Evolution of epigenetic transmission when selection acts on fecundity versus viability

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Abstract

Existing theory on the evolution of parental effects and the inheritance of nongenetic factors has mostly focused on the role of environmental change. By contrast, how differences in population demography and life history affect parental effects is poorly understood. To fill this gap, we develop an analytical model to explore how parental effects evolve when selection acts on fecundity versus viability in spatiotemporally fluctuating environments. We find that regimes of viability selection, but not fecundity selection, are most likely to favour parental effects. In case of viability selection, locally adapted phenotypes have a higher survival than maladapted phenotypes and hence become enriched in the local environment. Hence, simply by being alive, a parental phenotype becomes correlated to its environment (and hence informative to offspring) during its lifetime, favouring the evolution of parental effects. By contrast, in regimes of fecundity selection, correlations between phenotype and environment develop more slowly: this is because locally adapted and maladapted parents survive at equal rates (no survival selection), so that parental phenotypes, by themselves, are uninformative about the local environment. However, because locally adapted parents are more fecund, they contribute more offspring to the local patch than maladapted parents. In case these offspring are also likely to inherit the adapted parents' phenotypes (requiring pre-existing inheritance), locally adapted offspring become enriched in the local environment, resulting in a correlation between phenotype and environment, but only in the offspring's generation. Because of this slower build-up of a correlation between phenotype and environment essential to parental effects, fecundity selection is more sensitive to any distortions due to environmental change than viability selection. Hence, we conclude that viability selection is most conducive to the evolution of parental effects.

Keywords: parental effects, nongenetic inheritance, phenotypic plasticity, information, transgenerational effects, environmental change
Running title: Fecundity vs viability and epigenetics

1 Introduction

An accumulating number of studies shows that parents provide a range of inputs that contribute to offspring development [1–3]. These parental effects can be mediated by a variety of mechanisms, such as maternal hormones [4], the transmission of chromatin modifications and epigenetic variants [5, 6], maternal nutrients (e.g., [7]) or the transmission of behaviour via social learning [8]. Crucially, these parental influences are a form of transgenerational plasticity, in which the phenotype of an offspring not only depends on its genes and its current environment, but also on the phenotypes or environments of its parents and grandparents [3, 9–11]. In order to understand when and where transgenerational plasticity is likely to be important, predictions are needed about the ecological and social conditions that favour versus disfavour parental influences on offspring phenotype determination.

Indeed, a series of models have started to consider the evolution of parental effects, finding that they are particularly likely to evolve in fluctuating environments in which parental and offspring environments are correlated [e.g., 12–16] and when this environment imposes strong selection [17, 18]. While these models have provided the field with novel and testable predictions (e.g., see [19]), a shortcoming is that most of these models focus exclusively on the role of environmental variability. By contrast, it is currently poorly understood whether other factors such as demography and life history are also of importance when predicting the evolution of transgenerational plasticity [20].

One major point of focus in life history theory are the differential consequences of fecundity versus viability selection. In certain cases, adapted individuals produce larger numbers of offspring than other individuals (fecundity selection; [21]), whereas in other cases such individuals instead experience higher levels of survival (viability selection). Here we therefore ask whether the evolution of transmission of epigenetic information to the next generation is differentially affected by fecundity versus viability selection.

Previous work on the evolution of mutation rates [22, 23] and stochastic epigenetic phenotype switching [24–27] in fluctuating environments gives us little indication that the mode of selection matters at all to the evolution of inheritance mechanisms: selection in these models can represent selection on fecundity or viability without much difference. Only when selection acts on combinations of male and female genotypes (fertility selection in the classical population genetic sense: [28]), have effects on the mutation rate been found [29]. However, when selection results in individual differences in fecundity regardless of partner phenotype, the mode of selection is irrelevant.

As these previous studies focus exclusively on well-mixed populations, it raises the question whether fecundity and viability selection still lead to equivalent results when relatives interact. It is well understood that larger amounts of phenotypic variability are selectively favoured when relatives interact, as this makes it more likely that at least some bearers of the same genotype (but with a different phenotype) are likely to survive environmental change [30–33]. Indeed, a previous model [20] showed that interactions between relatives increased the propensity for any parental phenotypic influences on offspring phenotype determination, as it allowed for greater phenotypic variation between offspring than can be achieved through genetic transmission alone. However, the model in [20] exclusively considered viability selection, raising the question how robust these results are to the mode of selection. From studies that focus on

the evolution of helping and altruism, we already know that fecundity and viability selection lead to differences in the structure of competition among kin [34–37], which could potentially also affect offspring sensitivity to the inheritance of parental epigenetic variants.

