only evolves when the advantage of providing offspring with a wrong signal in environment e_i outweighs the disadvantage of providing offspring with a wrong signal in environment e_j too. As a consequence, there is no scope for parents to independently adjust the probability of a signal being given in each environment, and so no possibility for parents to skew offspring responses in their own favour by misrepresenting the environment in a biased manner. Overall, this raises the question which mechanism is more realistic: are maternal effects indeed constrained as in the model of Uller & Pen (2011), or is there sufficient flexibility as required by the current model? Because maternal effects like hormones are often highly flexible (e.g., Müller *et al.*, 2004; Krist & Munclinger, 2015) and characterized by continuous (rather than discrete) variation across environments (e.g., Pavitt *et al.*, 2014; Lessells *et al.*, 2016), we suggest that the scope for maternal manipulation, as described by the current model, is likely to be substantial. Perhaps one way through which both the model of Uller & Pen (2011) and the current one can be reconciled, however, is when offspring are able to enforce honesty in maternal signals, so that maternal manipulation is not possible. It is therefore important that future models assess the evolutionary potential for enforcing honesty in maternal signals, whether this indeed leads to offspring winning the conflict, and how transgenerational plasticity is affected by honest signals. Yet, we emphasize that the potential for manipulation cannot be ruled out a priori for any signaling system (Dawkins & Krebs, 1978; Laidre & Johnstone, 2013), and maternal signals are no exception to this.

Another key conclusion of our model is that a reduction in maternal information transfer does not necessarily imply that either mothers (or offspring) win the conflict. Rather, the outcome of the conflict typically depends on the nature of the disagreement between mothers and young (see Figure 1), which depends on the specific trait that is studied. We suggest that scenarios in which mothers favour a more even mixture of phenotypes than do offspring (see Figure 1A) are more likely to result in partially informative signals and mothers winning the conflict (see Figure 3A). This type of outcome is particularly likely when alternative offspring phenotypes impose roughly similar costs on their mothers. One possible example is when individuals bet-hedge defences against multiple stressors, as they do when resistance to one strain of parasite trades off against resistance to another strain (strain-specific immunity: Little *et al.*, 2003; Schmid-Hempel, 2005. While resistance in such contexts is often studied in the context of heterozygosity (e.g., Penn *et al.*, 2002), an accumulating number of studies have shown that parasite resistance is, in part, influenced by transgenerational effects (Little *et al.*, 2003; Boulinier & Staszewski, 2008; Rechavi, 2014; Pigeault *et al.*, 2016). Our model predicts that parents would be selectively favored to produce more even mixtures of offspring resistant to one parasite strain versus another, while offspring themselves favour resistance against the parasite that is commonest in current local environment. In contexts like these, we would expect that mothers only provide their offspring with limited amounts of information about local parasite prevalence (leading to limited amounts of transgenerational plasticity - Uller, 2008; Holeski *et al.*, 2012), resulting in mothers winning the conflict.

For those traits for which offspring always favour overproduction of the costliest phenotype relative to mothers (see Figure 1B), it is more difficult to predict who wins the conflict: dependent on the parameters involved, either the offspring, the mother, or neither wins the conflict (Figure 3B). More important, however, is our finding that maternal information transfer can completely break down in this scenario, resulting in an absence of transgenerational plasticity (Figure 2B),

which is particularly likely to occur when costs of maladaptation are modest. We believe that the battleground depicted in Figure 1B applies to numerous traits that have been previously studied in the context of parent-offspring conflict. For example, when the trait in question is offspring size (Smith & Fretwell, 1974), offspring will always favour a larger size than mothers themselves (Parker & Macnair, 1978; Einum & Fleming, 2000; Parker *et al.*, 2002; Kuijper & Johnstone, 2012). Similarly, when the trait in question is germination or diapause, offspring favour earlier germination than do their mothers because this enhances their probability of survival, while mothers favour later germination because this reduces competition with siblings (Ellner, 1986). Finally, in the context of sex allocation, mothers favour overproduction of the cheaper sex (Trivers, 1974; Kuijper & Pen, 2014), or the sex that is least affected by local competition (Werren *et al.*, 2002; Pen, 2006; Wild & West, 2009).

For those traits for which offspring favour less extreme mixtures relative to their mothers (see Figure 1C), we predict that it is nearly always offspring who win the conflict, unless costs of maladaptation are very high (Figure 3C). More importantly, we predict that maternal signals are often fully informative in such scenarios. However, we struggle to think of specific traits that are likely to fit these assumptions.

Summing up, we make two main, testable predictions. First, since parent-offspring conflict will often partially or completely destabilise maternal signalling, we predict that informative maternal effects are more likely to evolve, and to exert stronger effects, where conflict between parent and offspring is less pronounced. In other words, informative maternal effects should be strongest in contexts of female monogamy or when females reproduce asexually. Such a prediction could, for example be tested among closely related species with different mating systems, as is the case for the nematode genus *Caenorhabditis* (Fierst *et al.*, 2015; Teotónio *et al.*, 2017). Second, given that the impact of parent-offspring conflict depends upon the nature of the disagreement between parent and offspring, we predict that at least partially informative maternal effects are most likely to evolve or persist (even in the face of parent-offspring conflict) when different phenotypes impose similar costs to mothers (e.g., bet-hedging against different strains of parasites), we would expect partially informative signals to evolve. By contrast, breakdown of maternal signalling is a more likely outcome for traits in which one offspring phenotype is more costly to mothers than other offspring phenotypes (e.g., dispersal, sex allocation, germination), particularly when the costs of local maladaptation are modest.

Acknowledgements BK has been funded by an EPSRC 2020 Science fellowship (grant number EP/I017909/1) and a Leverhulme Trust Early Career Research Fellowship (ECF 2015-273). RAJ was funded by an EPSRC sandpit grant on transgenerational effects, grant number EP/H031928/1. This work has made use of the Carson computing cluster at the Environment and Sustainability Institute at the University of Exeter. In addition, the authors acknowledge the use of the UCL Legion High Performance Computing Facility (Legion@UCL) and associated support services in the completion of this work. The Dutch Academy of Arts and Sciences (KNAW) and the Lorentz Centre at the University of Leiden, the Netherlands funded a workshop on nongenetic effects that contributed to this article.

References

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

Badyaev, A. V. & Uller, T., 2009. Parental effects in ecology and evolution: mechanisms, processes and implications. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **364**: 1169–1177. doi:10.1098/rstb.2008.0302.

Boulinier, T. & Staszewski, V., 2008. Maternal transfer of antibodies: raising immuno-ecology issues. *Trends Ecol. Evol.* **23**: 282–288. doi:10.1016/j.tree.2007.12.006.

Danchin, É., Charmantier, A., Champagne, F. A., Mesoudi, A., Pujol, B. & Blanchet, S., 2011. Beyond DNA: integrating inclusive inheritance into an extended theory of evolution. *Nat. Rev. Genet.* **12**: 475–486. doi:10.1038/nrg3028.

Dawkins, R. & Krebs, J. R., 1978. Animal signals: information or manipulation. In: *Behavioural Ecology: an Evolutionary Approach*, (eds.) J. R. Krebs & N. B. Davies. Oxford: Blackwell Scientific, pages 282–309.

Day, T. & Bonduriansky, R., 2011. A unified approach to the evolutionary consequences of genetic and nongenetic inheritance. *Am. Nat.* **178**: E18–E36. doi:10.1086/660911.

Dercole, F. & Rinaldi, S., 2008. *Analysis of Evolutionary Processes: the Adaptive Dynamics Approach and its Applications*. Princeton: Princeton University Press.

Dieckmann, U. & Law, R., 1996. The dynamical theory of coevolution: a derivation from stochastic ecological processes. *J. Math. Biol.* **34**: 579–612. doi:10.1007/BF02409751.

Einum, S. & Fleming, I. A., 2000. Highly fecund mothers sacrifice offspring survival to maximize fitness. *Nature* **405**: 565–567. doi:10.1038/35014600.

Ellner, S., 1986. Germination dimorphisms and parent-offspring conflict in seed germination. *J. Theor. Biol.* **123**: 173–185. doi:10.1016/S0022-5193(86)80151-5.

English, S., Pen, I., Shea, N. & Uller, T., 2015. The information value of non-genetic inheritance in plants and animals. *PLoS One* **10**: e0116996. doi:10.1371/journal.pone.0116996.

Fierst, J. L., Willis, J. H., Thomas, C. G., Wang, W., Reynolds, R. M., Ahearne, T. E. *et al.*, 2015. Reproductive mode and the evolution of genome size and structure in *Caenorhabditis* nematodes. *PLoS Genet.* **11**: e1005323. doi:10.1371/journal.pgen.1005323.

Galloway, L. F. & Etterson, J. R., 2007. Transgenerational plasticity is adaptive in the wild. *Science* **318**: 1134–1136. doi:10.1126/science.1148766.

García-Robledo, C. & Horvitz, C. C., 2012. Parent-offspring conflicts, 'optimal bad motherhood' and the 'mother knows best' principles in insect herbivores colonizing novel host plants. *Ecol. Evol.* **2**: 1446–1457. doi:10.1002/ece3.267.

Geritz, S., Kisdi, É., Meszéna, G. & Metz, J., 1998. Evolutionarily singular strategies and the adaptive growth and branching of the evolutionary tree. *Evol. Ecol.* **12**: 35–57. doi:10.1023/A:1006554906681.

Godfray, H. C. J., 1991. Signalling of need by offspring to their parents. *Nature* **352**: 328–330. doi:10.1038/352328a0.

Godfray, H. C. J., 1995. Evolutionary theory of parent-offspring conflict. *Nature* **376**: 133–138. doi:10.1038/376133a0.

Godfray, H. C. J. & Johnstone, R. A., 2000. Begging and bleating: the evolution of parent-offspring signalling. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **355**: 1581–1591. doi:10.1098/rstb.2000.0719.

Godfray, H. C. J., Parker, G. A. & Haig, D., 1991. Clutch size, fecundity and parent-offspring conflict [and discussion]. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **332**: 67–79. doi:10.1098/rstb.1991.0034.

Groothuis, T. G. & Schwabl, H., 2008. Hormone–mediated maternal effects in birds: mechanisms matter but what do we know of them? *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **363**: 1647–1661. doi:10.1098/rstb.2007.0007.

Heard, E. & Martienssen, R. A., 2014. Transgenerational epigenetic inheritance: myths and mechanisms. *Cell* **157**: 95–109. doi:10.1016/j.cell.2014.02.045.

Hinde, C. A., Johnstone, R. A. & Kilner, R. M., 2010. Parent-offspring conflict and coadaptation. *Science* **327**: 1373–1376. doi:10.1126/science.1186056.

Holeski, L. M., Jander, G. & Agrawal, A. A., 2012. Transgenerational defense induction and epigenetic inheritance in plants. *Trends Ecol. Evol.* **27**: 618–626. doi:10.1016/j.tree.2012.07.011.

Hoyle, R. B. & Ezard, T. H. G., 2012. The benefits of maternal effects in novel and in stable environments. *J. Roy. Soc. Interface* **9**: 2403–2413. doi:10.1098/rsif.2012.0183.

Jaenike, J., 1986. Feeding behavior and future fecundity in *Drosophila*. *Am. Nat.* **127**: 118–123. doi:10.1086/284473.

Janz, N., Bergström, A. & Sjögren, A., 2005. The role of nectar sources for oviposition decisions of the common blue butterfly *Polyommatus icarus*. *Oikos* **109**: 535–538. doi:10.1111/j.0030-1299.2005.13817.x.

Johnstone, R. A., 1999. Signaling of need, sibling competition, and the cost of honesty. *Proc. Natl. Acad. Sci. USA.* **96**: 12644–12649. doi:10.1073/pnas.96.22.12644.

Johnstone, R. A. & Godfray, H. C. J., 2002. Models of begging as a signal of need. In: *The Evolution of Begging, Competition, Cooperation and Communication*, (eds.) J. Wright & M. L. Leonard. Dordrecht: Kluwer, pages 1–20. doi:10.1007/0-306-47660-6_1.

Kawecki, T. J. & Ebert, D., 2004. Conceptual issues in local adaptation. *Ecol. Lett.* **7**: 1225–1241. doi:10.1111/j.1461-0248.2004.00684.x.

Kilner, R. M. & Hinde, C. A., 2008. Information warfare and parent-offspring conflict. *Adv. Stud. Behav.* **38**: 283–336. doi:10.1016/S0065-3454(08)00006-5.

Kimura, M. & Crow, J. F., 1964. The number of alleles that can be maintained in a finite population. *Genetics* **49**: 725–738.

Krist, M. & Munclinger, P., 2015. Context dependence of maternal effects: testing assumptions of optimal egg size, differential, and sex allocation models. *Ecology* **96**: 2726–2736. doi:10.1890/14-2450.1.

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

Kuijper, B. & Johnstone, R. A., 2012. How dispersal influences parent-offspring conflict over investment. *Behav. Ecol.* **23**: 898–906. doi:10.1093/beheco/ars054.

Kuijper, B. & Johnstone, R. A., 2013. How should mothers adjust the size of their offspring to local environmental cues? *J. Evol. Biol.* **26**: 1488–1498. doi:10.1111/jeb.12156.

Kuijper, B. & Johnstone, R. A., 2016. Parental effects and the evolution of phenotypic memory. *J. Evol. Biol.* **29**: 265–276. doi:10.1111/jeb.12778.

Kuijper, B., Johnstone, R. A. & Townley, S., 2014. The evolution of multivariate maternal effects. *PLoS Comput. Biol.* **10**: e1003550. doi:10.1371/journal.pcbi.1003550.

Kuijper, B. & Pen, I., 2014. Conflict over condition-dependent sex allocation can lead to mixed sex-determination systems. *Evolution* **68**: 3229–3247. doi:10.1111/evo.12513.

Laidre, M. E. & Johnstone, R. A., 2013. Animal signals. *Curr. Biol.* **23**: R829–R833. doi:10.1016/j.cub.2013.07.070.

Lehmann, L. & Rousset, F., 2010. How life history and demography promote or inhibit the evolution of helping behaviours. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **365**: 2599–2617. doi:10.1098/rstb.2010.0138.

Leimar, O., 2005. The evolution of phenotypic polymorphism: randomized strategies versus evolutionary branching. *Am. Nat.* **165**: 669–681. doi:10.1086/429566.

