Transgenerational plasticity and bet-hedging: a framework for reaction norm evolution.

Jens Joschinski[1*], Dries Bonte[1]

[1] Terrestrial Ecology Unit (TEREC), Department of Biology, Ghent University, Ghent, Belgium.

*** Correspondence:**
Jens Joschinski
Jens.Joschinski@ugent.be

Keywords: Phenotypic plasticity, Trans-generational plasticity, Bet-hedging, Coin-flipping, GxE, Canalization, Adaptation, Climate change

Abstract

Decision-making under uncertain conditions favors bet-hedging (avoidance of fitness variance), whereas predictable environments favor phenotypic plasticity. However, entirely predictable or entirely unpredictable conditions are rarely found in nature. Intermediate strategies are required when the time lag between information sensing and phenotype induction is large (e.g. transgenerational plasticity) and when cues are only partially predictive of future conditions. Nevertheless, current theory regards plasticity and bet-hedging as distinct entities. We here develop a unifying framework: based on traits with binary outcomes like seed germination or diapause incidence we clarify that diversified bet-hedging (risk-spreading among one's offspring) and transgenerational plasticity are mutually exclusive strategies, arising from opposing changes in reaction norms (allocating phenotypic variance among or within environments). We further explain the relationship of this continuum with arithmetic mean optimization vs. conservative bet-hedging (a risk-avoidance strategy), and canalization vs. phenotypic variance as a three-dimensional continuum of reaction norm evolution. We discuss under which scenarios costs and limits may constrain the evolution of reaction norm shapes.

1 Introduction

Changing conditions can promote evolutionary change in various ways (Botero et al., 2015; Tufto, 2015). One commonly envisioned mode of evolution is the continuous change of trait means as result of changing mean conditions (Darwin, 1859). Yet, although trait changes in response to novel conditions are widely observed (e.g. due to climate change, Piao et al., 2019), they frequently result from phenotypic plasticity (Boutin and Lane, 2014), i.e. changes of the phenotype in response to an environmental cue. Phenotypic plasticity may provide a short-term relief from changing conditions (Charmantier et al., 2008; Chevin et al., 2010), but also shield a genotype from selection and thereby prevent evolution (Oostra et al., 2018), or it may facilitate evolution via genetic accommodation (Kelly, 2019). In any case, phenotypic plasticity is a pervasive evolutionary strategy, and considered a major factor in a rapidly changing climate (Fox et al., 2019).

The time scale of phenotypic plasticity depends on the time scale of environmental fluctuation (Rando and Verstrepen, 2007; Stomp et al., 2008). Fluctuations over very rapid timescales can be addressed by reversible plasticity, which includes, for example, the induction of plant defense when herbivores are present (Green and Ryan, 1972). Gradual long-term changes, on the other hand, are addressed by genetic adaptation. Between those extremes lie environmental fluctuations that are roughly on the scale of one life span. When environments change over the course of an organism's development, they can be tackled by irreversible developmental plasticity, i.e. plastic adjustment of developmental pathways that lead to alternative phenotypes (Botero et al., 2015). For example, some *Daphnia* can produce protective phenotypes when chemical cues from predators are sensed during development (Krueger and Dodson, 1981). When environments are constant throughout an organism's life time but change from one generation to the next, phenotypic change can be induced in the offspring generation. These are referred to as anticipatory parental effects (Burgess and Marshall, 2014) or intergenerational inheritance (Perez and Lehner, 2019). For example, aphids that live under crowded conditions may produce winged offspring that can leave the colony and avoid high predation pressure or plant deterioration (Braendle et al., 2006). Lastly, when environmental fluctuations last for several generations, epigenetic modifications may be integrated into the germ line and affect multiple succeeding generations. This is referred to as transgenerational plasticity or non-genetic inheritance (Perez and Lehner, 2019; Adrian-Kalchhauser et al., 2020). For the remainder of the article we will refer to all these irreversible changes simply as phenotypic plasticity, ignoring the potential physiological constrains that may limit their evolution. They all have in common that there is a long delay between information sensing and phenotype induction.

Although often assumed, phenotypic plasticity does not need to be adaptive (Ghalambor et al., 2007; Arnold et al., 2019). Plasticity requires some environmental cue on which the induction of phenotypic change is based, and uncertainty around the future environmental state may turn plasticity maladaptive (Burgess and Marshall, 2014; Donelson et al., 2018). Such unpredictable conditions instead favor bet-hedging strategies, which refer to the reduction of fitness variance (Cohen, 1966; Seger and Brockmann, 1987; Starrfelt and Kokko, 2012). This can be achieved by avoiding risky investments (conservative bet-hedging), or by spreading the risk among one's offspring (diversified bet-hedging), i.e. producing offspring with varying phenotypes (Seger and Brockmann, 1987; Starrfelt and Kokko, 2012). Although empirical evidence is difficult to obtain (Simons, 2011), bet-hedging is a likely explanation for high trait variance or unexpected trait means in many systems, such as the seed dormancy of desert annuals (Cohen, 1966), diapausing strategies of insects (Hopper, 1999) and annual killifish (Furness et al., 2015), wing dimorphisms (Grantham et al., 2016), and the evolution of facultative sexual reproduction (Gerber and Kokko, 2018), dispersal and partial migration (Goossens et al., 2020).

At fluctuations of intermediate time scales where there is a delay between information sensing and phenotype induction, both phenotypic plasticity (e.g. Baker et al., 2019) and bet-hedging (e.g. Venable, 2007) may be expected to evolve. Various theoretical studies have clarified the conditions that may lead to one or the other (Botero et al., 2015; Tufto, 2015), but although occurring potentially simultaneously, bet-hedging and plasticity are nevertheless often treated independently (Donelson et al., 2018). Moreover, when diversified bet-hedging and plasticity are considered jointly, there is no clear consensus about their exact relationship. Adaptive offspring variance that is needed for diversified bet-hedging might be either established by developmental instability (Simons and Johnston, 1997; Kærn et al., 2005; Veening et al., 2008; Woods, 2014; Dueck et al., 2016; Perrin, 2016) or by overly relying on cues with little predictive power ("microplasticity", Simons and Johnston, 2006; "hyperplasticity", Scheiner and Holt, 2012). With this article we aim to clarify the relationship between bet-hedging and plasticity, with special attention to readers that are familiar with plasticity but less familiar with bet-hedging theory. We will first use one simple numerical example (insect diapause) to explain the relationship of diversified bet-hedging, conservative bet-hedging and arithmetic mean optimization in detail. We will then extend the consideration to a range of environments whose state is partially predictable, thereby adding the potential for phenotypic plasticity. Lastly, we generalize from our example and describe a method to quantify phenotypic plasticity and bet-hedging based on reaction norm shapes.

2 An example

Common examples of bet-hedging are trans-generational biphenisms, i.e. the parent decides among two possible physiological states of the offspring in the face of uncertainty (e.g. Cohen, 1966; Grantham et al., 2016; Maxwell and Magwene, 2017; see Simons, 2011 for further examples). One of these examples is the timing of insect diapause (Halkett et al., 2004; Pélisson et al., 2013), which we will use to illustrate the theory throughout this article.

Multivoltine insects benefit from exponential population growth throughout the growing season, but need to produce an overwintering (diapausing) generation before the onset of cold weather (Kivela et al., 2016). Aphids, for example, reproduce by parthenogenesis during summer, which enables particularly quick population growth; in autumn they invest in sexual offspring that produce diapausing eggs, as frost kills the soft-bodied insects and only eggs survive (Simon et al., 2002). The struggle to keep the growing season long on one hand and to avoid death on the other hand puts diapause timing under intense selection pressure. If the onset of frost would be invariant, day length could be used as reliable cue of impeding winter, so plasticity to day length is expected to evolve. However, if just one generation faces early frosts, all offspring may simultaneously die and the genotype is driven to extinction, regardless of their otherwise high growth rates. Under unpredictable or only partially predictable conditions, bet-hedging strategies may therefore be expected to evolve (Halkett et al., 2004).

3 Arithmetic mean optimization, diversified bet-hedging and conservative bet-hedging

We wish to use the diapause example to explain the bet-hedging concept in detail with a few numerical examples. We first consider an entirely unpredictable environment, in which an aphid mother cannot collect any information about the potential environment of their offspring i.e. there is a 50% chance that the offspring will face beneficial summer conditions (E_1), but also a 50% chance for harsh winter conditions (E_2). Let us further assume that parthenogenetic offspring (P_1) have a fitness value of 4 in arbitrary units in E_1 (summer), but only 0.1 in E_2 (winter), whereas diapausing offspring (P_2) have 1 fitness regardless of environmental conditions. A genotype that only invests in parthenogenesis (P_1) maximizes the arithmetic mean fitness and achieves on average 2.05 fitness, which is twice as much as a genotype that invests exclusively in diapause (P_2) (table 1). Nevertheless, the latter strategy (risk-aversion) is more successful on the long term, because the former nearly dies out every two years. The arithmetic mean obviously fails as predictor of long-term population growth. If there are multiple decisions to make and the outcome is multiplicative, such as for population growth over multiple years, the geometric mean is a much better predictor, because it is sensitive to variance among years (Cohen, 1966; Seger and Brockmann, 1987; Starrfelt and Kokko, 2012). It correctly shows that the strategy of investing exclusively in diapause is superior to investing exclusively in parthenogenesis, because the lower arithmetic mean fitness is more than compensated by the reduction in fitness variance. The risk-aversive strategy of investing in lower fitness fluctuation at the cost of arithmetic mean fitness is called conservative bet-hedging (CBH), akin to investing in gold when stock markets fluctuate.