In this study, we assess the role of fecundity versus viability selection on the transmission of epigenetic variants from parents to offspring. For the sake of tractability, we consider an individual's phenotype to be a binary variable, in accordance with previous models (e.g., [25, 38–41]). We then consider the gradual evolution of a genetically controlled locus that determines whether a parent's phenotype affects the offspring's phenotype. In case selection does not favour parental influences on offspring phenotype determination, offspring phenotypes are considered to be genetically determined. By contrast, when an offspring's phenotype evolves to depend on the parental phenotype (i.e., parental effects evolve), it can either develop the same phenotype as that of its parent (i.e., stable epigenetic transmission) or develop a phenotype opposite to that of its parent (corresponding to a so-called negative parental effect, where parental epigenetic variants induce the opposite epigenetic variant in offspring). As the transmission of epigenetic variants is assumed to strictly depend on the parent's phenotype (as opposed to environmental cues), we a model of a cascading parental effect [42], in which a parental epigenetic variant may influence the epigenetic variant expressed in offspring and that of subsequent generations. This study then assesses how evolution shapes the transmission of these epigenetic variants in regimes of fecundity versus viability selection.

2 The model

The model assumes a spatially structured population that is divided into infinitely many patches [43], which are linked by juvenile dispersal. Analytical models of phenotypic transmission are necessarily involved, as they need to keep track of the distribution of phenotypes across different patches and different environments over time. Each patch has a fixed population size of n breeders and switches back and forth between two possible environmental states $\mathbf{e} = \{e_1, e_2\}$. For reasons of tractability we restrict the analysis to $n = 2$ breeders per patch. Stochastic individual-based simulations show, however, that much larger numbers of breeders per patch (e.g., $n = 25$) result in qualitatively similar differences between fecundity and viability selection (see Supplementary Figure S4). The model is described in Online Supplementary Information and the Supplementary Mathematica Sheet. Source code for the individual-based simulations is also available at `https://github.com/bramkuijper/fecundity_vs_viability`.

2.1 Phenotypes and inheritance

Individuals can adopt one of two possible phenotypes $\mathbf{z} = \{z_1, z_2\}$ making them locally adapted when their phenotype z_i matches the environmental state e_i of the local patch. Within this fluctuating environment, we then focus on the evolution of a genetically determined strategy (p_1, p_2), the elements of which determine the probability of producing a phenotype z_1 offspring by z_1 or z_2 parents respectively. When these phenotype determination probabilities evolve to be independent of the parental phenotype (i.e., $p_1 = p_2 \equiv p$) no parental influences evolve. In this case, offspring phenotypes

are considered to be either randomly determined when $0 < p < 1$ (bet-hedging, [44]) or genetically determined when either $p = 1$ (resulting in a monomorphic population with strict [genetic] inheritance of the z_1 phenotype) or $p = 0$ (resulting in a monomorphic population with strict [genetic] inheritance of the z_2 phenotype). However, when $p_1 \neq p_2$, the offspring's phenotype is a function of the parent's phenotype. In this case, strict genetic transmission of both phenotypes is possible when $p_1 = 1$ (z_1 parents always produce z_1 offspring and $p_2 = 0$ (z_2 parents always produce z_2 offspring). In all other cases where $p_1 \neq p_2$, however, we would have a scenario in which parents influence offspring phenotype determination with a lower fidelity of transmission, resembling scenarios in which offspring are influenced by their parental phenotype, for example through the transmission of epigenetic variants. The selectively favoured fidelity at which this transmission occurs is then investigated.

We employ an adaptive dynamics approach [45–47] to model the evolution of (p_1, p_2), assuming that evolutionary change occurs through the successive substitution of mutations that have small phenotypic effects. We assume a separation of the ecological and evolutionary timescales, implying that demographic changes (environmental change, deaths and births) occur at a much faster timescale than evolutionary change in the genetic modifiers that specify the probabilities of phenotypic inheritance.

Population dynamics: environmental change and breeder viability/fecundity The population dynamics of the model are described by a continuous-time dynamic, so that events occur one-at-a-time [48–50]. The following two events can occur: i) environmental change, where a patch in environmental state e_j switches to environmental state e_i with rate $s_{e_j \to e_i}$. When the environment changes, all adapted phenotypes become maladapted and all maladapted individuals become adapted. $s_{e_j \to e_i} = 0$ implies a constant environment, whereas $s_{e_j \to e_i} = \infty$ reflects an ever-changing environment. Importantly, $s_{e_j \to s_{e_i}}$ reflects the rate of change of an individual patch, rather than the global environment as a whole. Hence, at equilibrium, the total frequency of patches f_{e_i} and $f_{e_j} = 1 - f_{e_i}$ in state e_i and e_j is constant and given by $f_{e_i} = s_{e_j \to e_i} / (s_{e_j \to e_i} + s_{e_i \to e_j})$. Because we vary both $s_{e_1 \to e_2}$ and $s_{e_2 \to e_1}$ independently, we use the aggregate variable

$$\bar{s} = \frac{1}{2} \log_{10} [s_{e_1 \to e_2}] + \frac{1}{2} \log_{10} [s_{e_2 \to e_1}] \tag{1}$$

to reflect the global average rate of environmental change in the population [20].