Leimar, O. & McNamara, J. M., 2015. The evolution of transgenerational integration of information in heterogeneous environments. *Am. Nat.* **185**: E55–E69. doi:10.1086/679575.

Lessells, C. M., Ruuskanen, S. & Schwabl, H., 2016. Yolk steroids in great tit *Parus major* eggs: variation and covariation between hormones and with environmental and parental factors. *Behav. Ecol. Sociobiol.* : 1–14doi:10.1007/s00265-016-2107-1.

Liebers, R., Rassoulzadegan, M. & Lyko, F., 2014. Epigenetic regulation by heritable RNA. *PLoS Genet.* **10**: e1004296. doi:10.1371/journal.pgen.1004296.

Little, T. J., O'Connor, B., Colegrave, N., Watt, K. & Read, A. F., 2003. Maternal transfer of strain-specific immunity in an invertebrate. *Curr. Biol.* **13**: 489–492. doi:10.1016/S0960-9822(03)00163-5.

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

Mayhew, P. J., 2001. Herbivore host choice and optimal bad motherhood. *Trends Ecol. Evol.* **16**: 165–167. doi:10.1016/S0169-5347(00)02099-1.

McCormick, M. I., 2006. Mothers matter: crowding leads to stressed mothers and smaller offspring in marine fish. *Ecology* **87**: 1104–1109. doi:10.1890/0012-9658(2006)87[1104:mmclts]2.0.co;2.

McGhee, K. E. & Bell, A. M., 2014. Paternal care in a fish: epigenetics and fitness enhancing effects on offspring anxiety. *Proc. R. Soc. Lond. B Biol. Sci.* **281**. doi:10.1098/rspb.2014.1146.

McGill, B. J. & Brown, J. S., 2007. Evolutionary game theory and adaptive dynamics of continuous traits. *Annu. Rev. Ecol. Evol. Syst.* **38**: 403–435. doi:10.1146/annurev.ecolsys.36.091704.175517.

McNamara, J. M., 1995. Implicit frequency dependence and kin selection in fluctuating environments. *Evol. Ecol.* **9**: 185–203. doi:10.1007/bf01237756.

McNamara, J. M., Dall, S. R. X., Hammerstein, P. & Leimar, O., 2016. Detection vs. selection: integration of genetic, epigenetic and environmental cues in fluctuating environments. *Ecol. Lett.* **19**: 1267–1276. doi:10.1111/ele.12663.

Mesoudi, A., Chang, L., Dall, S. R. & Thornton, A., 2016. The evolution of individual and cultural variation in social learning. *Trends Ecol. Evol.* **31**: 215–225. doi:10.1016/j.tree.2015.12.012.

Meylan, S., Miles, D. B. & Clobert, J., 2012. Hormonally mediated maternal effects, individual strategy and global change. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **367**: 1647–1664. doi:10.1098/rstb.2012.0020.

Mousseau, T. & Fox, C. W., 1998. *Maternal Effects as Adaptations*. Oxford: Oxford University Press.

Müller, W., Eising, C. M., Dijkstra, C. & Groothuis, T. G. G., 2004. Within-clutch patterns of yolk testosterone vary with the onset of incubation in black-headed gulls. *Behav. Ecol.* **15**: 893–397. doi:10.1093/beheco/arh091.

Müller, W., Lessells, C. M., Korsten, P. & Von Engelhardt, N., 2007. Manipulative signals in family conflict? On the function of maternal yolk hormones in birds. *Am. Nat.* **169**: E84–E96. doi:10.1086/511962.

Otto, S. P. & Day, T., 2007. *A Biologist's Guide to Mathematical Modeling in Ecology and Evolution*. Princeton: Princeton University Press.

Parker, G. A. & Macnair, M. R., 1978. Models of parent–offspring conflict. I. Monogamy. *Anim. Behav.* **26**: 97–110. doi:10.1016/0003-3472(78)90009-X.

Parker, G. A., Royle, N. J. & Hartley, I. R., 2002. Intrafamilial conflict and parental investment: a synthesis. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **357**: 295–307. doi:10.1098/rstb.2001.0950.

Pavitt, A. T., Walling, C. A., McNeilly, A. S., Pemberton, J. M. & Kruuk, L. E. B., 2014. Variation in early-life testosterone within a wild population of red deer. *Funct. Ecol.* **28**: 1224–1234. doi:10.1111/1365-2435.12260.

Pen, I., 2006. When boys want to be girls: effects of mating system and dispersal on parent-offspring sex ratio conflict. *Evol. Ecol. Res.* **8**: 103–113.

Penn, D. J., Damjanovich, K. & Potts, W. K., 2002. MHC heterozygosity confers a selective advantage against multiple-strain infections. *Proc. Natl. Acad. Sci. U.S.A.* **99**: 11260–11264. doi:10.1073/pnas.162006499.

Pigeault, R., Garnier, R., Rivero, A. & Gandon, S., 2016. Evolution of transgenerational immunity in invertebrates. *Proc. R. Soc. Lond. B Biol. Sci.* **283**: 20161136. doi:10.1098/rspb.2016.1136.

Proulx, S. R. & Teotónio, H., 2017. What kind of maternal effects can be selected for in fluctuating environments? *Am. Nat.* **189**: E118–E137. doi:10.1086/691423.

Rapaport, L. G., 2011. Progressive parenting behavior in wild golden lion tamarins. *Behav. Ecol.* **22**: 745–754. doi:10.1093/beheco/arr055.

Räsänen, K. & Kruuk, L. E. B., 2007. Maternal effects and evolution at ecological time-scales. *Funct. Ecol.* **21**: 408–421. doi:10.1111/j.1365-2435.2007.01246.x.

Rechavi, O., 2014. Guest list or black list: heritable small RNAs as immunogenic memories. *Trends Cell Biol.* **24**: 212–220. doi:10.1016/j.tcb.2013.10.003.

Rousset, F., 2004. *Genetic Structure in Subdivided Populations*. Princeton: Princeton University Press.

Royle, N. J., Hartley, I. R. & Parker, G. A., 2002. Begging for control: when are offspring solicitation behaviours honest? *Trends Ecol. Evol.* **17**: 434–440. doi:10.1016/S0169-5347(02)02565-X.

Schmid-Hempel, P., 2005. Natural insect host-parasite systems show immune priming and specificity: puzzles to be solved. *Bioessays* **27**: 1026–1034. doi:10.1002/bies.20282.

Shea, N., Pen, I. & Uller, T., 2011. Three epigenetic information channels and their different roles in evolution. *J. Evol. Biol.* **24**: 1178–1187. doi:10.1111/j.1420-9101.2011.02235.x.

Smiseth, P. T., Wright, J. & Kölliker, M., 2008. Parent–offspring conflict and co–adaptation: behavioural ecology meets quantitative genetics. *Proc. R. Soc. Lond. B Biol. Sci.* **275**: 1823–1830. doi:10.1098/rspb.2008.0199.

Smith, C. C. & Fretwell, S. D., 1974. The optimal balance between size and number of offspring. *Am. Nat.* **108**: 499–506. doi:10.1086/282929.

Starrfelt, J. & Kokko, H., 2012. Bet-hedging – a triple trade-off between means, variances and correlations. *Biol. Rev.* **87**: 742–755. doi:10.1111/j.1469-185X.2012.00225.x.

Storm, J. J. & Lima, S. L., 2010. Mothers forewarn offspring about predators: a transgenerational maternal effect on behavior. *Am. Nat.* **175**: 382–390. doi:10.1086/650443.

Taylor, P. D. & Frank, S. A., 1996. How to make a kin selection model. *J. Theor. Biol.* **180**: 27–37. doi:10.1006/jtbi.1996.0075.

Taylor, P. D., Wild, G. & Gardner, A., 2007. Direct fitness or inclusive fitness: how shall we model kin selection? *J. Evol. Biol.* **20**: 301–309. doi:10.1111/j.1420-9101.2006.01196.x.

Teotónio, H., Estes, S., Phillips, P. C. & Baer, C. F., 2017. Experimental evolution with *Caenorhabditis* nematodes. *Genetics* **206**: 691. doi:10.1534/genetics.115.186288.

Tobler, M. & Smith, H. G., 2010. Mother–offspring conflicts, hormone signaling, and asymmetric ownership of information. *Behav. Ecol.* **21**: 893–897. doi:10.1093/beheco/arq085.

Trivers, R. L., 1974. Parent-offspring conflict. *Am. Zool.* **14**: 249–264. doi:10.1093/icb/14.1.249.

Uller, T., 2008. Developmental plasticity and the evolution of parental effects. *Trends Ecol. Evol.* **23**: 432–438. doi:10.1016/j.tree.2008.04.005.

Uller, T., Nakagawa, S. & English, S., 2013. Weak evidence for anticipatory parental effects in plants and animals. *J. Evol. Biol.* **26**: 2161–2170. doi:10.1111/jeb.12212.

Uller, T. & Pen, I., 2011. A theoretical model of the evolution of maternal effects under parent-offspring conflict. *Evolution* **65**: 2075–2084. doi:10.1111/j.1558-5646.2011.01282.x.

von Engelhardt, N. & Groothuis, T. G., 2011. Maternal hormones in avian eggs. In: *Hormones and Reproduction of Vertebrates*, (eds.) D. O. Norris & K. H. Lopez. London: Academic Press, pages 91–127. doi:10.1016/B978-0-12-374929-1.10004-6.

Wells, J. C., 2007a. Flaws in the theory of predictive adaptive responses. *Trends Endocrinol. Metab.* **18**: 331–337. doi:10.1016/j.tem.2007.07.006.

Wells, J. C., 2010. Maternal capital and the metabolic ghetto: An evolutionary perspective on the transgenerational basis of health inequalities. *Am. J. Hum. Biol.* **22**: 1–17. doi:10.1002/ajhb.20994.

Wells, J. C. K., 2003. Parent-offspring conflict theory, signaling of need, and weight gain in early life. *Q. Rev. Biol.* **78**: 169–202. doi:10.1086/374952.

Wells, J. C. K., 2007b. The thrifty phenotype as an adaptive maternal effect. *Biol. Rev.* **82**: 143–172. doi:10.1111/j.1469-185X.2006.00007.x.

Werren, J. H., Hatcher, M. J. & Godfray, H. C. J., 2002. Maternal-offspring conflict leads to the evolution of dominant zygotic sex determination. *Heredity* **88**: 102–111. doi:10.1038/sj.hdy.6800015.

Wild, G. & West, S. A., 2009. Genomic imprinting and sex allocation. *Am. Nat.* **173**: E1–E14. doi:10.1086/593305.

Wolf, J. B. & Wade, M. J., 2009. What are maternal effects (and what are they not)? *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **364**: 1107–1115. doi:10.1098/rstb.2008.0238.

Wright, S., 1931. Evolution in Mendelian populations. *Genetics* **16**: 97–159.

5 Figure captions

Figure 1 Stable probabilities of producing a phenotype z_1 offspring in environments e_1 (blue lines) and e_2 (red lines) respectively, plotted against the cost of maladaptation c_2 in environment e_2. Panel A: both offspring phenotypes are equally costly to produce to mothers ($\beta_1 = \beta_2 = \gamma_1 = \gamma_2 = 1$). Consequently, mothers (dashed lines) favour more even mixtures of offspring phenotypes. By contrast, offspring favour more extreme mixtures that are biased towards the phenotype with the highest survival in the local environment (i.e., offspring favour more z_2 in environment e_2 and more z_1 in environment e_1). Panel B: when phenotype z_2 is more costly to produce in both environments ($\beta_1 = \beta_2 = 1,\ \gamma_1 = \gamma_2 = 2$), the probability of producing z_2 offspring is reduced. However, as offspring are more related to themselves than to their mothers, offspring favour a greater probability of producing more costly z_2 offspring in both environments. Panel C: Maternal production costs are environment dependent, so z_2 young are more costly (less costly) to produce than z_1 young in environment e_1 (in environment e_2); ($\beta_1 = 1, \beta_2 = 2, \gamma_1 = 2, \gamma_2 = 1$). Consequently, mothers favour more extreme mixtures of offspring phenotypes that are biased towards the phenotype that is cheaper to produce in each environment. By contrast, offspring favour a larger probability of developing as the more costly phenotype, leading them to favour more even mixtures of offspring phenotypes. Parameters: $d = 0.1$, $\ell = 0.5$, $\sigma_{12} = 0.2$, $\sigma_{21} = 0.25$, $n = 1$, $c_1 = 0.83$.

Figure 2 Phenotype determination when offspring rely on the maternal signal: the information content of the maternal signal $\mathbf{s} = (s_1, s_2)$ for the three different scenarios of conflict considered in Figure 1. Panel A: offspring phenotypes are equally costly to produce to mothers. For a wide range of costs of maladaptation, mothers evolve signals that are partially informative to offspring, although other outcomes also occur. Panel B: when the z_2 phenotype is more costly to produce for mothers in both environments, maternal signals always evolve to be uninformative when conflict occurs. Panel C: when the z_1 and z_2 phenotypes are more costly to produce in the respective environments e_2 and e_1, maternal signals typically evolve to be fully informative, apart from a narrow boundary in which signals are only partially informative. The information content H of the signal is calculated as a measure of entropy weighed by the probability of receiving and not receiving a maternal signal in both environments: $H = 1 - \frac{1}{2}(s_1 + s_2)F[s_1/(s_1 + s_2)] - [1 - \frac{1}{2}(s_1 + s_2)]F[(1 - s_1)/(1 - s_1 + 1 - s_2)]$, where $F[x] = -x\log_2(x) - (1-x)\log_2(1-x)$. See Figure S2 for the corresponding equilibrium probabilities of producing offspring with phenotype z_1 when offspring rely on a maternal signal. In addition, Figure S3 plots outcomes for an asymmetric environment where e_1 patches are more common than e_2 patches. Parameters: $d = 0.1$, $\ell = 0.5$, $\sigma_{12} = 0.15$, $\sigma_{21} = 0.15$, $n = 1$.

Figure 3 Phenotype determination when offspring rely on the maternal signal: who wins the conflict? Panel A: when offspring favour more extreme mixtures of phenotypes than mothers, mothers can be said to win the conflict by restricting the information content of the maternal signal. Panel B: when offspring favour mixtures that are more biased towards one phenotype (z_2) relative to their mothers, the conflict is either won by offspring, mothers or neither of them, dependent on the relative strength of the costs of maladaptation in each environment. Panel C: when offspring favour mixtures that are less extreme relative to what is favored by their mothers, offspring win the conflict, as a fully informative maternal signal never results in more extreme mixtures than what is favored by offspring. Parameters: $d = 0.1$, $\ell = 0.5$, $\sigma_{12} = 0.1$, $\sigma_{21} = 0.25$, $n = 1$.