Now let us consider a genotype with high developmental instability, i.e. whose offspring phenotype is randomly determined. Investing equally in both phenotypes (P_1 and P_2) breaks down the fitness correlation among the offspring, as half of the offspring takes a risk, while the other half plays it safe (Starrfelt and Kokko, 2012). This means that the arithmetic mean fitness is not reduced as strongly as that of the risk-aversive phenotype (100% P_2), but the fitness fluctuation between E_1 and E_2 (2.5 vs 0.55) is also not as great as that of the arithmetic mean optimizer (100% P_1). This strategy is similar to investing in a portfolio of stocks rather than a single stock and is called diversified bet-hedging (DBH).

By definition, risk aversion strategies can only be equated with conservative bet-hedging (CBH), and developmental instability only with diversified bet-hedging (DBH), when they increase geometric mean fitness; and both bet-hedging strategies require that arithmetic mean fitness is reduced. In line with these definitions we will refer to different phenotype proportions as risk-aversion, developmental instability and arithmetic mean optimization, and reserve the terms CBH and DBH for the case that these proportions increase geometric mean fitness. In summary, a genotype may maximize arithmetic mean fitness (100% P_1), reduce individual fitness variance by risk aversion (100% P_2), or reduce the fitness correlation among its offspring through developmental instability (50 % each).

The geometric mean can be calculated for any phenotype proportion p between 0 and 100% (Fig.1, blue line), showing that actually neither of the three strategies (AMO, CBH, DBH) is optimal. Instead, a phenotype proportion of .61 yields the highest geometric mean fitness (Table 1). The same principles also apply when the frequency of E_1 and E_2 is not 0.5. For example, when the frequency of E_2 (winter) is only 20%, the optimal proportion of P_2 is 0.17 (Fig. 1, orange line). Geometric mean fitness thus changes along a gradient ranging from arithmetic mean optimization (p = 0) over developmental instability (p =0.5) to risk aversion (p=1), and the optimal strategy accordingly may range from AMO over DBH to CBH. The more seasoned reader of bet-hedging literature will notice that this description of a gradient appears to contrast with the view of Starrfelt and Kokko (2012), who see fitness optimization as a three-way trade-off between AMO, CBH and DBH rather than a linear gradient. We will clarify the apparent contradiction in box 1.

4 Calculating optimal reaction norm shapes

We so far discussed the optimal phenotype proportion in a single, isolated environment. However, the benefit of diapause lies in adapting to a continually changing environment. Like in many other insects, aphid diapause is mainly governed by day length. Aphids exclusively reproduce by parthenogenesis under long-day conditions, but transition to the production of sexual forms under long-night conditions (Marcovitch, 1923). The diapause decision can hence be visualized as a biphenic reaction norm, in which the x-axis represents a continuous night length and the y-axis represents a probability (or, from the mother's perspective, a proportion) of diapause induction between 0 and 100%. This reaction norm to night length generally follows a logit-curve that ranges from a probability of zero under short nights to a probability of 1 under long nights, and the inflection point at which half of the offspring are diapausing forms is called critical day length (Danilevskii, 1965). The day length response is additionally modulated by temperature (warm temperatures delay diapause), but we ignore the additional plasticity to temperature in our considerations.

Imagine an environment in which winter onsets over many years always occur at 14h night length. Obviously day length would be a reliable cue and plasticity to day length can be expected to evolve. A normal distribution with a mean of 14h and some standard deviation, on the other hand, describes a cue that predicts environmental change only partially. We now use three different scenarios to illustrate optimal reaction norm shapes to such partially predictable conditions: a normal distribution $N_1(14, 1)$ that is distributed around a cue c with a mean of 14 and standard deviation 1 (Fig. 2A, blue line); a distribution $N_2(14, 4)$ that simulates lower predictability by day length (orange line); and 0.5 * $N_3(14,2)$, i.e. a distribution of intermediate variance, but where winter is mild in half of the cases.

The cumulative distribution function of N describes the probability that winter will occur at a night length of c or lower (Fig. 2B). If an aphid lives in an environment of exactly 14 hours night length, it can expect that the offspring will experience winter conditions with a 50% probability (blue and orange line). At 15 hours night length winter onset is quite probable (85%) for environment N_1 (blue line), but the probability is only 60% for N_2 (orange line), because winter onset is more variable. In N_3 the probability is further reduced, to 35%, because there is a high chance that winter is mild (green line). With fitness values as introduced earlier (parthenogenesis: 4/1; diapause: 1/1), the optimal proportion can be calculated as 1, 0.76 and 0.39, respectively, for the three distributions under 15h day length. This way the optimal response to any environmental cue c, i.e. the complete optimal reaction norm, can be calculated if mean and standard deviation of the environment-cue relationship are known (Fig. 2C, D).

With these considerations we explained the reaction norm shape as a series of binary decisions. In each of these decisions, phenotype proportions may range from arithmetic mean optimization to risk-aversion, with developmental instability in between. From our examples it is obvious that both the degree of developmental instability (slope) and the proportion of risk-aversive phenotypes (skewness) change with environmental predictability, but the relative contribution of each is difficult to quantify. Furthermore, our examples feature nearly logistic reaction norms, but depending on the environmental cue, other shapes (e.g. bimodal, sinusoid) are possible. We hence require summary statistics that adequately describe the reaction norm shape.

5 Classification of reaction norm shapes

First, let us assume a “plastic” reaction norm (Fig. 3A, blue line). A step function describes a sudden switch from one phenotype (the arithmetic mean optimizer) to the other (the risk-aversive one), and the number of environments in which a mix of phenotypes is produced is minimized. This function maximises the standard deviation of phenotype proportions across environments (p_i). We refer to the variance of p_i as σ^2_{among}. The opposite of a step function is one in which the mother’s decision is entirely independent of the environmental cue, i.e. left to developmental instability, and both phenotypes are produced in equal measure (Fig. 3A, orange line). While σ^2_{among} is zero, there is variance in phenotypes within each environment (σ^2_{within}). This variance is calculated as $p_i * (1-p_i)$, since the trait choice is a Bernoulli draw. The two variance components complement each other, i.e. it is not possible to maximize both σ^2_{among} (steep slope, high range) and σ^2_{within} (minimal departure from 50%). Intermediate reaction norms are possible, however (Fig. 3A, green line). The trade-off between σ^2_{among} and σ^2_{within} can be described by their ratio *r*. *r* thus describes the degree of developmental (in)stability across environments.

Another type of reaction norm is that of a highly canalized genotype (Fig 3B, blue lines). In the extreme case, the genotype does not react to the environment at all, and produces a single phenotype in every environment. In this case both variances ($\sigma^2_{within}$and σ^2_{among}) are zero. As with Fig. 3A, less extreme reaction norm shapes are also possible: a reaction norm may be, for example, flat at $p_i = 0.1$ (Fig. 3B, orange line), or have a steep slope but only range from $p = 0$ to $p = 0.2$ (Fig 3B, green line). Reaction norms can thus vary from complete canalization to high phenotypic variance, and we express total phenotypic variance *s* by the sum of the two variance components. A canalized reaction norm may be only expressing risk-aversive phenotypes, or only expressing arithmetic mean optimizers, whereas high phenotypic variance may indicate steep reaction norms or high developmental instability.

A last consideration is the overall frequency of the conservative phenotypes across environments. The reaction norm may, for example shift along the x-axis (Fig. 3C, orange line),e.g. when winter onset is highly unpredictable. A flat reaction norm (Fig. 3C, green line) that shifts the balance from developmental instability ($p_i = 0.5$) to risk-aversion ($p_i = 1$) equally in all environments also increases the frequency. We denote the frequency of conservative phenotypes as *f*.

In summary we discussed three important parameters that describe a reaction norm shape: The frequency *f*, the total phenotypic variance *s*, and the variance composition *r* (among:within environments). These three parameters are partially interdependent of one another, and can be drawn as three perpendicular axes (Fig. 3D). The resulting parameter space has three distinct ends which conform to ideal plasticity, risk-aversion, and arithmetic mean optimization.

6 Phenotypic plasticity, bet-hedging and adaptive canalization

So far we described optimal strategies in a single environment, calculated optimal reaction norm shapes, and explored which reaction norm shapes are generally possible. We are now interested in how the optimal reaction norm shape parameters change with changing environmental means, variability and amplitude, by focusing back on our diapause example. First, mean winter onset may vary with latitude, with earlier winter onset at high latitudes (Danilevskii, 1965). Secondly, winter onset dates may vary among years, which is the condition that should lead to bet-hedging in diapause timing (Halkett et al., 2004). Lastly, aphid populations in warmer climates frequently lost the ability to produce sexual forms and reproduce by parthenogenesis throughout the year (anholocyclic life cycles, Simon et al., 2002). The preparation for winter makes only sense if there is sufficient change in environmental conditions, so this kind of canalization (obligate development) is expected at southern latitudes.