ii) breeder mortality: adult breeders with phenotype z_i in an e_j environment have a mortality rate given by $0 < M_{z_i e_j} < \infty$ where a breeder maladapted to its local environment (i.e., $i \neq j$) has a higher mortality rate than an adapted breeder: $M_{z_i e_i} < M_{z_i e_j}$. Prior to the occurrence of mortality event, all adult breeders produce offspring, with phenotype z_i breeders living in environment e_j having relative fecundity $0 < B_{z_i e_j} < \infty$. The now vacant breeding position in an environment e_j is subsequently filled (maintaining a local population size of $n = 2$) with a juvenile offspring, as a result of competition among all locally born offspring remaining on their natal site and any immigrant offspring. Specifically, the probability that a newborn offspring born from any local adult breeder takes over the vacant patch is then given by $h \equiv \sum_k B_{kj}(1 - d) / \left[\sum_k B_{kj}(1 - d) + \bar{B}d\right]$. Here, B_{kj} is the fecundity of the kth adult breeder in the local e_j patch and $1 - d$ the probability that offspring remain at the natal patch. In addition, $\bar{B}d$ reflects the total number of immigrant offspring, where $\bar{B}$ is the average fecundity in the global environment and d the juvenile dispersal probability. Consequently, in

case of a breeder mortality event, viability selection occurs first (through differential breeder mortality), followed by fecundity selection to determine the parent which will provide offspring that fills the vacant breeding position.

To give a brief example of how fecundity and viability selection enter our model (reflected by the $B_{..}$ and $M_{..}$ terms respectively), here we provide the rate of change $\Delta f_{(e_i,n_a+1)\to(e_i,n_a)}$ with which patches in state (e_i, n_a+1) (i.e., environmental state e_i and containing n_a+1 adapted breeders) become patches in state (e_i, n_a) in the next time step (same environmental state but containing n_a adapted breeders). This reduction in the number of adapted breeders from n_a+1 to n_a while the environmental state remains the same can only occur through mortality of an adapted breeder (at rate $M_{z_i e_i}$) and its subsequent replacement by a maladapted juvenile. Hence, the rate of change is given by

$$\Delta f_{(e_i,n_a+1)\to(e_i,n_a)} = f\left(e_i, n_a+1\right)\left(n_a+1\right) M_{z_i e_i}\left[l_m\left(n_a+1, e_i\right) + \tilde{l}_m\left(n_a+1, e_i\right)\right], \quad (2)$$

where $f(e_i, n_a+1)$ reflects the current frequency of patches in state e_i containing n_a+1 adapted breeders and the mortality rate of n_a+1 adapted breeders is given by $(n_a+1)M_{z_i e_i}$. The probability with which a maladapted juvenile subsequently claims the vacant breeding position is given by $l_m(n_a+1, e_i)$ when this juvenile is born in the local patch and $\tilde{l}_m\left(n_a+1, e_i\right)$ when this juvenile is born in the remote patch. An expression for $l_m(n_a+1, e_i)$ is given below and in eq. (S4), while an expression for $\tilde{l}_m\left(n_a+1, e_i\right)$ can be derived in the same fashion and is provided in the online supplement (see eq. [S8]):

$$l_m\left(n_a+1, e_i\right) = (1-d)\,\frac{\left(n_a+1\right) B_{z_i e_i}\pi_{z_i\to z_j} + \left(n-n_a-1\right) B_{z_j e_i}\pi_{z_j\to z_j}}{(1-d)\left[n_a B_{z_i e_i} + \left(n-n_a\right) n_a B_{z_j e_i}\right] + d\bar{B}}. \quad (3)$$

With probability $1-d$ a newborn stays at the local site. This newborn is either born from one of the n_a+1 adapted breeders (note that births are assumed to occur prior to mortality) at rate $B_{z_i e_i}$, or from one of the $n-n_a-1$ maladapted breeders at rate $B_{z_j e_i}$. When born from an adapted parent, the newborn inherits a maladapted phenotype (rather than the parent's adapted phenotype) with probability $\pi_{z_i\to z_j}$ (where $z_i \neq z_j$). When born from a maladapted parent, the newborn inherits the parental maladapted phenotype with probability $\pi_{z_j\to z_j}$ (see eqns. [S5a-S5d]). Finally, the denominator reflects the total number of juveniles competing for the breeding position, $(1-d)[n_a B_{z_i e_i} + (n-n_a)\, n_a B_{z_j e_i}]$ of which are locally born, while $d\bar{B}$ are remotely born (see eq. [S1]). The total rate of change in frequency of patch in state (n_a, e_i) is given in eq. (S9).

Evolutionary dynamics We model evolution in the two phenotype determination strategies p_1, p_2, by considering the successive invasion of rare mutants that slightly differ in one of these three traits from the otherwise monomorphic resident population. In the Supplementary Mathematica Notebook, we derive an expression of the instantaneous change in fitness $\mathrm{d}W\left(p_i^{\mathrm{mut}} \mid x, \hat{n}_a, e_i\right)/\mathrm{d}t$ of an adult focal mutant who has phenotype $x \in \{z_1, z_2\}$, lives in environment e_i and shares the patch with $\hat{n}_a$ other individuals that are adapted (and with $n-\hat{n}_a-1$ other maladapted individuals). This focal mutant uses a slightly different mutant strategy $p_i^{\mathrm{mut}} = p_i + \delta p_i$ than the rest of the population, which uses the resident strategy p_i. Because mutants can have different reproductive values dependent on their local environment, their phenotype and

the phenotypes of their neighbours, these reproductive values must be taken into account in the calculation of the mutant's invasion fitness [51]. To this end, each of the fitness effects are weighed by the respective gains and losses in terms of reproductive value of the mutant allele [e.g., 50–54].