Figure 4 Examples of phenotype determination strategies (top row) and resulting signalling strategies (bottom row). Panels A, B: when offspring favour more extreme mixtures of phenotypes than mothers, mothers evolve only partially informative signals (panel B). As offspring only receive a limited amount of environmental information, offspring produce less extreme phenotypic mixtures, and mothers win the conflict (panel A). Panels C, D: when offspring favour phenotypic mixtures that are more biased towards one phenotype (z_2), uninformative maternal signals commonly evolve (panel D), so that neither parents nor offspring win the conflict (panel C). Panels E, F: when offspring favour less extreme mixtures of phenotypes than mothers, mothers evolve fully informative signals (panel F). As a result, offspring obtain complete environmental information, resulting in offspring winning the conflict (panel C). Parameters: $d = 0.1$, $\ell = 0.5$, $\sigma_{12} = \sigma_{21} = 0.15$, $n = 1$. Specific parameters for the different panels: A, B: $c_1 = c_2 = 0.95$, $\beta_1 = \beta_2 = \gamma_1 = \gamma_2 = 1$; C, D: $c_1 = 0.5$, $c_2 = 0.8$, $\beta_1 = \beta_2 = 1$, $\gamma_1 = \gamma_2 = 2$; E, F: $c_1 = 0.2$, $c_2 = 0.8$, $\beta_1 = \gamma_2 = 1$, $\beta_2 = \gamma_1 = 2$. The scenario in panels C, D where offspring favour phenotypic mixtures that are more biased towards one phenotype (z_2) is further highlighted in Supplementary Figure S5.

6 Figures

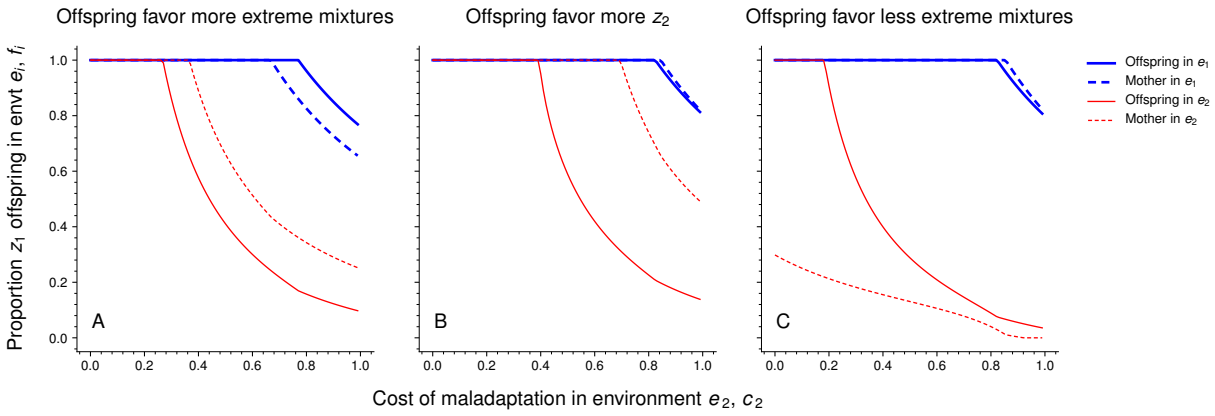
Offspring favor more extreme mixtures
Offspring favor more z_2
Offspring favor less extreme mixtures
Proportion z_i offspring in envt e_i, f_i
A
B
C
0.0
0.2
0.4
0.6
0.8
1.0
Cost of maladaptation in environment e_2, c_2
Offspring in e_1
Mother in e_1
Offspring in e_2
Mother in e_2

Figure 1:

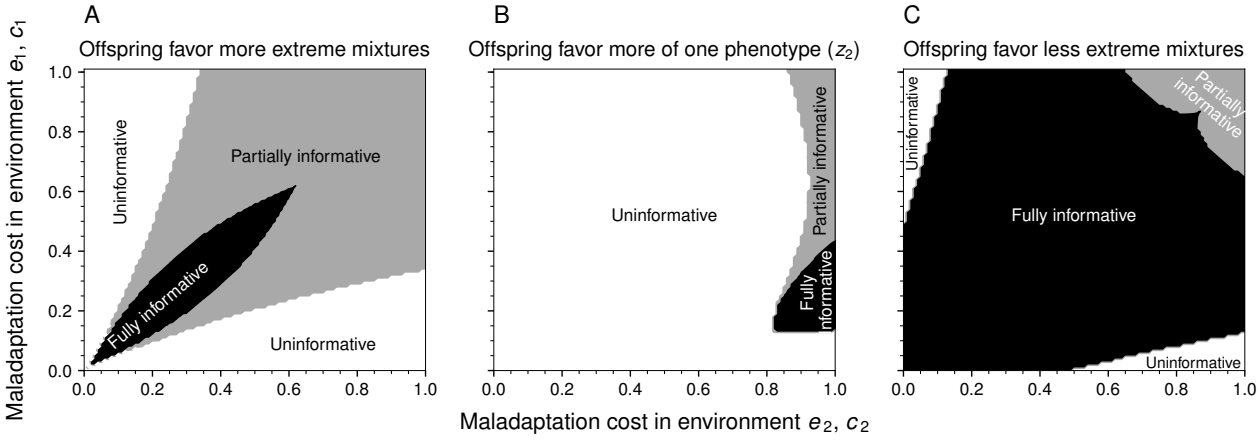
A
Offspring favor more extreme mixtures
B
Offspring favor more of one phenotype (z_2)
C
Offspring favor less extreme mixtures
Maladaptation cost in environment e_1, c_1
Maladaptation cost in environment e_2, c_2
Uninformative
Partially informative
Fully informative
Uninformative
Uninformative
Partially informative
Fully informative
Uninformative
Partially informative
Fully informative
Uninformative
0.0
0.2
0.4
0.6
0.8
1.0

Figure 2:

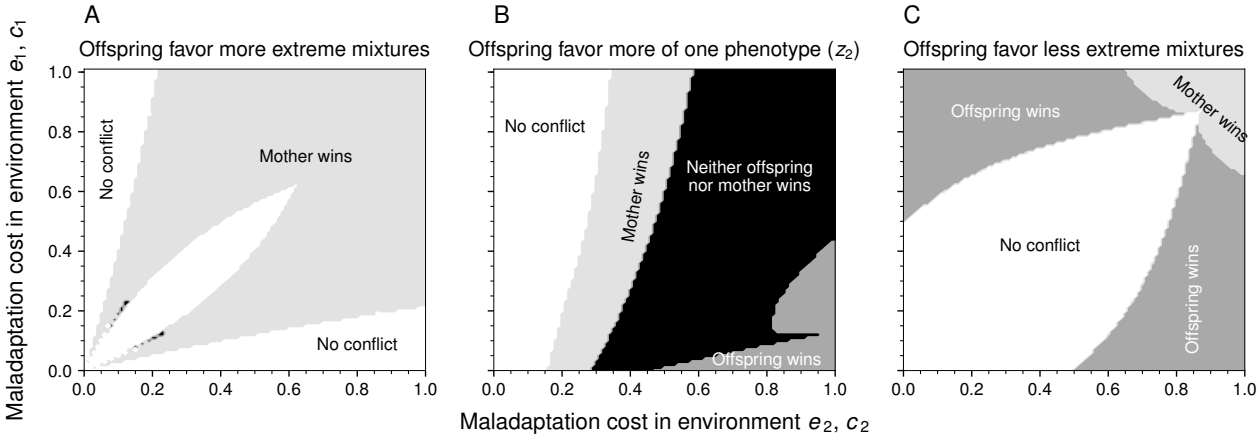
A
Offspring favor more extreme mixtures
B
Offspring favor more of one phenotype (z_2)
C
Offspring favor less extreme mixtures
Maladaptation cost in environment e_1, c_1
Maladaptation cost in environment e_2, c_2
No conflict
Mother wins
No conflict
No conflict
Mother wins
Neither offspring nor mother wins
Offspring wins
Offspring wins
Mother wins
No conflict
Offspring wins
0.0
0.2
0.4
0.6
0.8
1.0

Figure 3:

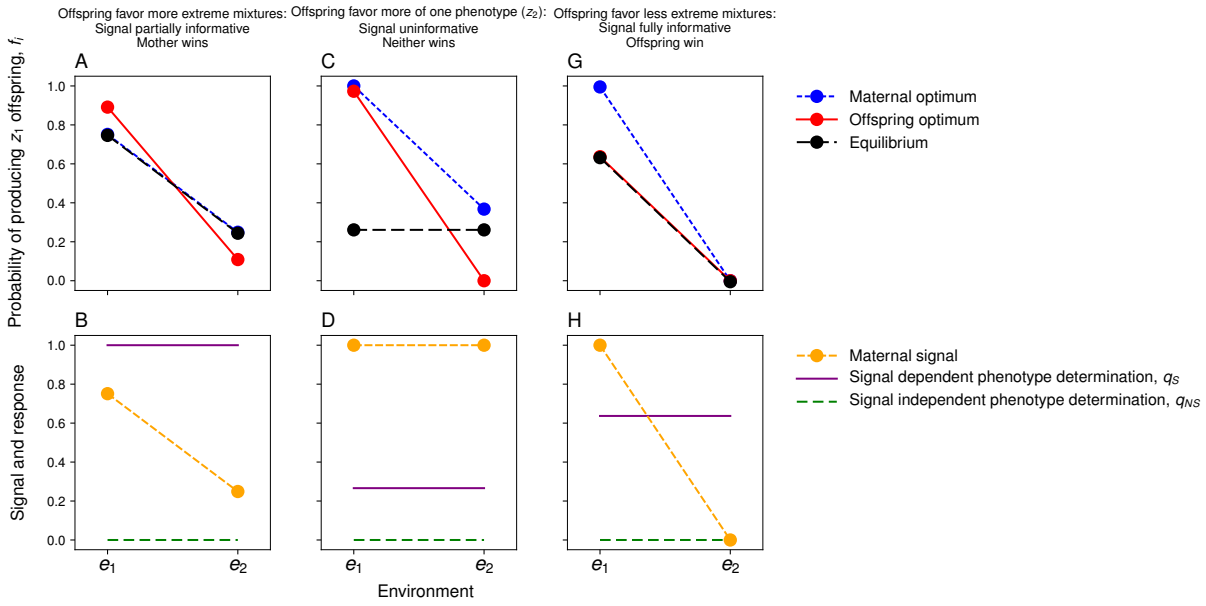
Offspring favor more extreme mixtures:
Signal partially informative
Mother wins
Offspring favor more of one phenotype (z_2):
Signal uninformative
Neither wins
Offspring favor less extreme mixtures:
Signal fully informative
Offspring win
A
C
G
B
D
H
Probability of producing z_1 offspring, f_i
Signal and response
1.0
0.8
0.6
0.4
0.2
0.0
e_1
e_2
Environment
Maternal optimum
Offspring optimum
Equilibrium
Maternal signal
Signal dependent phenotype determination, q_S
Signal independent phenotype determination, q_{NS}

Figure 4:

Maternal Effects and Parent-Offspring Conflict

Online Supplement

S1 Supplementary Figures

Figure S1 Stable probabilities of producing a phenotype z_1 offspring in environments e_1 (first column) and e_2 (second column) respectively, plotted against the cost of maladaptation in each environment for comparable parameter values as in Figure 1. Panels A, B: both offspring phenotypes are equally costly to produce to mothers ($\beta_1 = \beta_2 = \gamma_1 = \gamma_2 = 1$). Panels C, D: phenotype z_2 is more costly to produce in both environments ($\beta_1 = \beta_2 = 1,\ \gamma_1 = \gamma_2 = 2$). Panels E, F: Maternal production costs are dependent on the local environment, so z_2 young are more costly (less costly) to produce than z_1 young in environment e_1 (in environment e_2); ($\beta_1 = 1, \beta_2 = 2, \gamma_1 = 2, \gamma_2 = 1$). Consequently, mothers favour more extreme mixtures of offspring phenotypes that are biased towards the phenotype that is cheaper to produce in each environment. By contrast, offspring favour a larger probability of developing as the more costly phenotype, leading them to favour more even mixtures of offspring phenotypes. Parameters: $d = 0.1$, $\ell = 0.5$, $\sigma_{12} = \sigma_{21} = 0.25$, $n = 1$.