We start with environments that vary in among-years predictability. We consider environments that are normally distributed around cues (*c*) with a mean of 14 and standard deviations ranging from 0 to 10. In our introduced example with growth rates of 4/0 (parthenogenetic) and 1/1 (diapausing), the ratio *r* decreases with environmental predictability (Fig. 4A, blue solid line), while the mean frequency *f* of risk-aversive (diapausing) phenotypes increases (Fig. 4B, blue solid line). The sum *s*, on the other hand, remains relatively stable (Fig. 4C). Thus, both DBH and CBH are expected to evolve simultaneously in unpredictable conditions (see also Fig. 4D). With decreasing growth rate of P_1 (parthenogenesis in summer) the ratio decreases less sharply and the diapausing frequency increases more strongly (solid orange and green lines). Here the riskier strategy pays off less, and the balance is shifted towards CBH. When the growth rate of P_1 in E_2 (winter) is raised to 0.33 (i.e. the environmental risk is lower), both *r* and *f* change less steeply with environmental unpredictability (dashed lines), i.e. the reaction norms tend towards arithmetic mean optimization. Increasing the growth rate in winter further to 0.66 leads to a very risk-prone strategy, because risk-aversion pays only off when the chance of mild (summer) conditions is very low. The range of environments that feature a sufficiently low chance of summer decreases with increasing environmental variance, causing a drop of both f and s as a sign of canalization to AMO (dotted lines). Overall, both CBH and DBH can be expected under unpredictable conditions, but their relative benefits vary depending on the arithmetic mean fitness of risk-aversive and risk-prone phenotypes.

To simulate the effect of low environmental amplitudes, e.g. mild winters, we multiply the normal distribution by 0.5 (see also Fig. 2A, B). This discourages risk-aversion and it no longer pays off to have all offspring diapausing (Fig. 5). When the growth rate of parthenogenesis is either 4 (summer) or zero (winter), the phenotypic variance stagnates at 0.2, and the frequency is fixed between 0.28 and 0.29 (solid blue line). This is because the reaction norm range is constrained (Fig. 5D). A lower growth rate of P1 in E1 restores phenotypic variance (Fig. 5B, orange and green lines), as it reduces arithmetic mean fitness of parthenogenesis and makes diapause again more profitable. This increases the range of the reaction norm again (Fig. 5D, orange line). Lowering the environmental risk further increases the benefit of arithmetic mean optimization (dashed lines) and eventually leads to AMO under all environmental conditions (dotted lines). Overall, Fig. 5 shows that occasional mild winters may discourage CBH, and instead favor AMO. For example, a lower risk of freezing in winter may explain the existence of anholocyclic lines.

A third axis of environmental variation concerns changes in mean environments. Moving the distribution of environments to a mean *c* of 10h simulates the change of winter onset with latitude, as well as the effects of a changing climate. Although highly relevant for the optimization of fitness, the changes in optimal reaction norm shapes are trivial to describe. We refer to supplementary Material S1 for further exploration.

In general, we find that *r* evolves with changes in environmental predictability (Fig. 4A), whereas *s* is a function of winter severity (Fig. 5 B). The mean frequency *f* depends strongly on the mean environment (Supp. S1), but may also change with environmental predictability as conservative bet-hedging strategy (Fig. 4C, solid lines), or as risk-prone strategy that seeks to optimize the arithmetic mean (Fig. 4C, dotted lines).

7 Discussion

Phenotypic plasticity can help organisms adapt to changing conditions (Fox et al., 2019), but this requires a predictable cue (Bonamour et al., 2019). Especially for transgenerational plasticity cues are not entirely predictable (Burgess and Marshall, 2014; Donelson et al., 2018), which, at least under some conditions, favours bet-hedging instead (Botero et al., 2015; Tufto, 2015). Nevertheless, the value of bet-hedging strategies as alternative to plasticity is frequently overlooked.

Starrfelt and Kokko (2012) have explained bet-hedging, including its mathematical foundation, in great detail. The main finding was that arithmetic mean fitness optimization, diversified bet-hedging and conservative bet-hedging form a three-way trade-off of conflicting strategies. However, it was difficult to see how these strategies play out in practice (Haaland et al., 2020). We provided a simple, detailed calculation of fitness based on insect diapause as example. Based on this system with only two possible phenotypes (biphenisms) we explained that the three strategies form a gradient, in which arithmetic mean optimization (AMO) and conservative bet-hedging (CBH) are represented by distinct phenotypes, and diversified bet-hedging (DBH) by a mixture of the two extremes. This view is not fundamentally different from Starrfelt and Kokko’s, but easier to imagine, and can be moreover easily placed into a reaction norm framework.

We also extended the concept to multiple environments, thereby incorporating phenotypic plasticity. We rephrased reaction norms as a series of decisions under different environmental frequencies, wherein the solution to each environment ranges from CBH to AMO. The reaction norm shape can be classified along three dimensions, and each of the 6 endpoints on the three-dimensional continuum can be associated with one evolutionary strategy.

7.1 Phenotypic plasticity vs diversified bet-hedging

Predictable conditions select for a high *r*, i.e. the phenotypes change with the environments but vary only little within each environment. This reaction norm pattern is commonly referred to as phenotypic plasticity, or, when the offspring phenotype is dictated by the (grand-) parental environment, as inter- or transgenerational plasticity. A low *r*, on the other hand, corresponds to high developmental instability across the range of possible environments, and occurs predominantly when environments are unpredictable. When higher developmental instability is adaptive, it is called diversified bet-hedging (Simons and Johnston, 1997). We therefore see phenotypic plasticity and diversified bet-hedging as a continuum of evolutionary strategies that is based on the reaction norm shape. The two ends of this strategy continuum differ in how much information about the environment is available and used, hence one may label this axis “information use”.

This definition extends classical concepts of bet-hedging and trans-generational plasticity. Plasticity has a long history of being related to reaction norm shapes (Woltereck, 1913; Bradshaw, 1965), but diversified bet-hedging is not as easily visualized, nor is the relationship with plasticity entirely clear. On the one hand, developmental instability has been seen as a cause of diversified bet-hedging (Simons and Johnston, 1997; Kærn et al., 2005; Woods, 2014; Dueck et al., 2016; Perrin, 2016). Low copy numbers e.g. of transcriptional regulators (Volfson et al., 2006) cause sampling errors that ultimately lead to expression of alternative phenotypes. On the other hand, DBH might be produced by a reaction norm to noise (“microplasticity”, Simons and Johnston, 2006; “hyperplasticity”, Scheiner and Holt, 2012). For example, Maxwell and Magwene (2017) engineered a yeast model that evolved a response to estradiol, a compound that was entirely unrelated to fitness but ensured phenotypic variance in a fluctuating environment. Accordingly, the relationship between diversified bet-hedging and plasticity might be perceived as nested or as one of two competing strategies. We instead distinguish them as the two extremes on a continuum of strategies, that correspond to a continuum of reaction norm shapes.

7.2 Fixed vs. flexible development

s scales with environmental amplitude, i.e. with the fitness difference between environments. When the fitness difference is low or there is a high chance that winters are mild, genotypes can afford to be canalized and never diapause. In high-amplitude environments, however, the selection pressure on phenotypic variance increases, and the genotypes are forced to express a second phenotype, be it by increasing variance among or within environments. This continuum from canalization to phenotypic variance corresponds to a second axis of evolutionary strategies, and we label the axis as “responsiveness” and the two ends as “fixed” and “flexible”, respectively.

The current use of the term canalization is ambiguous (Debat and David, 2001), as environmental canalization may be considered either the opposite of plasticity (Waddington, 1942; Van Buskirk and Steiner, 2009) or of developmental noise (Gibson and Wagner, 2000; Zhang and Hill, 2005). We take an integrative view and see environmental canalization as the opposite of phenotypic variance, including both variance components. Phenotypic plasticity is regarded an essential component of climate change adaptation (Fox et al., 2019), precisely because it prevents canalization to a single environment; moreover, de-canalization by phenotypic plasticity may accelerate evolution through genetic accommodation (Kelly, 2019). We argue that the same mechanisms may apply for all modes of phenotypic variance, including diversified bet-hedging.

7.3 Arithmetic mean optimization vs. conservative bet-hedging

we showed that *f* corrleates with the frequency of E_2 in most environmental conditions, as could be intuitively expected as an AMO strategy. In highly unpredictable environments, however, we found that the frequency of diapause phenotypes is higher than expected. When there are more risk-aversion phenotypes across the environmental range than expected by the environmental frequency, the fitness variance is reduced at the cost of arithmetic mean fitness. An adaptive increase of *f* hence constitutes conservative bet-hedging.

Our framework made clear that arithmetic mean optimization and variance avoidance form exact opposites on a gradient of strategies that is reflected by *f*; therefore, frequencies, means or midpoints of reaction norms that do not match environmental means might serve a function. Recent climate change imposes novel environmental conditions, and species or populations whose trait means do not evolve in concert with environmental means are often considered as under risk (e.g. Charmantier and Gienapp, 2014), which ignores the potential benefit (or at least reduced loss) due to CBH. This is not to say that CBH can be invoked whenever environmental variance is observed (Simons, 2011), but any combination of mean optimization and variance avoidance (*f*) has the potential to be adaptive depending on life history and environmental variance.