In order to arrive at an expression of evolutionary change, the average instantaneous change ΔW in fitness due to actions of the focal mutant in any of the different states that it can attain (i.e., in terms of its own state x, the number of adapted (and maladapted) individuals it has as neighbours $\hat{n}_a$ and the local state of the environment e_i). We then have

$$\frac{\mathrm{d}W\left(p_i^{\mathrm{mut}}\right)}{\mathrm{d}t} = \sum_{n_a=0}^{2} \sum_{e_i=\{e_1,e_2\}} f(n_a, e_i) \left[\frac{n_a}{n} \frac{\mathrm{d}W\left(p_i^{\mathrm{mut}} \mid x = a, n_a - 1, e_i\right)}{\mathrm{d}t} + \frac{n - n_a}{n} \frac{\mathrm{d}W\left(p_i^{\mathrm{mut}} \mid x = m, n_a, e_i\right)}{\mathrm{d}t} \right],$$

where $f(n_a, e_i)$ is the frequency of patches containing n_a adapted individuals in environmental state e_i and the fractions n_a/n and $(n - n_a)/n$ reflect the probabilities that the focal mutant is locally adapted or maladapted respectively.

If evolution proceeds slowly, so that an individual's lifespan represents only an infinitesimal fraction of evolutionary time, a standard result in adaptive dynamics [e.g., 55] allows one to describe changes in character values over time using the following evolutionary dynamic (e.g., see eq. C5 in 53)

$$\frac{\mathrm{d}}{\mathrm{d}t} \begin{bmatrix} p_1 \\ p_2 \end{bmatrix} = (\text{positive constant}) \times \left[\begin{array}{c} \frac{\partial}{\partial p_1^{\mathrm{mut}}} \frac{\mathrm{d}W\left(p_1^{\mathrm{mut}}\right)}{\mathrm{d}t} \\ \frac{\partial}{\partial p_2^{\mathrm{mut}}} \frac{\mathrm{d}W\left(p_2^{\mathrm{mut}}\right)}{\mathrm{d}t} \end{array} \right] \Bigg|_{p_1^{\mathrm{mut}}=p_1, p_2^{\mathrm{mut}}=p_2}, \tag{4}$$

where we assume that any pleiotropic mutations are absent. We then find evolutionary endpoints (which reflect candidate evolutionarily stable strategies) by iterating the adaptive dynamic in (4) starting from a particular set of values $[p_1, p_2]_{t=0}$ until it vanishes, $\frac{\mathrm{d}}{\mathrm{d}t}[p_1, p_2]^{\mathrm{T}} = \mathbf{0}$. During each time step of the iteration, we numerically solved for equilibrium values of patch type frequencies, reproductive values and relatedness for the current values of $[p_1, p_2]_t$ using a root finding algorithm written in C. Convergence was determined when the largest difference in values of $[p_1, p_2]$ between consecutive time steps was $\leq 10^{-7}$. Starting values used in our iterations are $[p_1, p_2]_{t=0} = [0.5, 0.5]$. The outcomes obtained from these numerical iterations are convergence stable by definition, and individual-based simulations revealed that values are also evolutionarily stable.

3 Results

We study the fidelity of phenotypic transmission by evolving the strategy vector (p_1, p_2), which specifies the proportion of z_1-offspring produced by a parent with phenotype z_1 or z_2 respectively. To recap, in case $p_1 = p_2$ offspring phenotypes are determined independently of a parent's phenotype (no parental effects). When $p_1 \neq p_2$ however, the offspring phenotype is a function of the parent's phenotype. Figure 1 illustrates how fecundity and viability selection impact on the prevalence of parental effects, for

an example case where juvenile dispersal is limited ($d = 0.1$) and the rate of environmental change is slow ($\bar{s} = -1.4$, see eq. [1]). Similar to previous analyses [20, 25, 26], we find that there are substantial regions where parental effects do not evolve at all (i.e., $p_1 = p_2$, white areas in Figure 1), particularly when one environment is much more common than the other. Here, parents of both phenotypes only produce the phenotype z_i that matches the most prevalent environment e_i, eventually resulting in a monomorphism of the phenotype z_i.

By contrast, when the frequencies of the two environments are more similar (i.e., around the middle in each panel), we find that $p_1 \neq p_2$, so that offspring phenotype determination now depends on the parental phenotype. Interestingly, the scope for parental effects appears to be much larger in regimes of viability selection relative to regimes of fecundity selection. Moreover, this difference between viability selection and fecundity selection appears to be relatively robust to larger number of breeders per patch n, where parental effects evolve in contexts of viability selection (but not in contexts of fecundity selection) up to $n = 50$ (Figure S4). For larger n, populations become essentially well-mixed in which case there are no differences between fecundity selection and viability selection (Figure S4).

General ecological conditions favouring phenotypic memory Next, we assess whether this differential effect of fecundity and viability selection is robust to changes in juvenile dispersal d or the average rate of environmental change $\bar{s}$. Figure 2 depicts the expected number of consecutive generations over which phenotype z_1 is inherited from parent to offspring, while (i) varying the frequencies of the two environments, (ii) the average rate of environmental change and (iii) the rate of dispersal (the duration of inheritance of phenotype z_2 is a mirror image of Figure 2, see Figure S2).