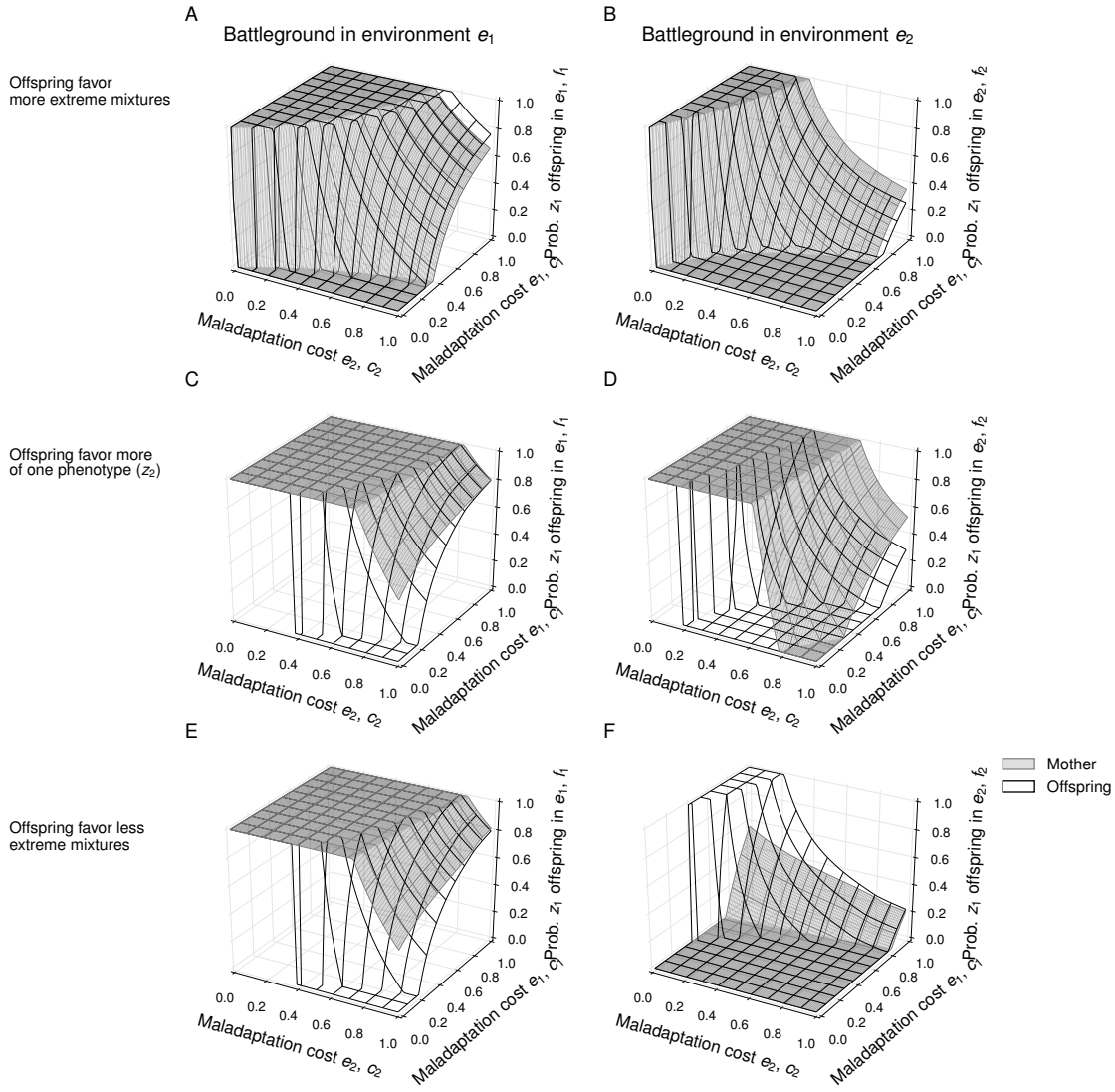
A
Battleground in environment e_1
B
Battleground in environment e_2
Offspring favor more extreme mixtures
C
D
Offspring favor more of one phenotype (z_2)
E
F
Offspring favor less extreme mixtures
Mother
Offspring
Maladaptation cost e_2, c_2
Maladaptation cost e_1, c_1
Prob. z_1 offspring in e_1, f_1
Prob. z_1 offspring in e_2, f_2
0.0
0.2
0.4
0.6
0.8
1.0

Figure S1:

Figure S2 Phenotype determination when offspring rely on the maternal signal: the equilibrium probabilities of producing offspring with phenotype z_1 in environment e_1 (panels A, C, E) and e_2 (panels B, D, F) respectively. Each row corresponds to the different scenarios of parent-offspring conflict shown in Figure 2. Panels A, B: both phenotypes are equally costly to produce to mothers ($\beta_1 = \beta_2 = \gamma_1 = \gamma_2 = 1$). Panels C, D: phenotype z_2 is twice as costly to produce in both environments than phenotype z_1 ($\beta_1 = \beta_2 = 1,\ \gamma_1 = \gamma_2 = 2$). Panels E, F: phenotype z_1 is twice as costly to produce as phenotype z_2 in environment e_2 and phenotype z_2 is twice as costly to produce as phenotype z_1 in environment e_1 ($\beta_1 = \gamma_2 = 1$, $\beta_2 = \gamma_1 = 2$). Parameters: $d = 0.1$, $\ell = 0.5$, $\sigma_{12} = \sigma_{21} = 0.25$, $n = 1$.

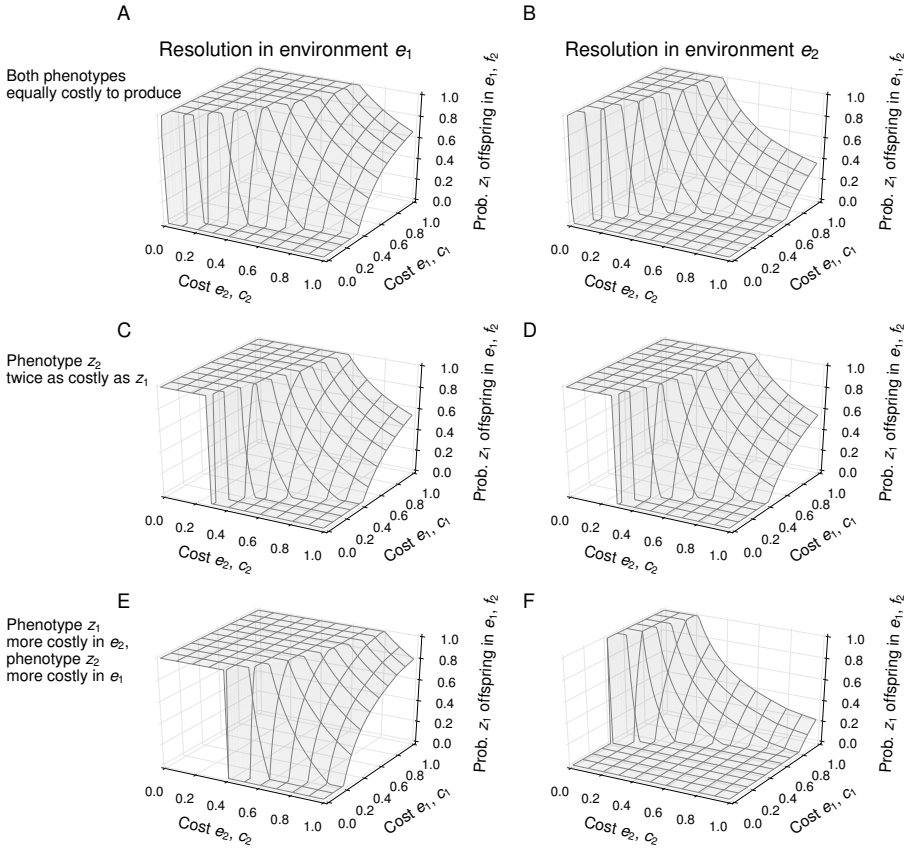
A
Resolution in environment e_1
Both phenotypes equally costly to produce
B
Resolution in environment e_2
C
Phenotype z_2 twice as costly as z_1
D
E
Phenotype z_1 more costly in e_2, phenotype z_2 more costly in e_1
F
Prob. z_1 offspring in e_1, f_2
Cost e_2, c_2
Cost e_1, c_1
0.0
0.2
0.4
0.6
0.8
1.0

Figure S2:

Figure S3 Phenotype determination when offspring rely on the maternal signal: the information content of the maternal signal $\mathbf{s} = (s_1, s_2)$ for various regimes of environmental change. Panels A-C: a very slowly changing environment where both types of environment are equally common. Note that fully informative signals can evolve regardless of the underlying battleground (see Figure 1). However, such fully informative signals are rarest when offspring favour overproduction of one phenotype (z_2) relative to their mothers (panel B). Panels D-F: a slowly fluctuating, asymmetric environment. Results are similar to panels A-C, except that fully informative signals do not evolve when offspring favour overproduction of one phenotype (z_2) relative to their mothers (panel E). Panels G-I: a randomly fluctuating environment. Fully informative strategies do not evolve, unless mothers favour more extreme mixtures than offspring (panel I). When costs of producing offspring are environment-dependent, mothers favor overproducing z_i offspring in environment e_i for the sake of fecundity, despite that offspring survival is random relative to phenotype. Offspring again favor a more even mixture, so that maternal signals again evolve to be fully informative. Panels J-L: a highly asymmetric environment, in which environment e_1 is common and environment e_2 is very rare. For a broad range of costs of maladaptation c_1 and c_2, we find that both parents and offspring agree on producing only z_1 offspring (top left corners in panels J, L). As a monomorphism is favored by both parents and offspring, environmental information via maternal signals is not selectively favored. Only when costs of maladaptation in the rarer environment e_2 are much more severe than costs in the common environment e_1 do we find that maternal signals can be partially or even fully informative (panels J, L). However, when offspring favour more of one phenotype (z_2) than their mothers (panel K), signaling still completely breaks down. This is because mothers will favour overproduction of $z_1$instead, as this phenotype matches the commoner environment e_1. Parameters: $d = 0.1$, $\ell = 0.5$, $n = 1$.

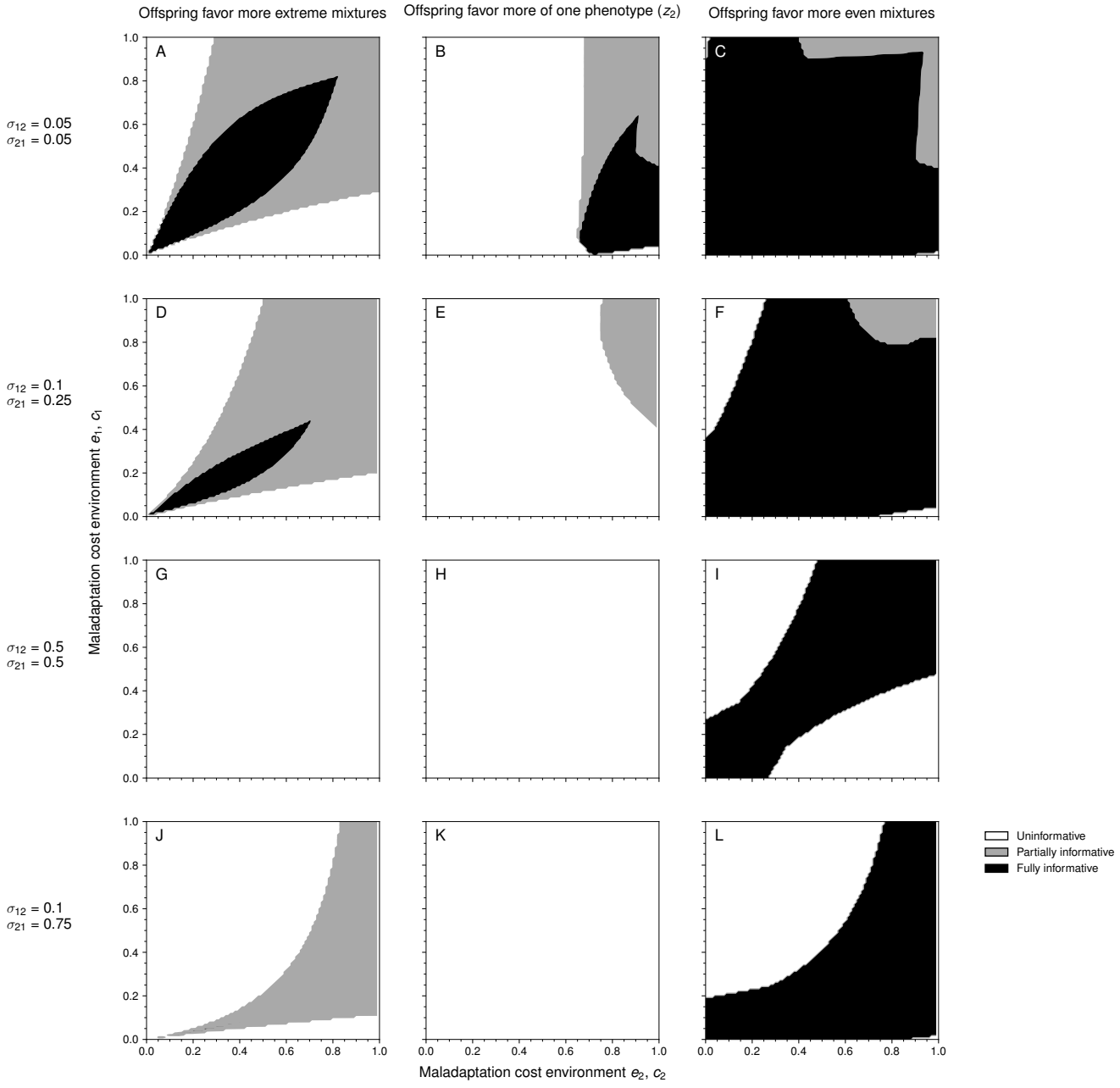
Offspring favor more extreme mixtures
Offspring favor more of one phenotype (z_2)
Offspring favor more even mixtures
σ_{12} = 0.05
σ_{21} = 0.05
σ_{12} = 0.1
σ_{21} = 0.25
σ_{12} = 0.5
σ_{21} = 0.5
σ_{12} = 0.1
σ_{21} = 0.75
A
B
C
D
E
F
G
H
I
J
K
L
Maladaptation cost environment e_1, c_1
Maladaptation cost environment e_2, c_2
0.0
0.2
0.4
0.6
0.8
1.0
Uninformative
Partially informative
Fully informative

Figure S3:

Figure S4 Phenotype determination when offspring rely on the maternal signal: who is winning the conflict? The key conclusion to take from this figure is that who wins the conflict is highly robust to the rate of environmental change and the relative frequency of environment e_1 versus e_2. Figure 3A in the main text concludes that when offspring favour more extreme mixtures, conflict is either absent or mothers win the conflict. Here we show that this holds for all environmental configurations (first column). Figure 3B concludes that it either mothers, neither or offspring win the conflict when offspring favour more of one phenotype (z_2). The second column of the current figure show that this result extends to other rates of change, unless environments change randomly (panel J), in which case offspring almost always win the conflict. Figure 3C concludes that offspring are the most likely to win the conflict when offspring favour more even mixtures relative to their parents. The third column shows that this result extends to other environmental configurations as well. Parameters: $d = 0.1$, $\ell = 0.5$, $n = 1$.