CBH and DBH have been separated as distinct strategies early on (Seger and Brockmann, 1987), yet they remain often discussed in conjunction (e.g. Simons, 2011). Similarly, DBH and plasticity were contrasted against each other (Cooper and Kaplan, 1982), but may be invoked simultaneously (e.g. Simons and Johnston, 2006; Maxwell and Magwene, 2017). Starrfelt and Kokko (2012) provided a useful framework which joined CBH, DBH and AMO; we built on this framework (although our views differ slightly), adding plasticity, canalization and phenotypic variance.

The world is simultaneously changing in climate means, variability and predictability (IPCC, 2014; Lenton et al., 2017; Bathiany et al., 2018), and we argue that a model on reaction norm evolution should reflect this three-dimensional relationship. Currently there are many phenomenological studies on responses to climate change (Parmesan and Yohe, 2003; Badeck et al., 2004; Cohen et al., 2018), but only few detailed case-studies on the mechanisms of adaptation (Nussey et al., 2005; Gienapp et al., 2013; Lane et al., 2018). One cannot assume that a matching trait mean or a high level of plasticity is always adaptive (Boutin and Lane, 2014), just like one cannot assume CBH or DBH to be an optimal solution (Simons, 2011) – but we can analyse the reaction norm shape to decide whether it has the *potential* for adaptive mean optimization, plasticity, bet-hedging or canalization.

There is ample room to extend our framework. We have restricted our arguments to binary trans-generationally inherited traits, as these are commonly treated both empirically (Venable, 2007; Maxwell and Magwene, 2017; Scholl et al., 2020) and theoretically (Cohen, 1966; Halkett et al., 2004; Starrfelt and Kokko, 2012; Kivela et al., 2016; Gerber and Kokko, 2018). For continuous traits, e.g. offspring size (Marshall et al., 2008), our calculations may not apply, because AMO, DBH and CBH need not lie on a linear gradient (i.e. intermediate trait values need not incur highest trait variance). Nevertheless, theory regarding Gaussian functions arrives at a similar conclusion: that offspring variance evolves to the amount of environmental mismatch that is not already covered by phenotypic plasticity (Tufto, 2015). This is equivalent to our finding that in high-amplitude environments only the variance composition (r) changes with environmental variability, whereas the degree of phenotypic variance (s) remains constant. Other possible extensions would include plastic responses that take place within an individual's life time. The opportunity for both within- and transgenerational plasticity may not only make one strategy obsolete (Luquet and Tariel, 2016), but also lead to complex interactions among the two (Fuxjäger et al., 2019). Lastly, there are also potential bet-hedging strategies that appear entirely unrelated to transgenerational plasticity. These include, for example, an iteroparous life history (Garcia-Gonzalez et al., 2015), hotspots for genetic mutations ("contingency loci", Rando and Verstrepen, 2007), and sexual reproduction in general (Li et al., 2017). A unification with these alternative strategies might lead to a better understanding of adaptation to rapid climate change.

8 Conclusion

In this review we rephrased reaction norm evolution as a complex trade-off among three axes of strategies. It is increasingly recognized that changes in climate extremes and in predictability are as important as changes in means (IPCC, 2014; Donelson et al., 2018) – focusing only on strategies to match the mean is hence not fruitful. For example, failure to shift mean phenology with climate change (Gienapp et al., 2013) is not problematic per se – it could be mitigated by concurrent changes in responsiveness. Similarly, the lack of both phenotypic plasticity and mean change may not have severe fitness consequences, if the lack of plasticity is mitigated by diversified bet-hedging. It is the combination along all three axes that defines fitness in a given environment.

Box 1: Fitness optimization – trade-off or gradient?

Our article describes arithmetic mean optimization (AMO), diversified bet-hedging (DBH) and conservative bet-hedging (CBH) as a gradual continuum. This appears to contrast the view of Starrfelt and Kokko (2012), who describe the strategies as a three-way trade-off. To clarify this apparent contradiction, we want to reiterate the example given by Starrfelt and Kokko (2012), which itself is borrowed from Seger and Brockman (1987). In their example, one genotype (“A_{wet}”) has 1 fitness in a wet environment and 0.6 in a dry one, whereas the other genotype (“A_{dry}”) has 0.58 fitness in the wet environment and 1 fitness in the dry environment. A genotype that produces a mix of both specialists is a diversified bet-hedger, because it achieves a lower arithmetic mean fitness but higher geometric mean fitness than a wet-adapted specialist. To explain CBH, however, the authors introduced another genotype with a fitness of .785 under both environments. It thus appears that AMO, CBH and DBH form three corners on a triangular continuum of strategies.

We ignore the conservative genotype for a moment and concentrate on the optimal proportion of A_{dry} and A_{wet} (Figure B1, blue line). The pure production of A_{wet} ($p = 0$) maximizes arithmetic mean fitness, whereas a mixed production ($p = 0.5$) minimizes the fitness correlation among the offspring and may hence constitute DBH. Individual fitness variance would be minimized by producing only A_{dry} ($p = 1$), but this is not a viable alternative to arithmetic mean optimization in this example, as the geometric mean fitness is much lower than that of $p = 0$. In other words, this example is not suited to explain CBH. To make CBH possible, one could change A_{dry} to 0.65 in wet environments, and 0.93 in dry environments, i.e. reduce the fitness variance further at only moderate reduction of arithmetic mean fitness (orange line). In this altered example the exclusive production of the dry phenotype is marginally better than AMO, though DBH at $p = 0.51$ would still be optimal. Lastly, one may change A_{dry} to 0.785 under both environments (green line). Now the dry-adapted specialist achieves a much higher geometric mean fitness than the wet-adapted specialist, despite a lower arithmetic mean fitness. This is also the conservative bet-hedger in the example of Starrfelt and Kokko (2012). The highest fitness is reached at a phenotype proportion of 0.7, i.e. about halfway between CBH and DBH. In all cases that make CBH a possible strategy, there is a gradient, not a three-way trade-off, from AMO to CBH, with DBH in between. In other words, it is impossible to think of a continuum between CBH and AMO that does not involve DBH.

9 Acknowledgments

JJ was financially supported by a DFG research fellowship. DB is funded by FWO project G018017N.

10 Author contributions

JJ and DB developed the theory and JJ wrote the first draft. Both authors contributed to the final version of the manuscript. DB supervised the work.

11 Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

12 Data availability statement

All datasets generated for this study are included in the supplementary file.

13 References

Adrian-Kalchhauser, I., Sultan, S. E., Shama, L., Spence-Jones, H., Tiso, S., Valsecchi, C. I. K., et al. (2020). Understanding "Non-genetic" Inheritance: Insights from Molecular-Evolutionary Crosstalk. *EcoEvoRxiv*. doi:10.32942/osf.io/gy5pr.

Arnold, P. A., Nicotra, A. B., and Kruuk, L. E. B. (2019). Sparse evidence for selection on phenotypic plasticity in response to temperature. *Phil Trans R Soc B* 374, 20180185. doi:10.1098/rstb.2018.0185.

Badeck, F. W., Bondeau, A., Bottcher, K., Doktor, D., Lucht, W., Schaber, J., et al. (2004). Responses of spring phenology to climate change. *New Phytol* 162, 295–309. doi:10.1111/j.1469-8137.2004.01059.x.

Baker, B. H., Sultan, S. E., Lopez-Ichikawa, M., and Waterman, R. (2019). Transgenerational effects of parental light environment on progeny competitive performance and lifetime fitness. *Phil Trans R Soc B* 374, 20180182. doi:10.1098/rstb.2018.0182.

Bathiany, S., Dakos, V., Scheffer, M., and Lenton, T. M. (2018). Climate models predict increasing temperature variability in poor countries. *Sci Adv* 4, eaar5809. doi:10.1126/sciadv.aar5809.

Bonamour, S., Chevin, L.-M., Charmantier, A., and Teplitsky, C. (2019). Phenotypic plasticity in response to climate change: the importance of cue variation. *Phil. Trans. R. Soc. B* 374, 20180178. doi:10.1098/rstb.2018.0178.

Botero, C. A., Weissing, F. J., Wright, J., and Rubenstein, D. R. (2015). Evolutionary tipping points in the capacity to adapt to environmental change. *PNAS* 112, 184–189. doi:10.1073/pnas.1408589111.

Boutin, S., and Lane, J. E. (2014). Climate change and mammals: evolutionary versus plastic responses. *Evol Appl* 7, 29–41. doi:10.1111/eva.12121.

Bradshaw, A. D. (1965). Evolutionary significance of phenotypic plasticity in plants. *Adv Gen* 13, 115–155. doi:10.1016/S0065-2660(08)60048-6.

Braendle, C., Davis, G. K., Brisson, J. A., and Stern, D. L. (2006). Wing dimorphism in aphids. *Heredity* 97, 192–199. doi:10.1038/sj.hdy.6800863.

Burgess, S. C., and Marshall, D. J. (2014). Adaptive parental effects: the importance of estimating environmental predictability and offspring fitness appropriately. *Oikos* 123, 769–776. doi:10.1111/oik.01235.

Charmantier, A., and Gienapp, P. (2014). Climate change and timing of avian breeding and migration: evolutionary versus plastic changes. *Evol Appl* 7, 15–28. doi:10.1111/eva.12126.