Similar to Figure 1, Figure 2 shows that when one environment predominates (dark red regions at the left or right hand sides of each panel), no parental effects evolve regardless of the presence of fecundity or viability selection. Rather, populations are monomorphic for the phenotype favoured in the most prevalent environment (z_1 when e_2 is rare, z_2 when e_2 is common), resulting in a monomorphism and long-term (genetic) inheritance.

Only when both environments are encountered at roughly similar rates (middle of each panel in Figure 2), do we find that the parental effects evolve: here, the inheritance of a phenotypic variant spans a limited number of generations, characteristic of what is observed for many epigenetic variants. However, comparing Figures 2A-D with Figures 2E-H shows that such short-term transmission of epigenetic variants is most commonly encountered in populations experiencing viability selection. By contrast, in populations which experience fecundity selection, this only evolves when environmental variation is large (i.e., when populations encounter both environments at almost exactly the same rate).

Next, Figure 2 shows that intermediate durations of phenotypic transmission are more likely to evolve in populations with limited dispersal, corroborating results from previous studies [e.g., 17, 20, 56], albeit with two key exceptions. First, we find that the positive effect of limited dispersal on parental effects is much weaker in populations experiencing fecundity selection. Second, in populations experiencing viability selection, we find a limited number of cases where parental effects even evolve when all juveniles disperse to (random) remote patches ($d = 1$, Figure 2D). To understand this surprising result, Supplementary Figure S2 shows that viability selection favours

a global mixture of z_1 and z_2 offspring, which can be achieved through many alternative combinations of p_1 and p_2 (i.e., a line of equilibria), where typically $p_1 \neq p_2$ thus resulting in parental effects. By contrast, in populations with fecundity selection and $d = 1$, a monomorphism of the phenotype that matches the commonest environment is always selectively favoured, selecting against any parental effects.

Interactions between fecundity and viability selection Finally, Figure 3 explores the evolution of phenotypic transmission when populations experience fecundity and viability selection simultaneously. In populations where viability selection prevails, increasing the strength of fecundity selection has little effect (Figure 3A). If anything, increasing levels of fecundity selection seem to enhance rather than inhibit the evolution of parental effects, at least when viability selection is already present. In populations where fecundity selection prevails (Figure 3B), increasing the effect of viability selection substantially increases the scope for parental effects. Hence, fecundity selection has little effect in populations which also experience viability selection, unless viability selection is weak. A more general overview of the interaction between fecundity and viability selection is given in Figure S3.

4 Discussion

The current analysis shows that the mode of selection matters to the evolution of phenotypic inheritance: in spatially structured populations that experience fecundity selection, high-fidelity inheritance that mimics that of genetic systems is favoured with little scope for parental effects. By contrast, in populations which experience viability selection, the evolutionary scope for parental effects and phenotypic inheritance of limited duration is substantially larger. Hence, life-history differences that affect the influence of fecundity versus viability selection may play an important role in the evolution of parental effects and phenotypic memory.

Why do we predict that viability and fecundity selection selection have these different consequences? Essential for the evolution of parental effects is that the parental phenotype evolves to be a reliable cue for offspring phenotype determination, which requires that the parental phenotype is correlated with the local environment [15, 18, 57]. Because viability selection causes locally adapted breeders to survive for longer than locally maladapted breeders, a breeder is more likely than not to have a phenotype matching the local environment, simply by being alive [58]. In other words, a correlation between phenotype and environment immediately builds up within the breeder's lifetime, because survival selection results in the enrichment of patches with phenotypes matching the local environment. This enrichment of locally adapted phenotypes is illustrated in Figure S5A: even when the distribution of offspring phenotypes would initially be random (i.e., when $p_1 = p_2 = 0.5$), viability selection causes each environment to become enriched with locally adapted phenotypes. Hence, in regimes of viability selection, a parental phenotype is informative about its local environment (as demonstrated by a large covariance between the parental phenotype and its environment (Figure S6A), selectively favouring a parent to transmit that phenotype to the next generation.

In regimes of fecundity selection, however, mortality rates of locally adapted and maladapted breeders are identical (because selection acts on the number of offspring

produced rather than on survival). Hence, simply being alive is not informative about whether a breeder's phenotype matches the local environment. A correlation between phenotypes and the local environment still develops, however, when two conditions are met: (i) more fecund, locally adapted parents produce most of the successfully established offspring in the local patch and (ii) the phenotypes of these successfully established offspring are correlated to the phenotypes of their parents. Although requirement (i) is a logical consequence of fecundity selection, note that correlations now only develop in the next generation (i.e., after successful offspring establishment), which is slower than in regimes of viability selection where correlations arise due to differential survival within one and the same generation. Hence, when selection acts on fecundity, it takes longer for correlations between phenotype and the local environment to build up again after the local environment has changed, as it requires breeder mortality and establishment by non-dispersing offspring (most likely from locally adapted parents) before enrichment of phenotypes to their matching environments occurs. By contrast, when selection acts on viability, differential breeder mortality is already sufficient to result in enrichment of phenotypes to their matching environments. Finally, requirement (ii) itself requires some fidelity of inheritance beforehand, so that parental effects should already have evolved in the first place. Indeed, Figure S5B shows that when offspring have their phenotype randomly assigned, there is no parameter space in which both phenotypes are associated with their respective environments simultaneously in regimes of fecundity selection. By contrast, when there is some inheritance fidelity, a correlation develops, but it is still substantially weaker than in regimes of viability selection (see Figure S6B). Hence, in regimes of fecundity selection, parental phenotypes are less likely to be informative cues about their local environment, thus narrowing the scope for their transmission to offspring.