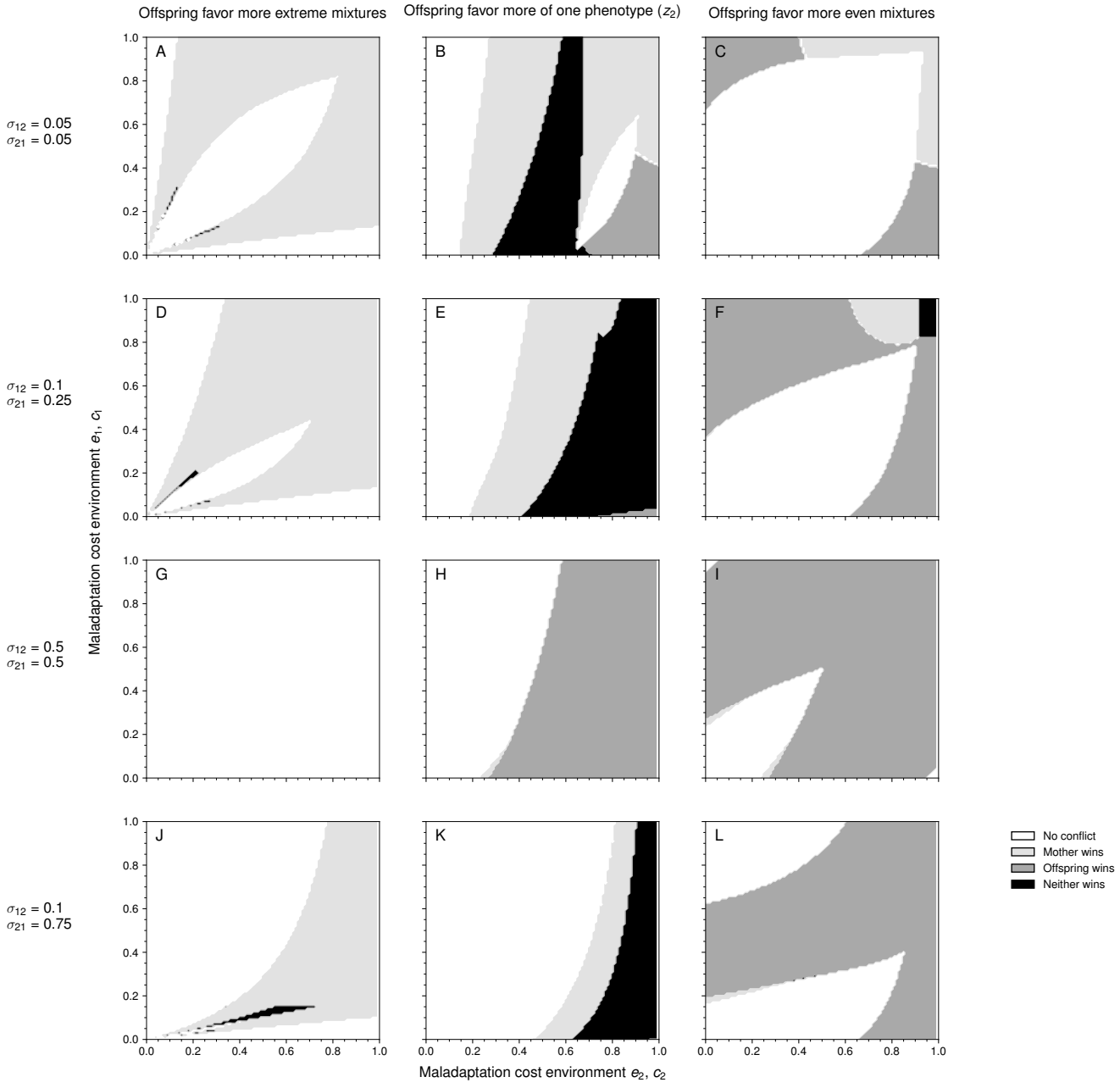
Offspring favor more extreme mixtures
Offspring favor more of one phenotype (z_2)
Offspring favor more even mixtures
σ_{12} = 0.05
σ_{21} = 0.05
σ_{12} = 0.1
σ_{21} = 0.25
σ_{12} = 0.5
σ_{21} = 0.5
σ_{12} = 0.1
σ_{21} = 0.75
A
B
C
D
E
F
G
H
I
J
K
L
Maladaptation cost environment e_1, c_1
Maladaptation cost environment e_2, c_2
0.0
0.2
0.4
0.6
0.8
1.0
No conflict
Mother wins
Offspring wins
Neither wins

Figure S4:

Figure S5 More examples of phenotype determination strategies (top row) and signalling strategies (bottom row) when offspring favour more of one phenotype (z_2) than mothers (see also Figure 4C, D) in the main text. Panels A, B: whereas maternal signals often evolve to be uninformative when offspring favour more of z_2 than their mothers (see Figure 3B), mothers are selected to provide offspring with information when the cost of maladaptation in environment e_2 becomes very high. By providing offspring with a signal, mothers prevent offspring from evolving an unconditional strategy that would result in the production of a high number of resource-costly z_2 offspring everywhere. However, mothers only provide offspring with a partially informative signal. In response to this uncertainty about the local environment, offspring are selected to produce less extreme mixtures of offspring, which brings the overall proportion of resource-costly z_2 offspring that are produced in environment e_2 closer to the maternal optimum. At the same time however, the proportion of z_2 offspring produced in environment e_1 lies further away from the maternal optimum. However, the absolute number of z_2 offspring produced in e_1 is still much lower than that in e_2, thus resulting in a lower overall resource cost to mothers. Panel C, D: when costs of maladaptation in environment e_2 are very high, yet low in environment e_2, mothers are selected to provide offspring with complete information about the environment. By contrast, a hypothetical reduction in the information content of the signal would favour offspring to produce a mixture that is heavily biased towards z_2 offspring, coming at a substantial cost to the mother. Only by providing offspring with a fully informative signal, can mothers prevent the overproduction of resource-costly z_2 offspring in both environments. As a consequence, however, offspring can be said to win the conflict. Parameters: $d = 0.1$, $\ell = 0.5$, $\sigma_{12} = \sigma_{21} = 0.15$, $n = 1$, $\beta_1 = \beta_2 = 1$, $\gamma_1 = \gamma_2 = 2$. Panels A, B: $c_1 = 0.5$, $c_2 = 0.95$. Panels C, D: $c_1 = 0.2$, $c_2 = 0.95$.

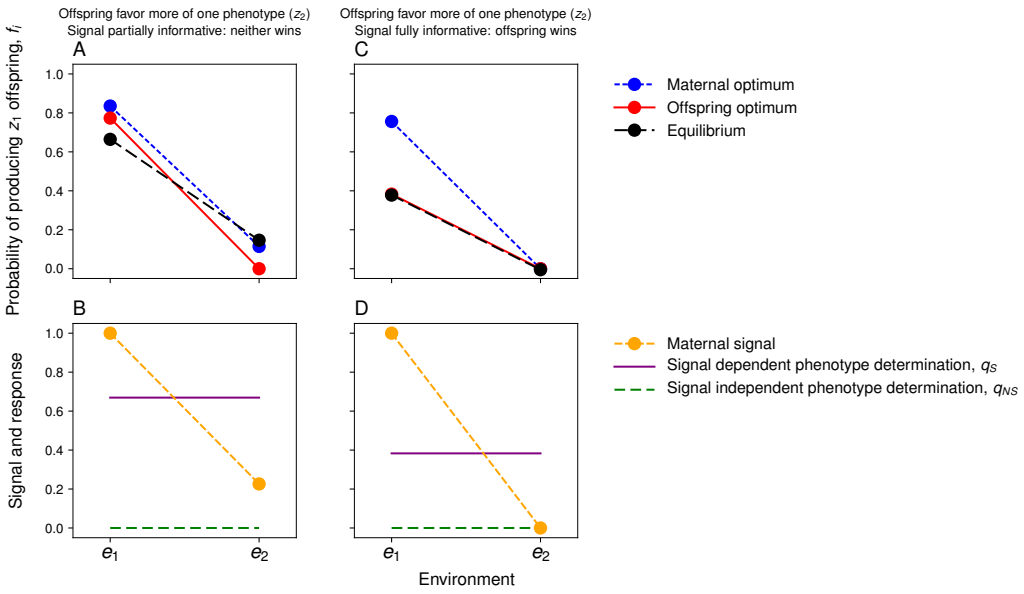
Offspring favor more of one phenotype (z_2)
Signal partially informative: neither wins
Offspring favor more of one phenotype (z_2)
Signal fully informative: offspring wins
A
C
B
D
Probability of producing z_1 offspring, f_i
Signal and response
1.0
0.8
0.6
0.4
0.2
0.0
e_1
e_2
Environment
Maternal optimum
Offspring optimum
Equilibrium
Maternal signal
Signal dependent phenotype determination, q_S
Signal independent phenotype determination, q_{NS}

Figure S5:

Figure S6 The evolution towards a partially informative equilibrium where phenotype determination diverges across the two environments: individual-based simulations. Both phenotypes are equally costly to produce for mothers: $\beta_1 = \beta_2 = 1$, $\gamma_1 = \gamma_2 = 1$. Dashed lines reflect the candidate ESSs found by the analytical model. Panels A, B: the evolution of the maternal signals s_1 and s_2 over time. Panels C, D: the evolution of the offspring phenotype determination loci q_S and q_{NS} over evolutionary time. Panels E, F: the evolution of the resulting phenotype determination probabilities over time. For each generation, a histogram (containing 200 bins) of phenotypic values is plotted, where values are quartic root transformed to aid visibility. Parameters: $c_1 = c_2 = 0.83$, $\sigma_{12} = \sigma_{21} = 0.25$, $n = 1$, $\ell = 0.5$, $d = 0.1$, $\mu = 0.01$, $\sigma_\mu^2 = 0.0001$.

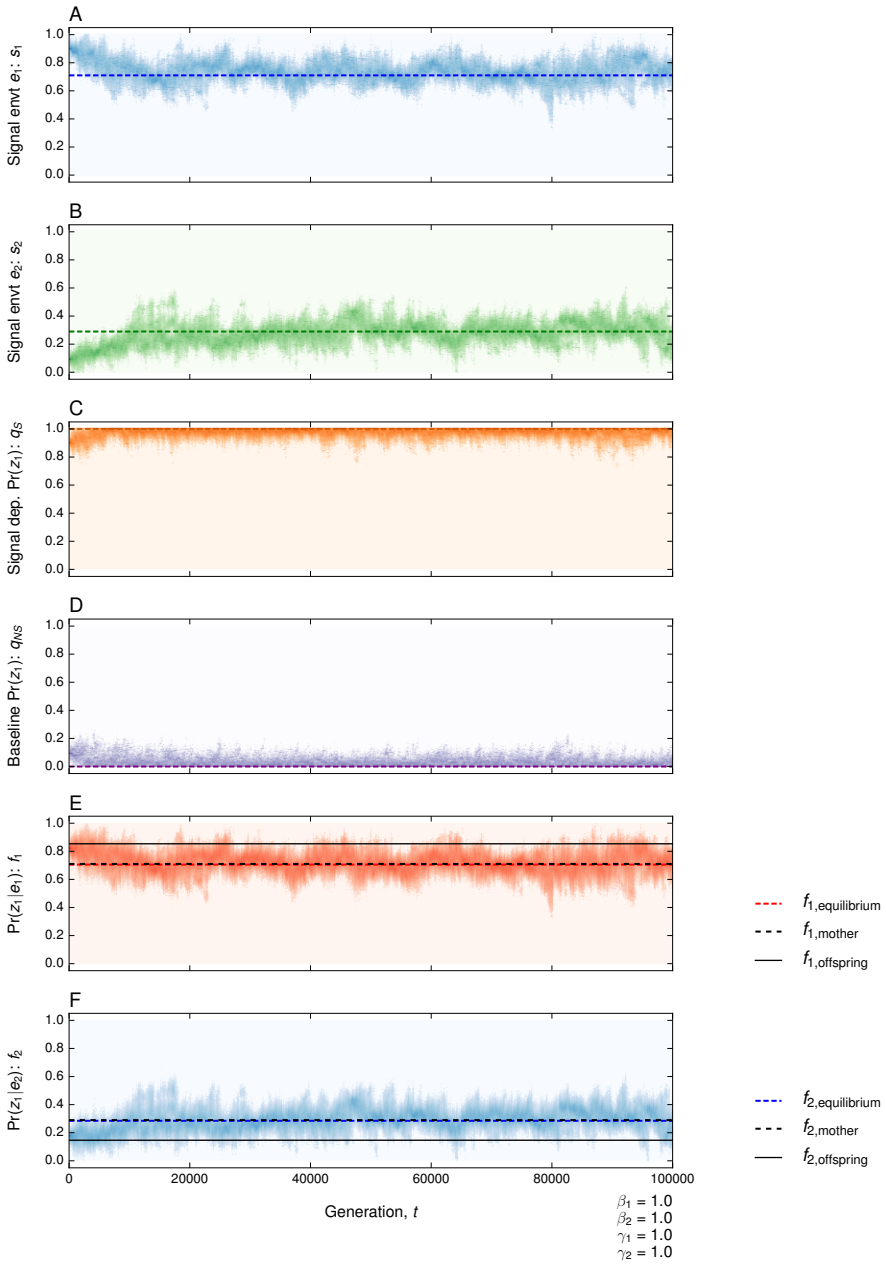
A
B
C
D
E
F
Signal envt e_1: s_1
Signal envt e_2: s_2
Signal dep. Pr(z_1): q_S
Baseline Pr(z_1): q_{NS}
Pr($z_1|e_1$): f_1
Pr($z_1|e_2$): f_2
1.0
0.8
0.6
0.4
0.2
0.0
0
20000
40000
60000
80000
100000
Generation, t
β_1 = 1.0
β_2 = 1.0
γ_1 = 1.0
γ_2 = 1.0
$f_{1,\text{equilibrium}}$
$f_{1,\text{mother}}$
$f_{1,\text{offspring}}$
$f_{2,\text{equilibrium}}$
$f_{2,\text{mother}}$
$f_{2,\text{offspring}}$

Figure S6:

Figure S7 Evolution towards an uninformative equilibrium where phenotype determination is identical across both environments: individual-based simulations. Phenotype z_2 is twice as costly to produce to mothers as phenotype z_1: $\beta_1 = \beta_2 = 1$, $\gamma_1 = \gamma_2 = 2$. See Figure S6 for a description of the individual panels and a listing of the corresponding parameters.