Charmantier, A., McCleery, R. H., Cole, L. R., Perrins, C., Kruuk, L. E. B., and Sheldon, B. C. (2008). Adaptive Phenotypic Plasticity in Response to Climate Change in a Wild Bird Population. *Science* 320, 800–803. doi:10.1126/science.1157174.

Chevin, L.-M., Lande, R., and Mace, G. M. (2010). Adaptation, Plasticity, and Extinction in a Changing Environment: Towards a Predictive Theory. *PLOS Biology* 8, e1000357. doi:10.1371/journal.pbio.1000357.

Cohen, D. (1966). Optimizing reproduction in a randomly varying environment. *J Theor Biol* 12, 119–129. doi:10.1016/0022-5193(66)90188-3.

Cohen, J. M., Lajeunesse, M. J., and Rohr, J. R. (2018). A global synthesis of animal phenological responses to climate change. *Nature Clim Change* 8, 224–228. doi:10.1038/s41558-018-0067-3.

Cooper, W. S., and Kaplan, R. H. (1982). Adaptive “coin-flipping”: a decision-theoretic examination of natural selection for random individual variation. *J Theor Biol* 94, 135–151. doi:10.1016/0022-5193(82)90336-8.

Danilevskii, A. S. (1965). *Photoperiodism and seasonal development of insects.* 1st ed. Edinburgh: Oliver & Boyd.

Darwin, C. (1859). *On the Origin of Species by Means of Natural Selection, or the Preservation of Favoured Races in the Struggle for Life*. London: J. Murray.

Debat, V., and David, P. (2001). Mapping phenotypes: canalization, plasticity and developmental stability. *Trends Ecol Evol* 16, 555–561. doi:10.1016/S0169-5347(01)02266-2.

Donelson, J. M., Salinas, S., Munday, P. L., and Shama, L. N. S. (2018). Transgenerational plasticity and climate change experiments: Where do we go from here? *Glob Change Biol* 24, 13–34. doi:10.1111/gcb.13903.

Dueck, H., Eberwine, J., and Kim, J. (2016). Variation is function: Are single cell differences functionally important? *BioEssays* 38, 172–180. doi:10.1002/bies.201500124.

Fox, R. J., Donelson, J. M., Schunter, C., Ravasi, T. C., and Gaitán-Espitia, J. D. (2019). Beyond buying time: the role of plasticity in phenotypic adaptation to rapid environmental change. *Phil Trans R Soc B* 374, 20180174. doi:10.1098/rstb.2018.0174.

Furness, A. I., Lee, K., and Reznick, D. N. (2015). Adaptation in a variable environment: Phenotypic plasticity and bet-hedging during egg diapause and hatching in an annual killifish. *Evolution* 69, 1461–1475. doi:10.1111/evo.12669.

Fuxjäger, L., Wanzenböck, S., Ringler, E., Wegner, K. M., Ahnelt, H., and Shama, L. N. S. (2019). Within-generation and transgenerational plasticity of mate choice in oceanic stickleback under climate change. *Phil. Trans. R. Soc. B* 374, 20180183. doi:10.1098/rstb.2018.0183.

Gerber, N., and Kokko, H. (2018). Abandoning the ship using sex, dispersal or dormancy: multiple escape routes from challenging conditions. *Phil Trans R Soc B* 373, 20170424. doi:10.1098/rstb.2017.0424.

Ghalambor, C. K., McKay, J. K., Carroll, S. P., and Reznick, D. N. (2007). Adaptive versus non-adaptive phenotypic plasticity and the potential for contemporary adaptation in new environments. *Funct Ecol* 21, 394–407. doi:https://doi.org/10.1111/j.1365-2435.2007.01283.x.

Gibson, G., and Wagner, G. (2000). Canalization in evolutionary genetics: a stabilizing theory? *BioEssays* 22, 372–380. doi:10.1002/(SICI)1521-1878(200004)22:4<372::AID-BIES7>3.0.CO;2-J.

Gienapp, P., Lof, M., Reed, T. E., McNamara, J., Verhulst, S., and Visser, M. E. (2013). Predicting demographically sustainable rates of adaptation: can great tit breeding time keep pace with climate change? *Phil Trans R Soc B* 368, 20120289. doi:10.1098/rstb.2012.0289.

Goossens, S., Wybouw, N., Van Leeuwen, T., and Bonte, D. (2020). The physiology of movement. *Mov Ecol* 8, 5. doi:10.1186/s40462-020-0192-2.

Grantham, M. E., Antonio, C. J., O'Neil, B. R., Zhan, Y. X., and Brisson, J. A. (2016). A case for a joint strategy of diversified bet hedging and plasticity in the pea aphid wing polyphenism. *Biol Lett* 12, 20160654. doi:10.1098/rsbl.2016.0654.

Green, T. R., and Ryan, C. A. (1972). Wound-Induced Proteinase Inhibitor in Plant Leaves: A Possible Defense Mechanism against Insects. *Science* 175, 776–777. doi:10.1126/science.175.4023.776.

Haaland, T. R., Wright, J., and Ratikainen, I. I. (2020). Generalists versus specialists in fluctuating environments: a bet-hedging perspective. *Oikos* 129, 879–890. doi:10.1111/oik.07109.

Halkett, F., Harrington, R., Hullé, M., Kindlmann, P., Menu, F., Rispe, C., et al. (2004). Dynamics of production of sexual forms in aphids: theoretical and experimental evidence for adaptive "coin-flipping" plasticity. *Am Nat* 163, E112–E125. doi:10.1086/383618.

Hopper, K. R. (1999). Risk-spreading and bet-hedging in insect population biology. *Annu Rev Entomol* 44, 535–560. doi:10.1146/annurev.ento.44.1.535.

IPCC (2014). *Climate Change 2014: Synthesis report. Contribution of Working Groups I, II and III to the fifth assessment report of the Intergovernmental Panel on Climate Change.* Geneva, Switzerland.

Kærn, M., Elston, T. C., Blake, W. J., and Collins, J. J. (2005). Stochasticity in gene expression: from theories to phenotypes. *Nat Rev Genet* 6, 451–464. doi:10.1038/nrg1615.

Kelly, M. (2019). Adaptation to climate change through genetic accommodation and assimilation of plastic phenotypes. *Phil Trans R Soc B* 374, 20180176. doi:10.1098/rstb.2018.0176.

Kivela, S. M., Valimaki, P., and Gotthard, K. (2016). Evolution of alternative insect life histories in stochastic seasonal environments. *Ecol Evol* 6, 5596–5613. doi:10.1002/ece3.2310.

Krueger, D. A., and Dodson, S. I. (1981). Embryological induction and predation ecology in Daphnia pulex. *Limnol Oceanogr* 26, 219–223. doi:10.4319/lo.1981.26.2.0219.

Lane, J. E., McAdam, A. G., McFarlane, S. E., Williams, C. T., Humphries, M. M., Coltman, D. W., et al. (2018). Phenological shifts in North American red squirrels: disentangling the roles of phenotypic plasticity and microevolution. *J Evol Biol* 31, 810–821. doi:10.1111/jeb.13263.

Lenton, T. M., Dakos, V., Bathiany, S., and Scheffer, M. (2017). Observed trends in the magnitude and persistence of monthly temperature variability. *Sci Rep* 7, 5940. doi:10.1038/s41598-017-06382-x.

Luquet, E., and Tariel, J. (2016). Offspring reaction norms shaped by parental environment: interaction between within- and trans-generational plasticity of inducible defenses. *BMC Evolutionary Biology* 16, 209. doi:10.1186/s12862-016-0795-9.

Marcovitch, S. (1923). Plant lice and light exposure. *Science* 58, 537–538. doi:10.1126/science.58.1513.537-a.

Marshall, D. J., Bonduriansky, R., and Bussière, L. F. (2008). Offspring size variation within broods as a bet-hedging strategy in unpredictable environments. *Ecology* 89, 2506–2517. doi:10.1890/07-0267.1.

Maxwell, C. S., and Magwene, P. M. (2017). When sensing is gambling: An experimental system reveals how plasticity can generate tunable bet-hedging strategies. *Evolution* 71, 859–871. doi:10.1111/evo.13199.

Nussey, D. H., Postma, E., Gienapp, P., and Visser, M. E. (2005). Selection on heritable phenotypic plasticity in a wild bird population. *Science* 310, 304–306. doi:10.1126/science.1117004.

Oostra, V., Saastamoinen, M., Zwaan, B. J., and Wheat, C. W. (2018). Strong phenotypic plasticity limits potential for evolutionary responses to climate change. *Nature Communications* 9. doi:10.1038/s41467-018-03384-9.

Parmesan, C., and Yohe, G. (2003). A globally coherent fingerprint of climate change impacts across natural systems. *Nature* 421, 37. doi:10.1038/nature01286.

Pélisson, P.-F., Bernstein, C., François, D., Menu, F., and Venner, S. (2013). Dispersal and dormancy strategies among insect species competing for a pulsed resource. *Ecological Entomology* 38, 470–477. doi:10.1111/een.12038.

Perez, M. F., and Lehner, B. (2019). Intergenerational and transgenerational epigenetic inheritance in animals. *Nature Cell Biology* 21, 143–151. doi:10.1038/s41556-018-0242-9.

Perrin, N. (2016). Random sex determination: When developmental noise tips the sex balance. *BioEssays* 38, 1218–1226. doi:10.1002/bies.201600093.

Piao, S., Liu, Q., Chen, A., Janssens, I. A., Fu, Y., Dai, J., et al. (2019). Plant phenology and global climate change: Current progresses and challenges. *Global Change Biology* 25, 1922–1940. doi:10.1111/gcb.14619.

Rando, O. J., and Verstrepen, K. J. (2007). Timescales of genetic and epigenetic inheritance. *Cell* 128, 655–668. doi:10.1016/j.cell.2007.01.023.

Scheiner, S. M., and Holt, R. D. (2012). The genetics of phenotypic plasticity. X. Variation versus uncertainty. *Ecol Evol* 2, 751–767. doi:10.1002/ece3.217.

Scholl, J. P., Calle, L., Miller, N., and Venable, D. L. (2020). Offspring polymorphism and bet hedging: a large-scale, phylogenetic analysis. *Ecology Letters* 23, 1223–1231. doi:10.1111/ele.13522.

Seger, J., and Brockmann, H. J. (1987). “What is bet-hedging?,” in *Oxford surveys in evolutionary biology (4)* (Oxford, UK: Oxford University Press), 182–211.

Simon, J.-C., Rispe, C., and Sunnucks, P. (2002). Ecology and evolution of sex in aphids. *Trends Ecol Evol* 17, 34–39. doi:10.1016/S0169-5347(01)02331-X.

Simons, A. M. (2011). Modes of response to environmental change and the elusive empirical evidence for bet hedging. *Proc R Soc B* 278, 1601–1609. doi:10.1098/rspb.2011.0176.

Simons, A. M., and Johnston, M. O. (1997). Developmental instability as a bet-hedging strategy. *Oikos* 80, 401–406. doi:10.2307/3546608.

Simons, A. M., and Johnston, M. O. (2006). Environmental and genetic sources of diversification in the timing of seed germination: implications for the evolution of bet hedging. *Evolution* 60, 2280–2292. doi:10.1111/j.0014-3820.2006.tb01865.x.

Starrfelt, J., and 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.

Stomp, M., van Dijk, M. A., van Overzee, H. M. J., Wortel, M. T., Sigon, C. A. M., Egas, M., et al. (2008). The timescale of phenotypic plasticity and its impact on competition in fluctuating environments. *Am Nat* 172, E169–E185. doi:10.1086/591680.

Tufto, J. (2015). Genetic evolution, plasticity, and bet-hedging as adaptive responses to temporally autocorrelated fluctuating selection: A quantitative genetic model. *Evolution* 69, 2034–2049. doi:10.1111/evo.12716.

Van Buskirk, J., and Steiner, U. K. (2009). The fitness costs of developmental canalization and plasticity. *J Evol Biol* 22, 852–860. doi:10.1111/j.1420-9101.2009.01685.x.

Veening, J.-W., Smits, W. K., and Kuipers, O. P. (2008). Bistability, epigenetics, and bet-hedging in bacteria. *Annu Rev Microbiol* 62, 193–210. doi:10.1146/annurev.micro.62.081307.163002.

Venable, D. L. (2007). Bet hedging in a guild of desert annuals. *Ecology* 88, 1086–1090. doi:10.1890/06-1495.

Volfson, D., Marciniak, J., Blake, W. J., Ostroff, N., Tsimring, L. S., and Hasty, J. (2006). Origins of extrinsic variability in eukaryotic gene expression. *Nature* 439, 861–864. doi:10.1038/nature04281.

Waddington, C. H. (1942). Canalization of development and the inheritance of acquired characters. *Nature* 150, 563–565. doi:10.1038/150563a0.

Woltereck, R. (1913). Weitere experimentelle untersuchungen über Artänderung, speziell über das Wesen quantitativer Artunterschiede bei Daphniden. *Mol Gen Genet* 9, 146–146. doi:10.1007/BF01876686.

Woods, H. A. (2014). Mosaic physiology from developmental noise: within-organism physiological diversity as an alternative to phenotypic plasticity and phenotypic flexibility. *J Exp Bio* 217, 35–45. doi:10.1242/jeb.089698.

Zhang, X.-S., and Hill, W. G. (2005). Evolution of the environmental component of the phenotypic variance: stabilizing selection in changing environments and the cost of homogeneity. *Evolution* 59, 1237–1244. doi:10.1111/j.0014-3820.2005.tb01774.x.

14 Figure legends

Fig. 1. Geometric mean fitness as function of the proportion of diapausing offspring when E2 occurs with frequencies of 0.5 (blue) and 0.2 (orange).

Fig. 2 Panel A) Probability of E_2 as function of a cue c. These curves are the probability density functions of three normal distributions N_1 (14,1), N_2(14,4) and 0.5 * N_3(14,2) (blue, orange, green). B) Probability that E changes given c. These curves are the cumulative probability functions of the normal distributions. C) optimal reaction norm shapes in the three environments of panel A and B when fitness of P_1 is 4 in E_1 and 0.1 in E_2 and fitness of P_2 is always 1. D) optimal reaction norm shapes in the three environments of panel A and B when fitness of P_1 is 2.5 in E_1 and 0.1 in E_2 and fitness of P_2 is always 1.2.

Fig. 3: Example reaction norm shapes. A) Three reaction norm shapes that differ in the ratio of the variance components. Blue: only variance among environments, orange: only variance within environments, green: intermediate variance among and within environments. B) Reaction norm shapes that differ in the sum of variance components. Black: canalization; orange: low degree of variance within environments; green: low degree of variance among environments. C) Three reaction norms that vary in mean frequency of P2 (blue: 0.5, orange: 0.3, green: 0.8) across environments. D) possible parameter space of variance composition, sum, and mean frequency. Grey dots depict sample reaction norms across the range of possible parameters, colored dots indicated samples from panel A(blue) and B (black, orange and green).

Fig. 4: Optimal reaction norm shapes for various growth rate functions and different levels of environmental predictability. Environments are normally distributed around a cue c with a mean of 14. Variance composition (Panel A), phenotypic variance (Panel B) and mean frequency (Panel C) are plotted against standard deviation of the environment. Growth rates of P2 (diapause) are always 1 for both environments (summer and winter); growth rates of P1 (parthenogenesis) in E1/E2 are 4/0 (blue, solid), 3/0 (orange, solid), 2/0 (green, solid); 4/0.33, 3/0.33, 2/0.33 (dashed); and 4/0.66, 3/0.66, 2/0.66 (dotted). Panel D shows three optimal reaction norms for Environments with standard deviation of 5.

Fig. 5: Optimal reaction norm shapes for various growth rate functions and different levels of environmental predictability. Environments are normally distributed around a cue c with a mean of 14, but multiplied by 0.5.

Fig. B1 [boxed text]. Geometric mean fitness as function of the proportion of Dry-year specialists in the example of Starrfelt and Kokko (2012). Blue lines indicate geometric mean fitness with the growth rates described by Starrfelt and Kokko, orange and green lines describe slightly modified examples. A_{wet} has always 1 fitness in wet environments and 0.6 fitness in dry environments; the fitness of A_{dry} is 0.58 or 1 (blue lines); 0.65 or 0.93 (orange lines); and 0.785 in both environments (green lines).

Table 1: Growth rate calculations for various phenotype proportions in a two-environment system

	Proportion of P_2			
	0	0.5	1	0.61
E_1	0 * 1 + 1* 4 = **4**	0.5 * 1 + 0.5 * 4 = **2.5**	1 * 1 + 0 * 4 = **1**	0.61 * 1 + 0.39 * 4 = **2.17**
E_2	0 * 1 + 1 * 0.1 = **0.1**	0.5 * 1 + 0.5 * 0.1 = **0.55**	1 * 1 + 0 * 0.1 = **1**	0.61 * 1 + 0.39 * 0.1 = **0.65**
Arithmetic mean	2.05	1.53	1	1.41
Geometric mean	0.63	1.17	1	1.19

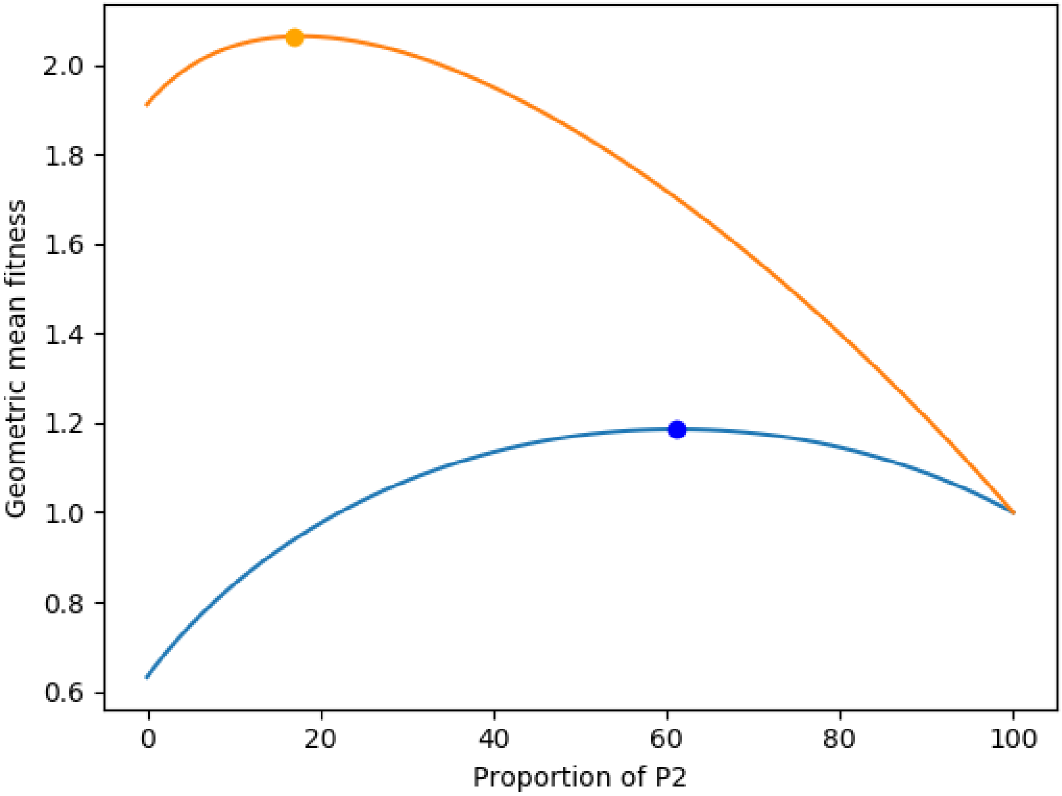

Fig. 1. Geometric mean fitness as function of the proportion of diapausing offspring when E2 occurs with frequencies of 0.5 (blue) and 0.2 (orange).

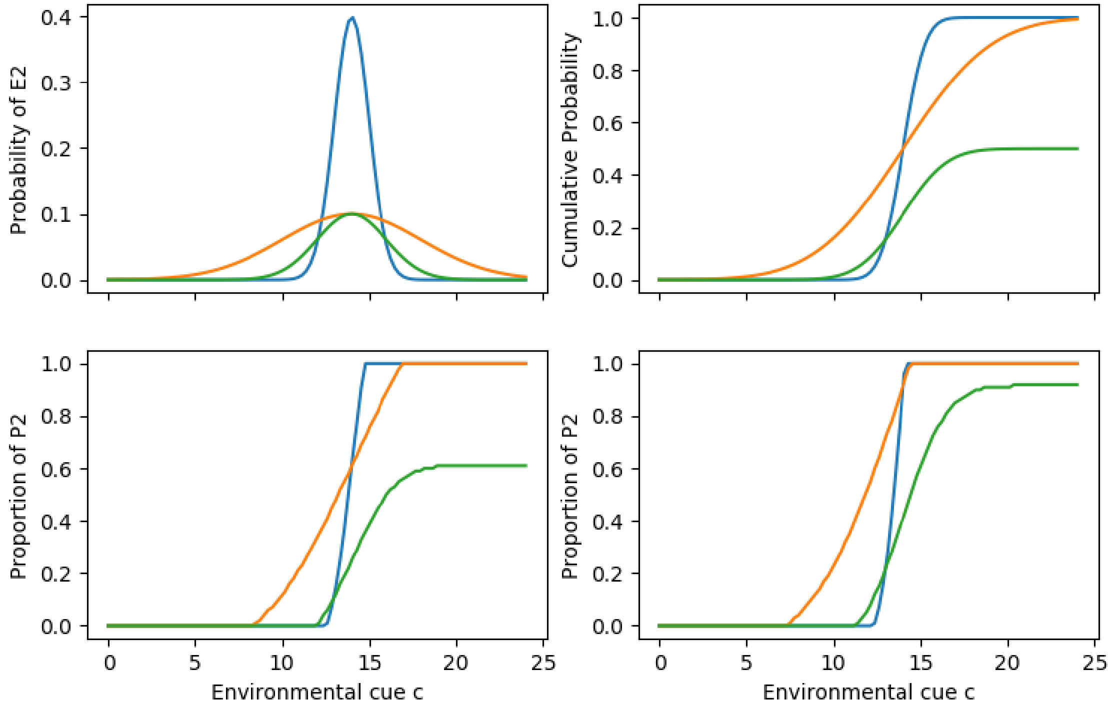

Fig. 2 Panel A) Probability of E_2 as function of a cue *c*. These curves are the probability density functions of three normal distributions N_1 (14,1), N_2(14,4) and 0.5 * N_3(14,2) (blue, orange, green). B) Probability that *E* changes given *c*. These curves are the cumulative probability functions of the normal distributions. C) optimal reaction norm shapes in the three environments of panel A and B when fitness of P_1 is 4 in E_1 and 0.1 in E_2 and fitness of P_2 is always 1. D) optimal reaction norm shapes in the three environments of panel A and B when fitness of P_1 is 2.5 in E_1 and 0.1 in E_2 and fitness of P_2 is always 1.2.

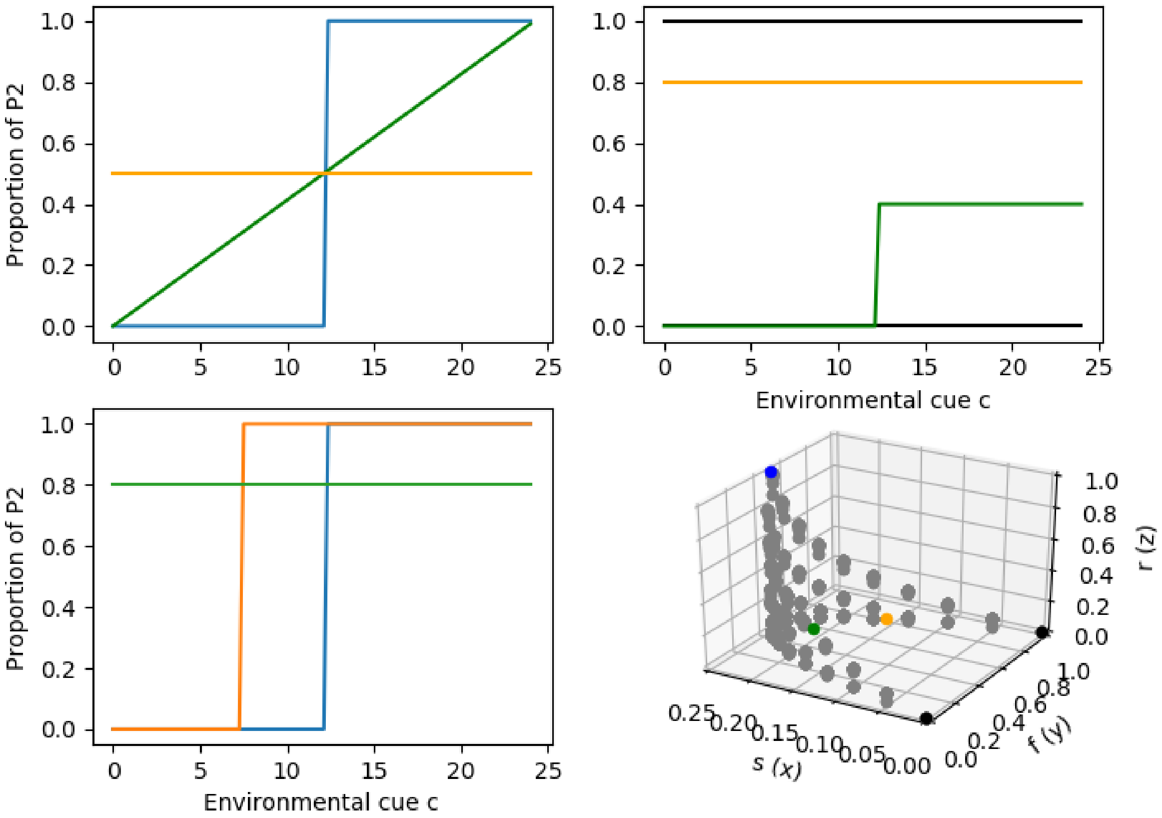

Fig. 3: Example reaction norm shapes. A) Three reaction norm shapes that differ in the ratio of the variance components. Blue: only variance among environments, orange: only variance within environments, green: intermediate variance among and within environments. B) Reaction norm shapes that differ in the sum of variance components. Black: canalization; orange: low degree of variance within environments; green: low degree of variance among environments. C) Three reaction norms that vary in mean frequency of P2 (blue: 0.5, orange: 0.3, green: 0.8) across environments. D) possible parameter space of variance composition, sum, and mean frequency. Grey dots depict sample reaction norms across the range of possible parameters, colored dots indicated samples from panel A(blue) and B (black, orange and green).

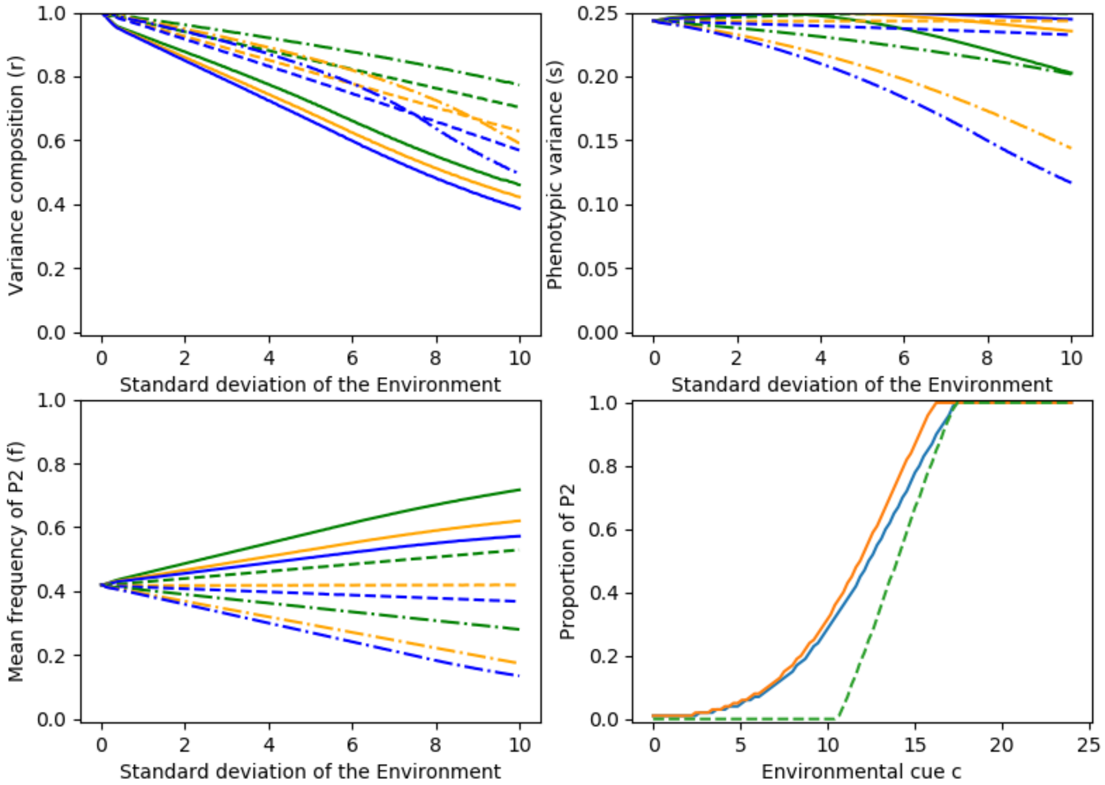

Fig. 4: Optimal reaction norm shapes for various growth rate functions and different levels of environmental predictability. Environments are normally distributed around a cue *c* with a mean of 14. Variance composition (Panel A), phenotypic variance (Panel B) and mean frequency (Panel C) are plotted against standard deviation of the environment. Growth rates of P2 (diapause) are always 1 for both environments (summer and winter); growth rates of P1 (parthenogenesis) in E1/E2 are 4/0 (blue, solid), 3/0 (orange, solid), 2/0 (green, solid); 4/0.33, 3/0.33, 2/0.33 (dashed); and 4/0.66, 3/0.66, 2/0.66 (dotted). Panel D shows three optimal reaction norms for Environments with standard deviation of 5.

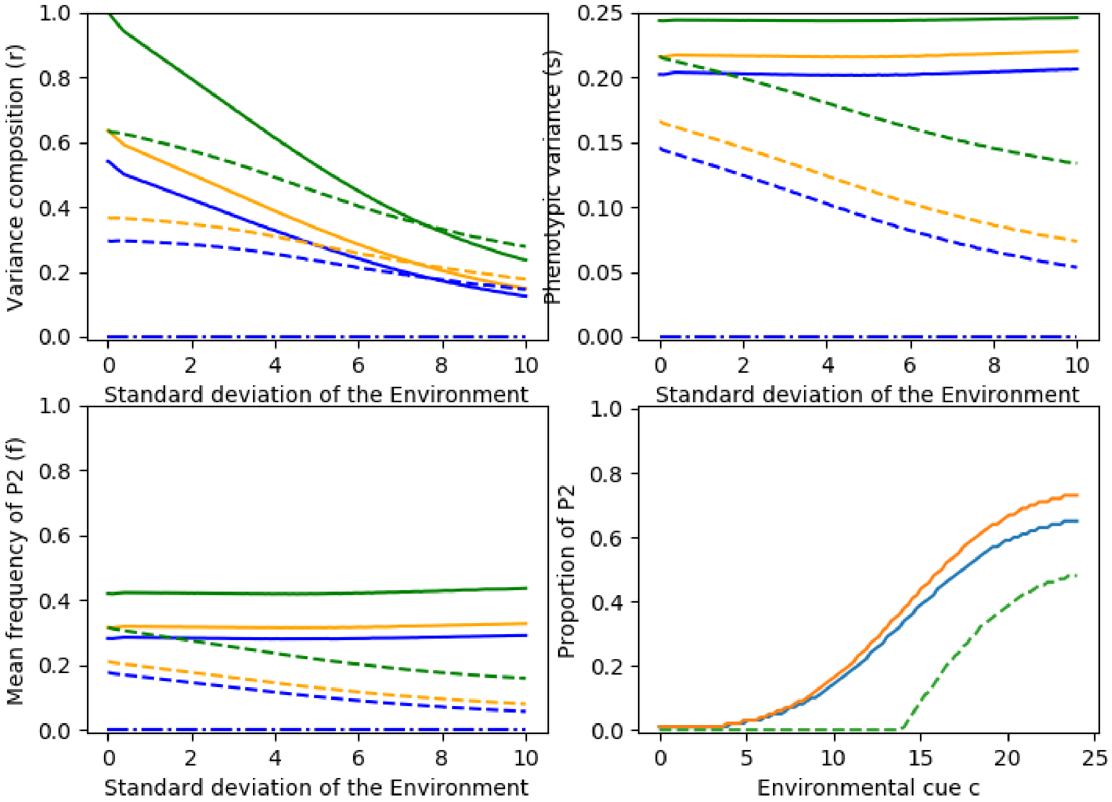

Fig. 5: Optimal reaction norm shapes for various growth rate functions and different levels of environmental predictability. Environments are normally distributed around a cue c with a mean of 14, but multiplied by 0.5.

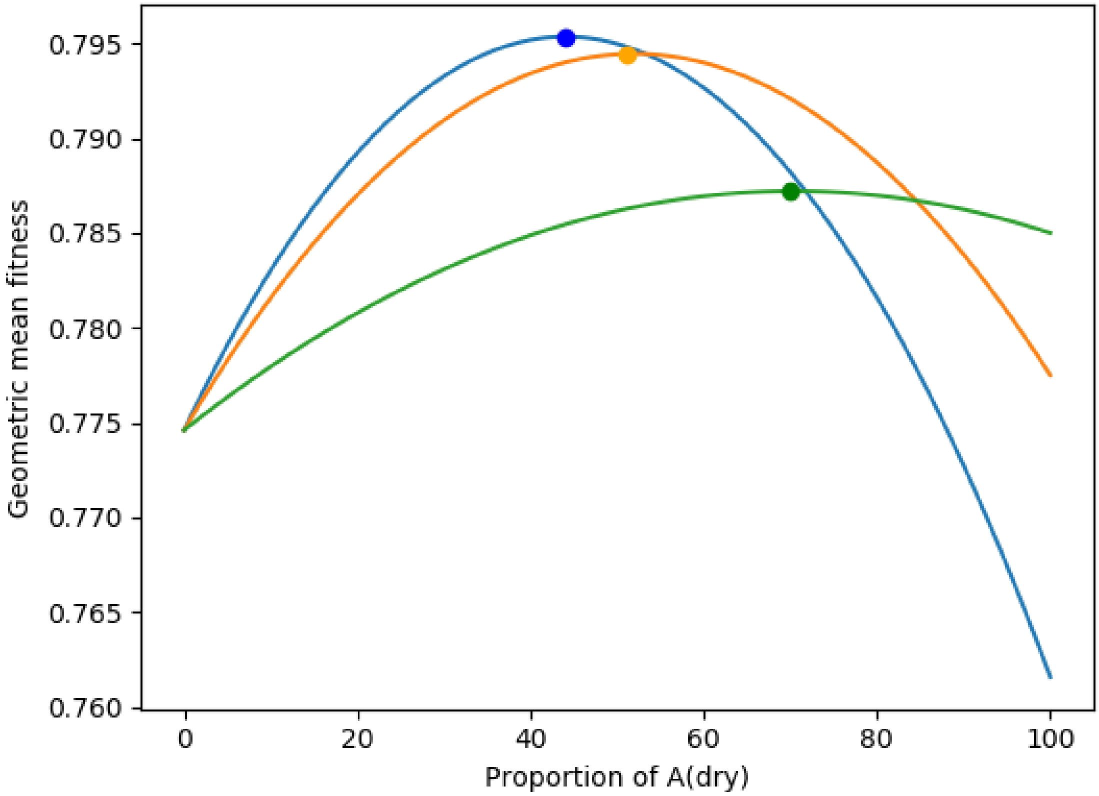

Fig. B1 [boxed text]. Geometric mean fitness as function of the proportion of Dry-year specialists in the example of Starrfelt and Kokko (2012). Blue lines indicate geometric mean fitness with the growth rates described by Starrfelt and Kokko, orange and green lines describe slightly modified examples. A_{wet} has always 1 fitness in wet environments and 0.6 fitness in dry environments; the fitness of A_{dry} is 0.58 or 1 (blue lines); 0.65 or 0.93 (orange lines); and 0.785 in both environments (green lines).