The prediction that inheritance fidelity depends on the mode of selection has a number of interesting consequences for the current study of inheritance systems [e.g., 1, 59–62]. First, our study would predict that the scope for extended inheritance is generally weaker in organisms with slow life histories (see also [63] for similar comments), which are often (but not always) characterised by weaker viability selection relative to fecundity selection during adult life [64–66]. Second, our model would predict stronger parental effects on those life-history traits which more strongly affect survival (such as predator defence traits that do not involve nest defence, e.g., [67]) versus traits that may have a stronger impact on fecundity (e.g., nest defence, onset of reproductive timing). Ideally, the strength of fecundity and viability selection should be separately measured (which is now facilitated by accumulating demographic data [68, 69]) when making inferences about how local adaptation shapes parental effects and the scope for nongenetic inheritance. Third, although our model is asexual, we would predict that similar results would hold for sexual populations (results not shown). In this case, sex differences in the degree of viability vs fecundity selection may affect the relative strength of paternal vs maternal effects. In the context of our current model, selection among males for fertilisations of female gametes can be equated with fecundity selection. Consequently, this would mean that traits involved in intrasexual selection – measured as competition among males for fertilisations – may well favour weaker paternal effects relative to traits related to survival. However, an explicit formal model of cascading paternal versus maternal effects in the context of sexual selection would be required to confirm this prediction.

How to test the predicted relationship between parental effects and viability versus

fecundity selection? One method of choice would be to experimentally evolve populations in fluctuating environments, where one contrasts lines that experience fluctuating selection on early fecundity with lines that experience fluctuating selection on longevity (e.g., through sampling offspring from individuals in later life). Experimental evolution in regimes that favour either fecundity or viability has been previously done in *Caenorhabditis* nematodes [e.g., 70]. Moreover, there is now accumulating evidence that parental effects can rapidly evolve in *Caenorhabditis* nematodes [19, 71], making this an ideal model system to assess the evolution of parental effects in different life-history contexts [72]. A key caveat is, however, that these experimental evolution studies have been performed on well-mixed populations, whereas our study suggests that a difference between viability and fecundity selection may only arise in spatially structured populations with limited dispersal and relatively small deme sizes. It would thus be interesting to consider the experimental evolution of transgenerational effects in such contexts, as has been done in *Caenorhabditis* within the context of local adaptation [73, 74].

Our model highlights that differences in the type of selection acting on the population may be much more important to the evolution of parental effects than currently anticipated, yet there is substantial scope for future improvements. Foremost, our model focuses on the evolution of 'cascading' parental effects [42], where offspring phenotype determination depends on cues about the parental phenotype, rather than on more direct cues about the parental environment (environmental maternal effects [75]), or cues about the parent's genes (maternal genetic effects). In contrast to the current model, we would expect that the difference between fecundity and viability selection is unlikely to be important for environmental maternal effects. This is because offspring now receive a direct cue about the maternal environment, hence the development of a correlation between the maternal phenotype and the local environment through viability or fecundity selection becomes irrelevant. However, in the context of maternal genetic effects, we would expect differences between fecundity and viability selection to be similarly important as in the current model. This is because the maternal genotype only becomes informative to offspring when it correlates to the local environment (through differential survival of its bearers). As the current paper shows, viability selection has a much higher efficacy in developing such correlations. It would be welcome to expose the different types of parental effects to a systematic evolutionary analysis across different selective contexts: a welcome first step in this direction was undertaken recently by [16] who compared both maternal genetic and maternal environmental effects in the context of viability selection.

Another assumption of the current analysis is that phenotypes are binary (z_1 and z_2), thus reflecting those traits which are dimorphic rather than continuous [76]. It would be interesting to extend the current analysis to the evolution of parental effects on continuous traits, as done by quantitative genetics modelling when populations are well-mixed [e.g., 12, 18] or are characterised by very high numbers of breeders per patch [56]. However, given previous findings that binary and continuous models of parental effects often result in qualitatively similar outcomes (e.g., increased importance of maternal effects when dispersal is limited and when environments are autocorrelated), we would expect that the current conclusions are robust to contexts where traits are continuous rather than discrete.