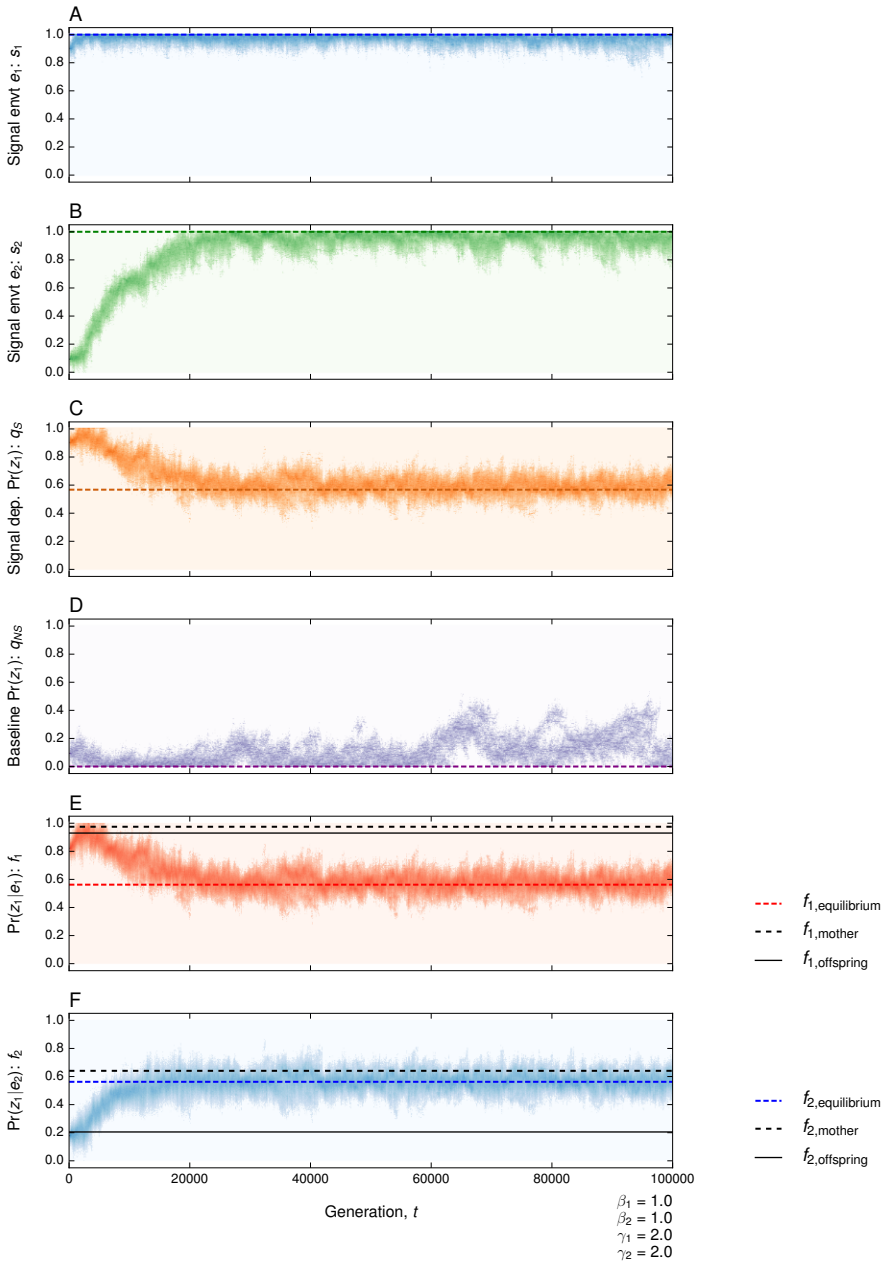
A
B
C
D
E
F
Signal envt e_1: s_1
Signal envt e_2: s_2
Signal dep. Pr(z_1): q_S
Baseline Pr(z_1): q_{NS}
Pr(z_1|e_1): f_1
Pr(z_1|e_2): f_2
1.0
0.8
0.6
0.4
0.2
0.0
0
20000
40000
60000
80000
100000
Generation, t
β_1 = 1.0
β_2 = 1.0
γ_1 = 2.0
γ_2 = 2.0
$f_{1,\text{equilibrium}}$
$f_{1,\text{mother}}$
$f_{1,\text{offspring}}$
$f_{2,\text{equilibrium}}$
$f_{2,\text{mother}}$
$f_{2,\text{offspring}}$

Figure S7:

Figure S8 Evolution towards a fully informative equilibrium where phenotype determination is strongly environment-dependent: individual-based simulations. Phenotype z_2 is twice as costly to produce to mothers as phenotype z_1 in environment e_1, while costs are reversed in environment e_1: $\beta_1 = 1,\ \beta_2 = 2,\ \gamma_1 = 2,\ \gamma_2 = 1$. See Figure S6 for a description of the individual panels and a listing of the corresponding parameters.

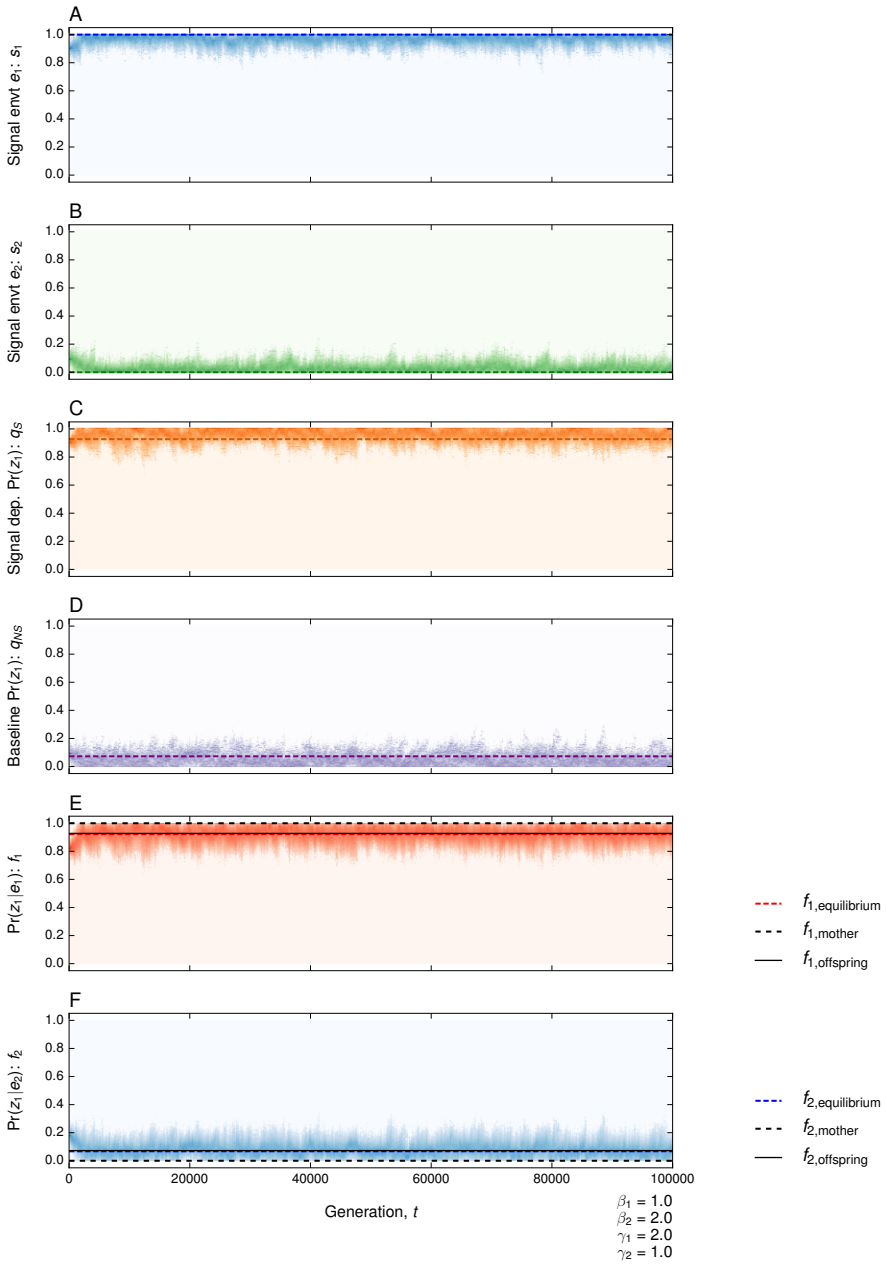
A
B
C
D
E
F
Signal envt e_1: s_1
Signal envt e_2: s_2
Signal dep. Pr(z_1): q_S
Baseline Pr(z_1): q_{NS}
Pr(z_1|e_1): f_1
Pr(z_1|e_2): f_2
1.0
0.8
0.6
0.4
0.2
0.0
0
20000
40000
60000
80000
100000
Generation, t
β_1 = 1.0
β_2 = 2.0
γ_1 = 2.0
γ_2 = 1.0
$f_{1,\text{equilibrium}}$
$f_{1,\text{mother}}$
$f_{1,\text{offspring}}$
$f_{2,\text{equilibrium}}$
$f_{2,\text{mother}}$
$f_{2,\text{offspring}}$

Figure S8:

S2 Model description

S2.1 Fitness expressions

Here we derive a demographical model of phenotype determination in a binary environment (see pp. 460-465 in Otto & Day, 2007 for a pedagogical introduction and Rousset, 2004; Lehmann & Rousset, 2010 for reviews). We use a direct fitness approach (Taylor & Frank, 1996; Taylor *et al.*, 2007), which calculates the expected number of descendants of a focal 'recipient allele' by summing the fitness effects of interactions with the different phenotypes of actors in the population (see Taylor & Frank, 1996 for an introduction to this approach).

Let w_{ji} describe the expected fitness of a mutant gene copy in an adult member of the population, reflecting the expected number of offspring (bearing the mutant gene copy) that successfully establish themselves as breeders in environment e_j when born from a focal mutant adult living in environment e_i. We assume that the focal mutant adult produces half its offspring via the female role (in which case it is fertilized by a random patch mate including itself), while the other half of its offspring result from sirings in the male role. We then have

$$\begin{aligned}
w_{ji} = {} & \frac{1}{2}\frac{\hat{f}_i^{\mathrm{f}}}{\tilde{f}_i^{\mathrm{f}}\beta_i + \left(1-\tilde{f}_i^{\mathrm{f}}\right)\gamma_i}\left[(1-d)\frac{n\sigma_{i\to j}\omega_{1j}}{C_{ji}\left(\bar{\mathbf{f}},\bar{\mathbf{f}}\right)} + d\sum_{k=1}^{2}\frac{p_k n\sigma_{k\to j}\omega_{1j}}{C_{jk}\left(\mathbf{f},\mathbf{f}\right)}\right] \\
& + \frac{1}{2}\frac{1-\hat{f}_i^{\mathrm{f}}}{\tilde{f}_i^{\mathrm{f}}\beta_i + \left(1-\tilde{f}_i^{\mathrm{f}}\right)\gamma_i}\left[(1-d)\frac{n\sigma_{i\to j}\omega_{2j}}{C_{ji}\left(\bar{\mathbf{f}},\bar{\mathbf{f}}\right)} + d\sum_{k=1}^{2}\frac{p_k n\sigma_{k\to j}\omega_{2j}}{C_{jk}\left(\mathbf{f},\mathbf{f}\right)}\right] \\
& + \frac{1}{2}\ell\frac{\hat{f}_i^{\mathrm{m}}}{\bar{f}_i\beta_i + \left(1-\bar{f}_i\right)\gamma_i}\left[(1-d)\frac{n\sigma_{i\to j}\omega_{1j}}{C_{ji}\left(\bar{\mathbf{f}},\bar{\mathbf{f}}\right)} + d\sum_{k=1}^{2}\frac{p_k n\sigma_{k\to j}\omega_{1j}}{C_{jk}\left(\mathbf{f},\mathbf{f}\right)}\right] \\
& + \frac{1}{2}\ell\frac{1-\hat{f}_i^{\mathrm{m}}}{\bar{f}_i\beta_i + \left(1-\bar{f}_i\right)\gamma_i}\left[(1-d)\frac{n\sigma_{i\to j}\omega_{2j}}{C_{ji}\left(\bar{\mathbf{f}},\bar{\mathbf{f}}\right)} + d\sum_{k=1}^{2}\frac{p_k n\sigma_{k\to j}\omega_{2j}}{C_{jk}\left(\mathbf{f},\mathbf{f}\right)}\right] \\
& + \frac{1}{2}(1-\ell)\sum_{k=1}^{2}p_k\frac{\hat{f}_k^{\mathrm{m,remote}}}{f_k\beta_k + (1-f_k)\gamma_k}\left[(1-d)\frac{n\sigma_{k\to j}\omega_{1j}}{C_{jk}\left(\mathbf{f},\mathbf{f}\right)} + d\sum_{h=1}^{2}\frac{p_h n\sigma_{h\to j}\omega_{1j}}{C_{jh}\left(\mathbf{f},\mathbf{f}\right)}\right] \\
& + \frac{1}{2}(1-\ell)\sum_{k=1}^{2}p_k\frac{1-\hat{f}_k^{\mathrm{m,remote}}}{f_k\beta_k + (1-f_k)\gamma_k}\left[(1-d)\frac{n\sigma_{k\to j}\omega_{2j}}{C_{jk}\left(\mathbf{f},\mathbf{f}\right)} + d\sum_{h=1}^{2}\frac{p_h n\sigma_{h\to j}\omega_{2j}}{C_{jh}\left(\mathbf{f},\mathbf{f}\right)}\right]. \qquad \text{(S1)}
\end{aligned}$$

The first line in the expression above reflects the number of successfully established mutant offspring who attain the z_1 phenotype, born from a focal adult individual acting as a female. The strategy $\hat{f}_i^{\mathrm{f}}$ denotes the probability that a focal's own offspring who bear the mutant allele (denoted by a ^) attain the z_1 phenotype when born in in environment e_i and when the focal adult individual acts as a female (denoted by superscript f). Following classical life-history theory (Smith & Fretwell, 1974), we assume that the focal's fecundity in the female role is given by $1/E_i(\tilde{f}_i^{\mathrm{f}})$, where $E_i(\tilde{f}_i^{\mathrm{f}})$ is the average investment per offspring in environment e_i: $E_i(\tilde{f}_i^{\mathrm{f}}) = \tilde{f}_i^{\mathrm{f}}\beta_i + \left(1-\tilde{f}_i^{\mathrm{f}}\right)\gamma_i$ (see section "phenotype determination" in the main text). Here, $\tilde{f}_i^{\mathrm{f}}$ denotes the average phenotype determination strategy in the focal's brood (denoted by a $\sim$) when the focal adult individual acts as a female. The part in straight brackets describes the fitness of the focal's offspring upon birth, who remain at the natal patch with probability $1-d$, subsequently experience environmental change

with probability $\sigma_{i\to j}$ and survive with probability ω_{1j} in the novel e_j environment. After survival, the focal's offspring compete with a total number of $C_{ji}(\bar{\mathbf{f}},\mathbf{f})$ philopatric and immigrant offspring for each of the n breeding positions (see eq. [S2] below), where $\bar{\mathbf{f}}=[\bar{f}_1,\bar{f}_2]$ and $\mathbf{f}=[f_1,f_2]$ denote the average phenotype determination strategies in the local patch and the population as a whole respectively. Alternatively, offspring disperse with probability d to randomly chosen remote patch. With probability $p_k=\sigma_{j\to k}/(\sigma_{k\to j}+\sigma_{j\to k})$, the environmental state of this patch is e_k. Following environmental change and survival, offspring compete with all $C_{jk}(\mathbf{f},\mathbf{f})$ juveniles for one of the n adult breeding positions. The second line in eq. (S1) reflects the successful establishment of z_2 offspring born from a focal adult when acting in the female role and can be derived in a similar fashion as the first line. Lines three and four reflect the expected number of successfully established z_1 and z_2 offspring when the focal adult acts as a male who mates randomly in the local patch (with probability ℓ), with the focal's strategy given by $\hat{f}^{\mathrm{m}}$Fecundity is now given by the average fecundity of all females in the local patch $1/(\bar{f}_i\beta_i+\left(1-\bar{f}_i\right)\gamma_i)$, where $\bar{f}_i$ is the average phenotype of offspring born in the local patch. Finally, lines five and six reflect the expected number of successfully established z_1 and z_2 offspring when the focal adult acts as male who mates in a randomly chosen remote patch (with probability $1-\ell$). In this case case the phenotype determination of the focal offspring in environment e_k is given by $\hat{f}_k^{\mathrm{m,remote}}$.

The total number of $C_{ji}(\bar{\mathbf{f}},\mathbf{f})$ surviving offspring that compete for breeding positions in e_i patch that later changes to environmental state e_j is given by

$$C_{ji}(\bar{\mathbf{f}},\mathbf{f})=\frac{n}{\bar{f}_i\beta_i+\left(1-\bar{f}_i\right)\gamma_i}(1-d)\left(\bar{f}_i\omega_{1j}+\left(1-\bar{f}_i\right)\omega_{2j}\right)+m_j(\mathbf{f})\,. \tag{S2}$$

The first part in the expression above reflects the total number of offspring produced in the natal patch, which is given by a female's average fecundity in the local patch $1/[\bar{f}_i\beta_i+(1-\bar{f}_i)\gamma_i]$ times the number of locally breeding females n. A proportion $1-d$ of these offspring remain in the natal patch and survive with probability $\bar{f}_i\omega_{1j}+(1-\bar{f}_i)w_{2j}$. The term $m_j(\mathbf{f})$ in the expression above reflects the total number of juveniles that are immigrant to the e_j patch:

$$m_j(\mathbf{f})=d\sum_{k=1}^{2}p_k\frac{n}{f_k\beta_k+(1-f_k)\gamma_k}\left(f_k\omega_{1j}+(1-f_k)\omega_{2j}\right), \tag{S3}$$

where immigrants have been born in patches in environmental state e_k (at frequency p_k), and the probability of producing a z_1 offspring is given by the global average phenotype f_k. Note that survival is determined by the environment of arrival, e_j.

Finally, the mutant transition matrix $\mathbf{B}$, with elements $b_{ji}=w_{ji}$, then comprises the transition probabilities between all the different classes of mutants:

$$\mathbf{B}=\begin{bmatrix} w_{11} & w_{12}\\ w_{21} & w_{22}\end{bmatrix}, \tag{S4}$$

while the matrix $\mathbf{A}\equiv\mathbf{B}|_{\hat{\mathbf{f}}^{\mathrm{f}}=\hat{\mathbf{f}}^{\mathrm{m}}=\tilde{\mathbf{f}}^{\mathrm{f}}=\tilde{\mathbf{f}}^{\mathrm{m}}=\bar{\mathbf{f}}=\mathbf{f}}$ denotes the resident transition matrix which comprises the transition probabilities between the different classes of residents.

S2.2 Selection gradients

Using a direct fitness approach (Taylor & Frank, 1996; Taylor *et al.*, 2007) and a standard result on evolution in class structured populations (e.g., Box 10.2 in Otto & Day 2007), the selection gradient

$\mathcal{F}_i$ on the phenotype determination strategy f_i is then given by

$$\mathcal{F}_i = \sum_{j=\{1,2\}} \sum_{k=\{1,2\}} v_j u_k \left[\frac{\partial b_{jk}}{\partial \hat{f}_i^{\mathrm{f}}} \hat{r}_k^{\mathrm{f}} + \frac{\partial b_{jk}}{\partial \tilde{f}_i^{\mathrm{f}}} \tilde{r}_k^{\mathrm{f}} + \frac{\partial b_{jk}}{\partial \hat{f}_i^{\mathrm{m}}} \hat{r}_k^{\mathrm{m}} + \frac{\partial b_{jk}}{\partial \hat{f}_i^{\mathrm{m,remote}}} \hat{f}_k^{\mathrm{m,remote}} + \frac{\partial b_{jk}}{\partial \bar{f}_i} \bar{r}_k \right] \Bigg|_{\hat{\mathbf{f}}^{\mathrm{f}}=\hat{\mathbf{f}}^{\mathrm{m}}=\tilde{\mathbf{f}}^{\mathrm{f}}=\tilde{\mathbf{f}}^{\mathrm{m}}=\bar{\mathbf{f}}=\mathbf{f}}, \tag{S5}$$

where v_j is the reproductive value of adults that have established themselves in a patch in environmental state e_j patch and u_k the stable class frequency of adult breeders in environment e_k. In addition, $\hat{r}_k^{\mathrm{f}}$ and $\hat{r}_k^{\mathrm{m}}$ denote relatedness between a focal adult individual living in environment e_k and its locally produced offspring born from its eggs and sperm respectively, while $\tilde{r}_k^{\mathrm{f}}$ denotes relatedness between a focal adult individual living in environment e_k and any offspring in its brood when acting as a female. The coefficient $\hat{r}_k^{\mathrm{m,remote}}$ denotes relatedness between a focal adult male and its remotely sired offspring. Finally, $\bar{r}_k$ denotes relatedness between a focal adult and any locally born offspring. Expressions for these relatedness coefficients are derived below. In addition, expressions for reproductive values $\mathbf{v} = [v_1, v_2]$ and $\mathbf{u} = [u_1, u_2]^{\mathrm{T}}$ (T denoting transposition) are obtained by calculating the left and right eigenvectors of the resident transition matrix $\mathbf{A}$. However, results are long and not very informative, so we will not provide these eigenvectors here.

S2.3 Identity between allele copies

Writing Q_i for the equilibrium probability of identity between two copies of a phenotype determination allele sampled in two distinct adults (i.e., sampling without replacement) in environment i, we have

$$Q_i = \sum_{k=1}^{2} \frac{p_k \sigma_{k\to i}}{\sum_{\ell=1}^{2} p_\ell \sigma_{\ell\to i}} h_{ki}^2 \left(\frac{1}{4} + \frac{1}{2}\ell + \frac{1}{4}\ell^2 \right) \left(\frac{1}{n} + \frac{n-1}{n} Q_k \right) \tag{S6}$$

$$h_{ki} = \frac{(1-d)}{C_{ki}(\mathbf{f},\mathbf{f})} \frac{f_k \omega_{1i} + (1-f_k)\omega_{2i}}{f_k \beta_k + (1-f_k)\gamma_k}. \tag{S7}$$

With probability $p_k \sigma_{k\to i} / \sum_{\ell=1}^{2} p_\ell \sigma_{\ell\to i}$ the local patch was in environmental state e_k at the time the current adults were born. Both sampled individuals are native to the local patch with probability h_{ki}^2. Both sampled individuals either have inherited their allele copies maternally with probability $1/4$. Both individuals either descend from the same mother with probability $1/n$ or from a different mother with probability $(n-1)/n$, in which case both mothers share alleles with probability Q_k. With probability $2 \times \frac{1}{4}\ell$, one individual has inherited its allele from a locally mating father while the other individual inherited its allele maternally. Again, with probability $1/n$, both sampled individuals either descend from the same parent, whereas with probability $(n-1)/n$ both descend from different parents. Finally, with probability $\frac{1}{4}\ell^2$, both individuals have inherited their alleles from a locally mating father. We can then solve the recursions in equation (S6) for the equilibrium values of $\hat{Q}_1$ and $\hat{Q}_2$ and substitute the resulting values in the expressions below.

S2.4 Maternal expression: relatedness

To calculate relatedness, we require an expression of the regression r_k between the recipient's genotypic value G and the phenotype P_k of the kth actor: $r_k = \mathrm{cov}(G, P_k) / \mathrm{cov}(G, P)$ (e.g., eq. [4] in

Taylor *et al.*, 2007). As we are considering haploid inheritance, $\mathrm{cov}(G,P) = 1$, so we only require an expression for $\mathrm{cov}(G,P_k)$. The coefficient $\hat{r}_i^{\mathrm{f}}$ reflects the covariance between the genetic value of a focal adult recipient who is, by definition, a mutant, hence $G = 1$ and the phenotypes $P_{\mathrm{off,eggs}}$ of her own mutant offspring born from her eggs. In case of maternal expression, the phenotypes of these offspring are expressed by the focal mutant mother itself, so these offspring have a mutant phenotype with certainty ($P_{\mathrm{off,eggs}} = 1$). Hence, we have

$$\hat{r}_i^{\mathrm{f}} = 1. \tag{S8}$$

The relatedness coefficient $\hat{r}_i^{\mathrm{m}}$ reflects the covariance between the genetic value of a focal mutant adult recipient and its own, locally sired, mutant offspring born from its sperm. Again $G = 1$ by definition as the focal bears the mutant allele. With probability $1/n$, the focal adult is also the mother (selfing), so that $P_{\mathrm{off,sperm}} = 1$. Alternatively, with probability $(1-n)/n$, another local adult is the mother, and the probability that she bears the mutant allele is $\hat{Q}_i$, in which case $P_{\mathrm{off,sperm}} = 1$. In any other case, $P_{\mathrm{off,sperm}} = 0$. Hence, we have

$$\hat{r}_i^{\mathrm{m}} = \frac{1}{n} + \frac{n-1}{n}\hat{Q}_i. \tag{S9}$$

Note that ℓ does not occur in the expression above as $\hat{r}_i^{\mathrm{m}}$ is conditional upon a sire mating locally.

The relatedness coefficient $\hat{r}_i^{\mathrm{m,remote}}$ is given by

$$\hat{r}_i^{\mathrm{m,remote}} = 0,$$

as the phenotype of a male's remotely sired offspring is determined by a remote mother, who does not bear the mutant allele.

The relatedness coefficient $\tilde{r}_i^{\mathrm{f}}$ reflects the covariance between the focal mutant adult ($G = 1$) when acting as a female and the average phenotype of her brood. However, as all offspring in her brood express the mutant maternal phenotype by definition, we have

$$\tilde{r}_i^{\mathrm{f}} = 1. \tag{S10}$$

The relatedness coefficient $\bar{r}_i$ between a focal mutant adult and any locally born juvenile is

$$\bar{r}_i = \frac{1}{n} + \frac{n-1}{n}\hat{Q}_i \tag{S11}$$

as with probability $1/n$, the locally born offspring has the mutant adult recipient as its mother, so that the phenotype is given by $P_{\mathrm{off}} = 1$. Alternatively, with probability $(n-1)/n$, the locally born offspring has another mother, who shares the mutant allele with probability $\hat{Q}_i$.

S2.5 Offspring expression: relatedness

The relatedness coefficient $\hat{r}_i^{\mathrm{f}}$ reflects the covariance between a focal mutant adult female recipient ($G = 1$) and the phenotype of her mutant offspring. As the phenotype is expressed by these offspring, $P_k = 1$, so

$$\hat{r}_i^{\mathrm{f}} = 1. \tag{S12}$$

By the same argument, $\hat{r}_i^{\mathrm{m}}$ in the case of offspring control is given by

$$\hat{r}_i^{\mathrm{m}} = \hat{r}_i^{\mathrm{m,remote}} = 1. \tag{S13}$$

While $\tilde{r}_i^{\mathrm{f}}$ is now given by

$$\tilde{r}_i^{\mathrm{f}} = \frac{1}{2} + \frac{1}{2}\ell\left(\frac{1}{n} + \frac{n-1}{n}\hat{Q}_i\right) \tag{S14}$$

as $1/2$ of the brood, on average, have received an allele copy from their focal mutant mother (hence a mutant copy by definition), and the phenotype of those offspring is $P_k = 1$. By contrast, the other $1/2$ of the brood receive their allele copy paternally. With probability ℓ, the father is one of the local breeders: with probability $1/n$, mother and father are identical (selfing occurs), so that again offspring inherit a mutant copy. With probability $(n-1)/n$, however, individuals have a distinct mother and father, and both share alleles with probability $\hat{Q}_i$.

Relatedness $\bar{r}_i$ between a focal adult individual and any locally born offspring is given by

$$\bar{r}_i = \frac{1}{2}\left(\frac{1}{n} + \frac{n-1}{n}\hat{Q}_i\right) + \frac{1}{2}\ell\left(\frac{1}{n} + \frac{n-1}{n}\hat{Q}_i\right), \tag{S15}$$

where with probability $1/2$, the sampled locally born offspring inherits its gene copy maternally. With probability $1/n$, it is born from the focal adult, so that identity is 1. Alternatively, with probability $(n-1)/n$, the individual has a different mother, who bears the mutant allele with probability $\hat{Q}_i$. Alternatively, with probability $1/2 \cdot \ell$, the sampled offspring inherits its gene copy paternally and the father is one of the local individuals. With probability $1/n$, the father is the focal individual, whereas with probability $(n-1)/n$, the father is one of the other individuals in the local patch.

S2.6 Numerical solutions

To calculate the battleground, we calculate the equilibrium values of f_1^* and f_2^* by numerically iterating the dynamic (Dieckmann & Law, 1996; McGill & Brown, 2007)

$$\begin{bmatrix} f_1 \\ f_2 \end{bmatrix}_{t+1} = \begin{bmatrix} f_1 \\ f_2 \end{bmatrix}_t + C \begin{bmatrix} \mathcal{F}_1 \\ \mathcal{F}_2 \end{bmatrix}_t$$

from the initial point $[f_1, f_2]_{t=0} = [0.5, 0.5]$ until convergence. The parameter C denotes a constant which reflects the effective rate at which new mutations arise in a population (e.g., McGill & Brown, 2007; Dercole & Rinaldi, 2008), which is $C = 0.01$ throughout the current study. We assume that convergence occurs when $|f_{i,t} - f_{i,t-1}| < 10^{-8}$. Numerical iterations are coded in Mathematica and C and are available on github: https://doi.org/10.5281/zenodo.826693.

S2.7 Resolving the conflict: fitness expressions

We note that the probability f_i of producing z_1 offspring in environment e_i is now given by

$$f_i(s_i, q_S, q_{NS}) = s_i q_S + (1 - s_i) q_{NS}. \tag{S16}$$

Considering the evolution of maternal signaling, we write $f_i(s_i) \equiv f_i(s_i, q_S, q_{NS})$.We then have

$$
\begin{aligned}
w_{ji} = {} & \frac{1}{2}\frac{f_i\left(\hat{s}_i^{\mathrm{f}}\right)}{f_i\left(\tilde{s}_i^{\mathrm{f}}\right)\beta_i+\left(1-f_i\left(\tilde{s}_i^{\mathrm{f}}\right)\right)\gamma_i}\left[(1-d)\frac{n\sigma_{i\to j}\omega_{1j}}{C_{ji}(\bar{\mathbf{s}},\mathbf{s})}+d\sum_{k=1}^{2}\frac{p_k n\sigma_{k\to j}\omega_{1j}}{C_{jk}(\mathbf{s},\mathbf{s})}\right] \\
& +\frac{1}{2}\frac{f_i\left(\hat{s}_i^{\mathrm{f}}\right)}{f_i\left(\tilde{s}_i^{\mathrm{f}}\right)\beta_i+\left(1-f_i\left(\tilde{s}_i^{\mathrm{f}}\right)\right)\gamma_i}\left[(1-d)\frac{n\sigma_{i\to j}\omega_{2j}}{C_{ji}(\bar{\mathbf{s}},\mathbf{s})}+d\sum_{k=1}^{2}\frac{p_k n\sigma_{k\to j}\omega_{2j}}{C_{jk}(\mathbf{s},\mathbf{s})}\right] \\
& +\frac{1}{2}\ell\frac{f_i\left(\hat{s}_i^{\mathrm{m}}\right)}{f_i(\bar{s}_i)\beta_i+(1-f_i(\bar{s}_i))\gamma_i}\left[(1-d)\frac{n\sigma_{i\to j}\omega_{1j}}{C_{ji}(\bar{\mathbf{s}},\mathbf{s})}+d\sum_{k=1}^{2}\frac{p_k n\sigma_{k\to j}\omega_{1j}}{C_{jk}(\mathbf{s},\mathbf{s})}\right] \\
& +\frac{1}{2}\ell\frac{f_i\left(\hat{s}_i^{\mathrm{m}}\right)}{f_i(\bar{s}_i)\beta_i+(1-f_i(\bar{s}_i))\gamma_i}\left[(1-d)\frac{n\sigma_{i\to j}\omega_{2j}}{C_{ji}(\bar{\mathbf{s}},\mathbf{s})}+d\sum_{k=1}^{2}\frac{p_k n\sigma_{k\to j}\omega_{2j}}{C_{jk}(\mathbf{s},\mathbf{s})}\right] \\
& +\frac{1}{2}(1-\ell)\sum_{k=1}^{2}p_k\frac{f_k\left(\hat{s}_k^{\mathrm{m,remote}}\right)}{f_k(s_k)\beta_k+(1-f_k(s_k))\gamma_k}\left[(1-d)\frac{n\sigma_{k\to j}\omega_{1j}}{C_{jk}(\mathbf{s},\mathbf{s})}+d\sum_{h=1}^{2}\frac{p_h n\sigma_{h\to j}\omega_{1j}}{C_{jh}(\mathbf{s},\mathbf{s})}\right] \\
& +\frac{1}{2}(1-\ell)\sum_{k=1}^{2}p_k\frac{f_k\left(\hat{s}_k^{\mathrm{m,remote}}\right)}{f_k(s_k)\beta_k+(1-f_k(s_k))\gamma_k}\left[(1-d)\frac{n\sigma_{k\to j}\omega_{2j}}{C_{jk}(\mathbf{s},\mathbf{s})}+d\sum_{h=1}^{2}\frac{p_h n\sigma_{h\to j}\omega_{2j}}{C_{jh}(\mathbf{s},\mathbf{s})}\right]. \qquad \text{(S17)}
\end{aligned}
$$

The total number C_{ji} of locally competing juveniles in an environment e_j patch that was previously in state e_i is given by

$$
C_{ji}(\bar{\mathbf{s}},\mathbf{s}) = \frac{n}{f_i(\bar{s}_i)\beta_i+(1-f_i(\bar{s}_i))\gamma_i}(1-d)\left(f_i(\bar{s}_i)\omega_{1j}+(1-f_i(\bar{s}_i))\omega_{2j}\right)+m_j(\mathbf{s}) \qquad \text{(S18)}
$$

$$
m_j(\mathbf{s}) = d\sum_{k=1}^{2}p_k\frac{n}{f_i(s_i)\beta_i+(1-f_k(s_i))\gamma_i}\left(f_i(s_i)\omega_{1j}+(1-f_k(s_i))\omega_{2j}\right). \qquad \text{(S19)}
$$

With regards to offspring responsiveness to the maternal signal, we write $\mathbf{q}=[q_S,q_{NS}]$ and $f_i(\mathbf{q})\equiv f_i(s_i,q_S,q_{NS})$. We then have

$$
\begin{aligned}
w_{ji} = {} & \frac{1}{2}\frac{f_i\left(\hat{\mathbf{q}}^{\mathrm{f}}\right)}{f_i\left(\tilde{\mathbf{q}}^{\mathrm{f}}\right)\beta_i+\left(1-f_i\left(\tilde{\mathbf{q}}^{\mathrm{f}}\right)\right)\gamma_i}\left[(1-d)\frac{\sigma_{i\to j}\omega_{1j}}{C_{ji}(\bar{\mathbf{q}},\mathbf{q})}+d\sum_{k=1}^{2}\frac{p_k\sigma_{k\to j}\omega_{1j}}{C_{jk}(\mathbf{q},\mathbf{q})}\right] \\
& +\frac{1}{2}\frac{f_i\left(\hat{\mathbf{q}}^{\mathrm{f}}\right)}{f_i\left(\tilde{\mathbf{q}}^{\mathrm{f}}\right)\beta_i+\left(1-f_i\left(\tilde{\mathbf{q}}^{\mathrm{f}}\right)\right)\gamma_i}\left[(1-d)\frac{\sigma_{i\to j}\omega_{2j}}{C_{ji}(\bar{\mathbf{q}},\mathbf{q})}+d\sum_{k=1}^{2}\frac{p_k\sigma_{k\to j}\omega_{2j}}{C_{jk}(\mathbf{q},\mathbf{q})}\right] \\
& +\frac{1}{2}\ell\frac{f_i\left(\hat{\mathbf{q}}^{\mathrm{m}}\right)}{f_i(\bar{\mathbf{q}})\beta_i+(1-f_i(\bar{\mathbf{q}}))\gamma_i}\left[(1-d)\frac{\sigma_{i\to j}\omega_{1j}}{C_{ji}(\bar{\mathbf{q}},\mathbf{q})}+d\sum_{k=1}^{2}\frac{p_k\sigma_{k\to j}\omega_{1j}}{C_{jk}(\mathbf{q},\mathbf{q})}\right] \\
& +\frac{1}{2}\ell\frac{f_i\left(\hat{\mathbf{q}}^{\mathrm{m}}\right)}{f_i(\bar{\mathbf{q}})\beta_i+(1-f_i(\bar{\mathbf{q}}))\gamma_i}\left[(1-d)\frac{\sigma_{i\to j}\omega_{2j}}{C_{ji}(\bar{\mathbf{q}},\mathbf{q})}+d\sum_{k=1}^{2}\frac{p_k\sigma_{k\to j}\omega_{2j}}{C_{jk}(\mathbf{q},\mathbf{q})}\right] \\
& +\frac{1}{2}(1-\ell)\sum_{k=1}^{2}p_k\frac{f_k\left(\hat{\mathbf{q}}^{\mathrm{m,remote}}\right)}{f_k(\mathbf{q})\beta_k+(1-f_k(\mathbf{q}))\gamma_k}\left[(1-d)\frac{\sigma_{k\to j}\omega_{1j}}{C_{jk}(\mathbf{q},\mathbf{q})}+d\sum_{h=1}^{2}\frac{p_h\sigma_{h\to j}\omega_{1j}}{C_{jh}(\mathbf{q},\mathbf{q})}\right]
\end{aligned}
$$

$$+\frac{1}{2}(1-\ell)\sum_{k=1}^{2}p_k\frac{f_k\left(\hat{\mathbf{q}}^{\mathrm{m,remote}}\right)}{f_k(\mathbf{q})\beta_k+(1-f_k(\mathbf{q}))\gamma_k}\left[(1-d)\frac{\sigma_{k\to j}\omega_{2j}}{C_{jk}(\mathbf{q},\mathbf{q})}+d\sum_{h=1}^{2}\frac{p_h\sigma_{h\to j}\omega_{2j}}{C_{jh}(\mathbf{q},\mathbf{q})}\right]. \tag{S20}$$

Expressions for $C_{ji}(\mathbf{q},\mathbf{q})$ and $m_j(\mathbf{q})$ can be derived in the same fashion as shown for eqns. (S18, S19).

S2.8 Resolving the conflict: selection gradients

Similar to eq. (S5), selection gradients on the maternal signal s_i are given by

$$\mathcal{S}_i=\sum_{j=\{1,2\}}\sum_{k=\{1,2\}}v_ju_k\left[\frac{\partial b_{jk}}{\partial\hat{s}_i^{\mathrm{f}}}\hat{r}_k^{\mathrm{f}}+\frac{\partial b_{jk}}{\partial\tilde{s}_i^{\mathrm{f}}}\tilde{s}_k^{\mathrm{f}}+\frac{\partial b_{jk}}{\partial\hat{s}_i^{\mathrm{m}}}\hat{r}_k^{\mathrm{m}}+\frac{\partial b_{jk}}{\partial\hat{s}_i^{\mathrm{m,remote}}}\hat{r}_k^{\mathrm{m,remote}}+\frac{\partial b_{jk}}{\partial\bar{s}_i}\bar{r}_k\right]\Bigg|_{\hat{\mathbf{s}}^{\mathrm{f}}=\hat{\mathbf{s}}^{\mathrm{m}}=\tilde{\mathbf{s}}^{\mathrm{f}}=\bar{\mathbf{s}}=\mathbf{s}}, \tag{S21}$$

with relatedness coefficients given by eqns. (S8-S11). Selection gradients on the offspring phenotype determination strategy $q_i\in(q_S,q_{NS})$ are given by

$$\mathcal{Q}_i=\sum_{j=\{1,2\}}\sum_{k=\{1,2\}}v_ju_k\left[\frac{\partial b_{jk}}{\partial\hat{q}_i^{\mathrm{f}}}\hat{r}_k^{\mathrm{f}}+\frac{\partial b_{jk}}{\partial\tilde{s}_i^{\mathrm{f}}}\tilde{s}_k^{\mathrm{f}}+\frac{\partial b_{jk}}{\partial\hat{q}_i^{\mathrm{m}}}\hat{r}_k^{\mathrm{m}}+\frac{\partial b_{jk}}{\partial\hat{q}_i^{\mathrm{m,remote}}}\hat{r}_k^{\mathrm{m,remote}}+\frac{\partial b_{jk}}{\partial\bar{q}_i}\bar{r}_k\right]\Bigg|_{\hat{\mathbf{q}}^{\mathrm{f}}=\hat{\mathbf{q}}^{\mathrm{m}}=\tilde{\mathbf{q}}^{\mathrm{f}}=\bar{\mathbf{q}}=\mathbf{q}}, \tag{S22}$$

with relatedness coefficients given by eqns. (S12-S15).

S2.9 Individual-based simulations

We also ran individual-based simulations to check that our model is robust to demographic stochasticity, finite population sizes and genetic variation. Moreover, individual-based simulations also showed that populations that arrived at the candidate ESS signaling or phenotype determination strategy did not undergo evolutionary branching (Geritz *et al.*, 1998). To this end, we simulated a population of $N=2000$ hermaphroditic individuals distributed over N/n patches. At the start of each generation, each individual chooses a random sperm donor among the n individuals in the local patch (including itself). Each individual bears four unlinked, diploid gene loci, which correspond to the traits s_1, s_2, q_S and q_{NS}, where alleles of each locus were assumed to have additive effects. Upon inheritance, each allele independently mutates with probability μ, which involves adding a random number from a normal distribution with mean 0 and variance $\nu^2=1\times10^{-4}$ to its current allelic value (i.e., a continuum-of-alleles model: Kimura & Crow, 1964). Subsequently, an offspring's phenotype is determined by drawing a randomly chosen number X from a uniform distribution: the offspring will attain a z_1 phenotype in case $X<s_i^{\mathrm{mother}}q_S^{\mathrm{offspring}}+(1-s_i^{\mathrm{mother}})q_{NS}^{\mathrm{offspring}}$ and a z_2 phenotype otherwise. Superscripts 'mother' and 'offspring' denote whether the trait value is expressed by mother or offspring. At the start of each generation, each adult breeder has $R=200$ units of resources to produce offspring, where the costs of producing an individual offspring is given by the parameters β_i and γ_i (see section "phenotype determination" in the main text). After

birth, offspring disperse with probability d to a randomly chosen patch. Subsequently, the environment in each patch changes with probability $\sigma_{i\to j}$, after which survival takes place among the immigrant and philopatric juveniles in each patch. Maladapted individuals survive with a probability c_1 or c_2 dependent on the local environment, whereas adapted individuals always survive. For each patch, juveniles are then randomly drawn to replace the adult breeders, after which the cycle repeats. Unless indicated otherwise, simulations ran for 20 000 generations, which guaranteed convergence to equilibria for all inspected cases. Simulations are coded in C and are available at zenodo/github: https://doi.org/10.5281/zenodo.826693.