Finally, for the sake of comparison with previous analyses on the evolution of parental effects [e.g., 15–17], we have focused on a simple trait that is under stabilising

selection in a spatiotemporally varying environment. Future models should consider, however, the evolution of these parental effects in the face of explicit life-history trade-offs, such as between offspring size and number [77, 78], between reproductive effort and survival [79, 80] or between all these traits [81]. Existing theoretical predictions on offspring size versus number decisions have been highly valuable (reviewed in [82]), yet these analyses only consider offspring phenotypes up and until juvenile survival (e.g., [77, 83, 84], but see [85]). By contrast, a more rigorous analysis of parental effects would consider whether a maternal decision about sizes/numbers of her young subsequently affects those same decisions when made by her offspring and by later descendants [86]. Consequently, future analyses are needed that track the evolution of parental effects from life-history traits in parents to life-history traits in offspring. Particularly welcome would be a comparison between the evolution of parental effects which affect offspring reproductive effort versus survival in adulthood (viability selection) and parental effects that affect offspring size versus number decisions (fecundity selection).

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5 Figure captions

Figure 1 Parental effects (grey areas where $p_1 \neq p_2$) are much more likely to occur in regimes of viability selection (panel A) than fecundity selection (panel B). Parameters: $M_a = 1$, $M_m = 2$, $B_a = B_m = 1$ (panel A), $M_a = M_m = 1$, $B_a = 2$, $B_m = 1$ (panel B), $d = 0.1$, $\bar{s} = -1.4$.

Figure 2 Viability selection (panels A-D) is much more likely to lead to intermediate fidelities of inheritance (characteristic of parental effects) of phenotype z_1 (orange to blue coloured areas) than fecundity selection (panels E-H). Dark-red areas indicate high-fidelity inheritance of phenotype z_1, which occurs in populations monomorphic for z_1, implying that parental effects are absent. Parameters: $M_a = 1$, $M_m = 2$, $B_a = B_m = 1$ (panels A-D), $M_a = M_m = 1$, $B_a = 2$, $B_m = 1$ (panels E-H), $n = 2$. See Figure S1 for the inheritance of phenotype z_2, which is a mirror image of the current figure.

Figure 3 The evolution of parental effects (i.e., $p_1 \neq p_2$: where dotted and solid lines do not overlap) in populations experiencing different combinations of viability selection and fecundity selection. Panel A: populations experience a constant, high level of viability selection $M_\mathrm{m} = 2 : M_\mathrm{a} = 1$ (viability selection on maladapted:adapted breeders) and varying levels of fecundity selection $B_\mathrm{m} = 1 : B_\mathrm{a}$ varies (fecundity selection on maladapted:adapted breeders). When combined with viability selection, fecundity selection slightly enhances (rather than inhibits) the scope for parental effects. Panel B: populations experience a constant, high level of fecundity selection and varying levels of viability selection. The scope for parental effects increases with ever increasing levels of mortality selection. Parameters as in Figure 1.

6 Figures

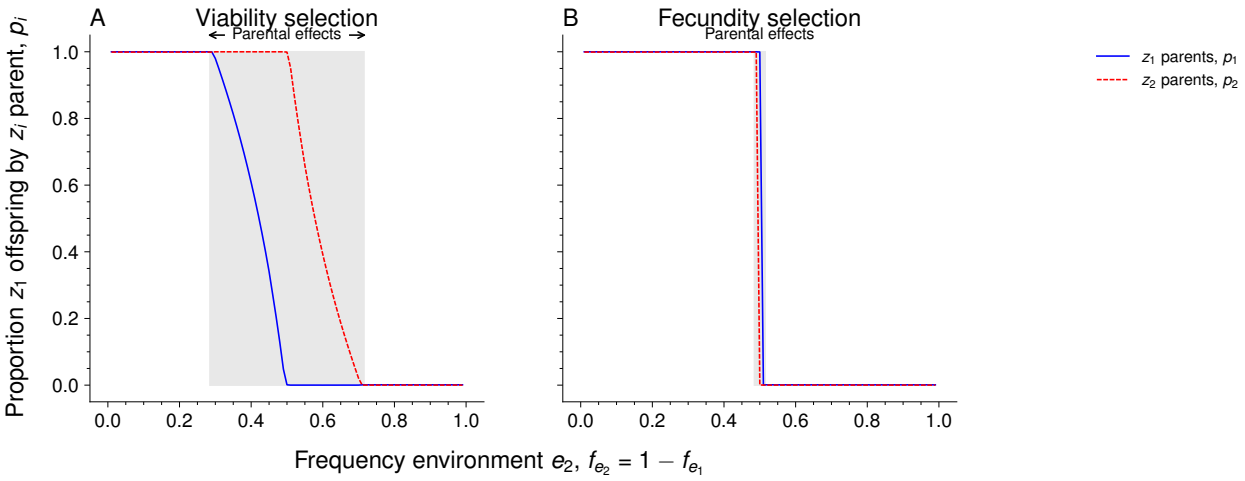
A
Viability selection
← Parental effects →
B
Fecundity selection
Parental effects
Proportion z_1 offspring by z_i parent, p_i
1.0
0.8
0.6
0.4
0.2
0.0
0.0
0.2
0.4
0.6
0.8
1.0
Frequency environment e_2, $f_{e_2} = 1 - f_{e_1}$
z_1 parents, p_1
z_2 parents, p_2

Figure 1:

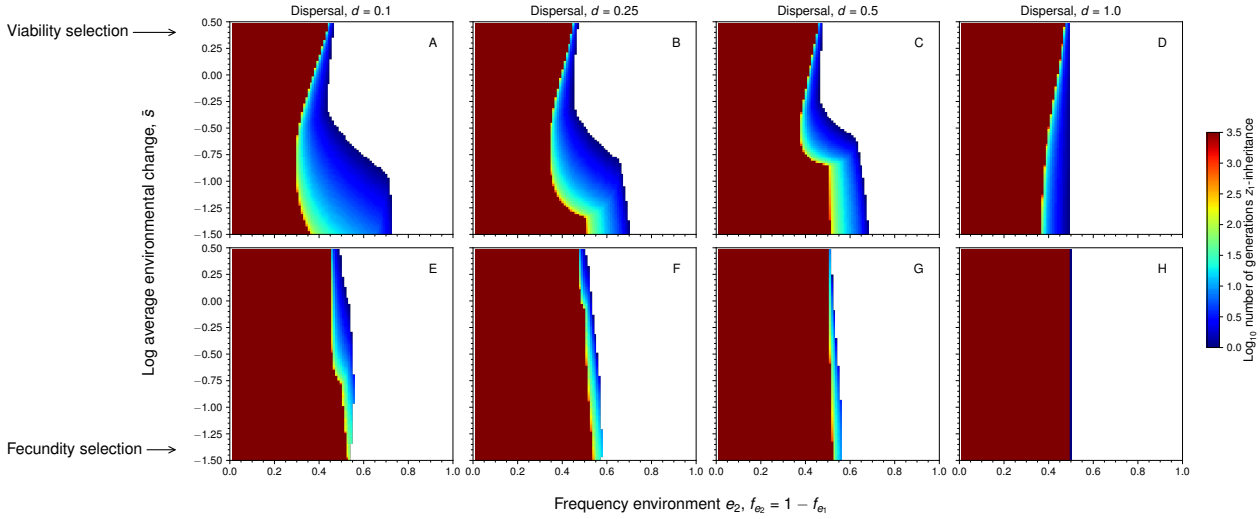
Dispersal, d = 0.1
Dispersal, d = 0.25
Dispersal, d = 0.5
Dispersal, d = 1.0
Viability selection
Fecundity selection
A
B
C
D
E
F
G
H
Log average environmental change, s̄
Frequency environment e2, fe2 = 1 − fe1
Log10 number of generations z1-inheritance

Figure 2:

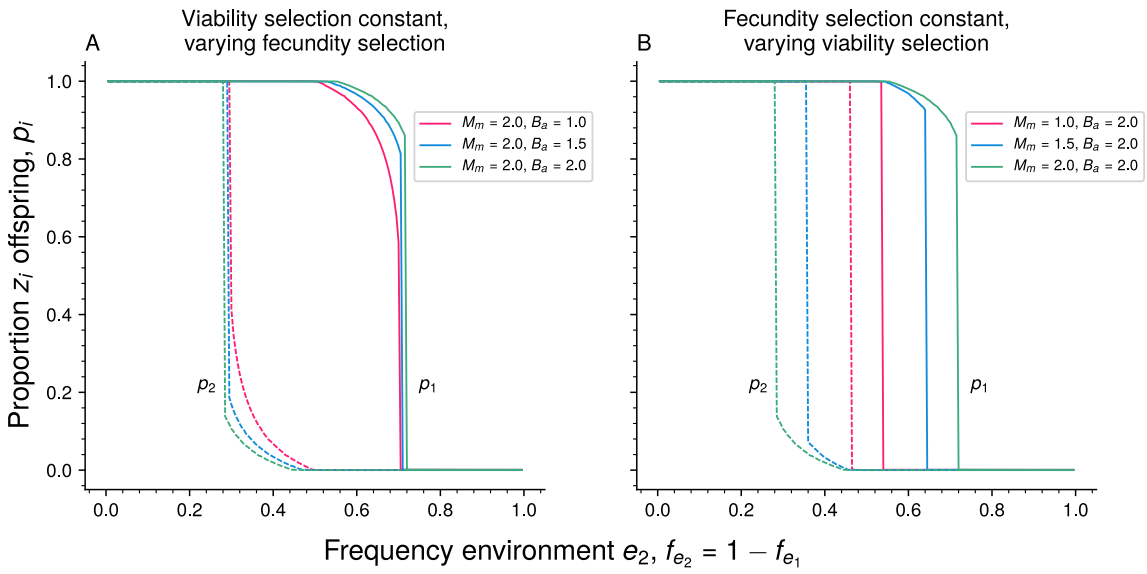
A
Viability selection constant,
varying fecundity selection
B
Fecundity selection constant,
varying viability selection
Proportion z_i offspring, p_i
Frequency environment e_2, $f_{e_2} = 1 - f_{e_1}$
M_m = 2.0, B_a = 1.0
M_m = 2.0, B_a = 1.5
M_m = 2.0, B_a = 2.0
M_m = 1.0, B_a = 2.0
M_m = 1.5, B_a = 2.0
M_m = 2.0, B_a = 2.0
p_2
p_1
0.0
0.2
0.4
0.6
0.8
1.0

Figure 3: