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PAPER

A mathematical model of the evolution of individual differences in developmental plasticity arising through parental bet-hedging

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Abstract

Children vary in the extent to which their development is shaped by particular experiences (e.g. maltreatment, social support). This variation raises a question: Is there no single level of plasticity that maximizes biological fitness? One influential hypothesis states that when different levels of plasticity are optimal in different environmental states and the environment fluctuates unpredictably, natural selection may favor parents producing offspring with varying levels of plasticity. The current article presents a mathematical model assessing the logic of this hypothesis – specifically, it examines what conditions are required for natural selection to favor parents to bet-hedge by varying their offspring's plasticity. Consistent with existing theory from biology, results show that between-individual variation in plasticity cannot evolve when the environment only varies across space. If, however, the environment varies across time, selection can favor differential plasticity, provided fitness effects are large (i.e. variation in individuals' plasticity is correlated with substantial variation in fitness). Our model also generates a novel restriction: Differential plasticity only evolves when the cost of being mismatched to the environment exceeds the benefits of being well matched. Based on mechanistic considerations, we argue that bet-hedging by varying offspring plasticity, if it were to evolve, would be more likely instantiated via epigenetic mechanisms (e.g. pre- or postnatal developmental programming) than genetic ones (e.g. mating with genetically diverse partners). Our model suggests novel avenues for testing the bet-hedging hypothesis of differential plasticity, including empirical predictions and relevant measures. We also discuss several ways in which future work might extend our model.

Research highlights

- We formalize Jay Belsky's bet-hedging hypothesis of differential plasticity.
- Results support the hypothesis' logical coherence, but only under restrictive conditions.
- Our model suggests novel avenues for empirically testing the bet-hedging hypothesis.
- We suggest multiple theoretical extensions of our model.

> . . . it is advisable to divide goods which are exposed to some danger into several portions rather than to risk them all together. (Daniel Bernoulli, 1738 (trans. 1954), p. 30)

> Put all your eggs in one basket and then watch that basket. (Mark Twain, 1894, Pudd'nhead Wilson and Other Tales)

Introduction

Developmental plasticity – the ability to adjust development based on experience – is ubiquitous in nature (Schlichting & Pigliucci, 1998), and evolves because it allows organisms to adaptively tailor their phenotypes to a range of environmental states (Dall, Giraldeau, Ollson, McNamara & Stephens, 2005; West-Eberhard, 2003). Empirical studies of humans show that the degree of plasticity itself may vary across individuals ('differential plasticity'); that is, some individuals are shaped more than others by the same kinds of experiences (Belsky, 1997, 2005; Belsky & Pluess, 2009a, 2009b; Boyce & Ellis, 2005; Ellis, Boyce, Belsky, Bakermans-Kranenburg & van IJzendoorn, 2011; for studies of non-human animals, see Dingemanse & Wolf, 2013). In some cases, highly plastic individuals are also more susceptible to

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experience: that is, they are more adversely affected by harmful environments as well as benefit more from supportive circumstances ('differential susceptibility'; Belsky, Jonassaint, Pluess, Stanton, Brummett *et al.*, 2009; Boyce, Chesney, Alkon, Tschann, Adams *et al.*, 1995; Pluess & Belsky, 2011, 2013; Ellis *et al.*, 2011). Such *for-better-and-for-worse* susceptibility (Belsky, Bakermans-Kranenburg & van IJzendoorn, 2007) implies that children may benefit or suffer differentially from such experiences as nurturance or abuse (Belsky & Pluess, 2009a, 2009b; Boyce *et al.*, 1995), as well as from prevention and intervention efforts (Belsky & van IJzendoorn, 2015; van IJzendoorn, Bakermans-Kranenburg, Belsky, Beach, Brody *et al.*, 2011). It is crucial to recognize that *for better and for worse*, in this context, refers to mental-health outcomes, not fitness payoffs. We note this up front because we will later propose that, in fitness terms, plastic individuals achieve relatively fixed payoffs (rather than variable payoffs) across environmental conditions, because plasticity enables the development of locally adaptive phenotypes (increasing fitness), but also comes at a cost (reducing fitness).

A theoretical question is why between-individual variation in plasticity exists. Explanations of differential plasticity include differences in genes and epigenetic regulation (Belsky *et al.*, 2009; Belsky & Pluess, 2009a, 2009b), as well as differences in prior experiences (Boyce & Ellis, 2005; Ellis *et al.*, 2011; Frankenhuis & Panchanathan, 2011a, 2011b; Pluess & Belsky, 2011). This article focuses on Belsky's proposal (1997, 2005; Belsky & Pluess, 2009a) that, when different levels of plasticity are optimal (in terms of biological fitness) in different environmental states and the environment fluctuates unpredictably, natural selection may favor parents producing offspring with varying levels of plasticity. Before proceeding, a note on terminology: There is no implication that parents deliberately weigh their options after estimating environmental variability and then decide on their offspring's levels of plasticity. Rather, reproductive decisions result from mechanistic processes that are the products of natural selection; organisms need not be consciously aware of these processes. Accordingly, when we refer to parental 'decisions' or 'choices' – for instance, to produce particular proportions of fixed and plastic offspring – we are referring to observable outcomes (epiphenomena) that result from the entire array of causal mechanisms that determine behavior (from molecules to neural networks), including but not limited to cognitive processes.

Mechanisms of bet-hedging

Belsky (2005; Pluess & Belsky, 2009, 2011) stipulated two mechanisms by which parents could diversify their offspring: one genetic by producing genetically diverse offspring, the other experiential through pre- and postnatal regulation of offspring plasticity. Belsky's experiential version of the bet-hedging hypothesis was inspired by the work of Boyce and Ellis (2005), who first proposed that between-individual variation in *biological sensitivity to context* – i.e. reactivity of neurobiological stress systems – can result from differences in experience (discussed below). In this paper, however, we are not primarily concerned with the biological mechanisms that instantiate plasticity, or with the pathways through which parents influence their offspring's levels of plasticity. We provide references here (Belsky & Pluess, 2013; Ellis *et al.*, 2011), and elsewhere in the paper, for readers seeking more information about these mechanisms and processes.

Motivating the model

Our main goal is to present a mathematical model that evaluates the logical coherence of Belsky's (1997, 2005; Belsky & Pluess, 2009a) bet-hedging hypothesis of differential plasticity. Specifically, we examine what are the necessary conditions for natural selection to favor parents to hedge their bets by varying offspring plasticity. The idea that diversification of investments can be adaptive in an unpredictably fluctuating environment is not novel: In both biology and economics, well-developed literatures have addressed this issue, even if psychologists – Belsky's (1997, 2005; Belsky & Pluess, 2009a) intended audience – have not thought in such terms. Economists have focused on the conditions in which investors should diversify their portfolio so as to maximize their profit in a temporally fluctuating market (Bernoulli, 1738, trans. 1954; Markowitz, 1952; Stearns, 2000). Biologists have focused on strategies that optimize organisms' reproductive success in randomly varying environments (reviewed in Childs, Metcalf & Rees, 2010; Donaldson-Matasci, Bergstrom & Lachmann, 2013; Donaldson-Matasci, Lachmann & Bergstrom, 2008; Ellis, Figueredo, Brumbach & Schlomer, 2009; Meyers & Bull, 2002; Simons, 2009, 2011). As we will show below, Belsky's (1997, 2005; Belsky & Pluess, 2009a) version of the bet-hedging argument is formally similar to some classical bet-hedging models from biology (e.g. Moran, 1992; Philippi & Seger, 1989; extended in Starrfelt & Kokko, 2012).

In biology, bet-hedging is typically discussed in the context of fixed traits, which are non-responsive to local conditions. Biologists have shown, for instance, that parents might bet-hedge by producing offspring of variable sizes (Hopper, 1999; Olofsson, Ripa & Jonzén, 2009), or variable developmental delay times (i.e.

diapause; Childs *et al*., 2010; Cohen, 1966). In contrast, Belsky applies the logic of bet-hedging to developmental plasticity itself (i.e. to contexts where trait development depends on local conditions). In this sense, Belsky's hypothesis is qualitatively distinct from existing bet-hedging accounts. If bet-hedging theory can be applied to developmental plasticity, this is interesting and may be empirically fruitful, because plasticity is a property of many physiological systems in many species (Fischer, Van Doorn, Dieckmann & Taborsky, 2014). Moreover, developmental plasticity is a basic property of many mechanisms of the human mind and hence a long-standing focus of inquiry in psychology in general (e.g. the effects of early-life experience on later-life outcomes) and developmental psychology in particular.

Our contribution is to capture and analyze Belsky's hypothesis within a formal specialist-generalist, bet-hedging framework. We conceptualize low-plasticity strategies (which produce the same phenotype despite variable environmental conditions) as *specialists* adapted to a particular niche, and high-plasticity strategies (which produce different phenotypes in different environmental conditions) as *generalists* across niches (see Wilson & Yoshimura, 1994). This interpretation roots developmental psychologists' growing interest in differential plasticity within a well-developed body of bet-hedging theory from evolutionary biology, and it could facilitate integration between models of bet-hedging and models of frequency-dependent selection that include specialists and generalists (Ellis, Jackson & Boyce, 2006; Ellis *et al*., 2011; Wilson & Yoshimura, 1994). We regard this as important given the general lack of consideration of evolutionary principles – and of evolutionary modeling in particular – in the field of developmental psychology, which hinders much-needed theoretical integration with – and consilience across – the life sciences. Our work might also inspire biologists to consider between-individual differences in plasticity as a product of bet-hedging.

Applying the logic of bet-hedging to plastic traits is reasonable when (a) phenotypic development is not fully reversible and (b) individuals use imperfect cues to estimate the environmental state, resulting in maladaptive developmental programming in some individuals. If both conditions hold, then within any generation some individuals develop phenotypes that are well matched to the environment, and others do not. On Belsky's (1997, 2005; Belsky & Pluess, 2009a) account, well-matched individuals are those whose genetic composition is well suited to the environment and/or those whose early-life cues accurately predicted their adult environment; mismatched ones are those whose genetic composition is poorly suited to the environment and/or those whose early-life cues failed to predict their adult environment.

Before presenting the model, we first chronicle individual differences in susceptibility to environmental influences (for more extensive review, see Aron, Aron & Jagiellowicz, 2012; Bakermans-Kranenburg & van IJzendoorn, 2007, 2011; Belsky & Pluess, 2009a; Kim-Cohen, Caspi, Taylor, Williams, Newcombe *et al*., 2006), including related theory. Because our main goal is to formalize and analyze Belsky's (1997, 2005; Belsky & Pluess, 2009a) bet-hedging hypothesis of differential plasticity, we will not compare theories (see Ellis *et al*., 2011).

From risk alleles to plasticity genes

The notion that individuals differ in their susceptibility to environmental effects has a long history in psychiatry and psychology (Belsky & Pluess, 2009a). In fact, most gene–environment interaction (G×E) research conducted over the past decade has been informed by the so-called diathesis-stress or dual-risk framework (Zuckerman, 1999), which stipulates that some individuals are especially susceptible to *negative* effects of contextual *adversity*. The focus of the differential-susceptibility model, including G×E, differs from the diathesis-stress framework. Whereas the latter focuses exclusively on vulnerability, the former stipulates that some individuals are not just more vulnerable to contextual adversity, but also *benefit* more from *supportive* environmental conditions. This appears to be the case for some polymorphisms long regarded as 'vulnerability genes' or 'risk alleles', leading to the proposal that they be regarded as 'plasticity genes' instead (Belsky *et al*., 2009) – with 'plasticity' referring to phenotypic outcomes, not fitness payoffs (as noted earlier, we will argue below that plastic individuals attain moderate, not variable, fitness payoffs). Notably, two recent meta-analyses of G×E research support the differential susceptibility theorizing: one focused on dopamine-related genes carried by children 10 years of age or younger (Bakermans-Kranenburg & van IJzendoorn, 2011), and the other on the *5-HTTLPR* polymorphism in Caucasian children under 18 years of age (van IJzendoorn, Belsky & Bakermans-Kranenburg, 2012). Both of these meta-analyses indicate that children carrying putative plasticity alleles are more susceptible to both the negative effects of environmental adversity and the positive effects of social support (in terms of mental health).

Importantly, recent experimental human intervention work that involves randomly assigning individuals to alternative contextual conditions (Belsky & Van IJzendoorn, 2015), thereby overcoming the risk that G×E findings are an artifact of gene–environment correlation,

provides additional support to the claim that some long-regarded vulnerability genes function as plasticity genes (for review, see Belsky & Pluess, 2013). Indeed, a recent meta-analysis of experimental interventions designed to promote well-being while chronicling truly causal environmental effects reveals that DRD4 and 5-HTTLPR function as plasticity genes, such that carriers of certain allelic variants of these polymorphisms benefit more – in mental-health terms – from these efforts (e.g. to promote early literacy, to prevent teen alcohol abuse) than do others (van IJzendoorn & Bakermans-Kranenburg, 2015). From an evolutionary perspective, two hypotheses have been proposed to explain genetic variation in plasticity that undergirds, by hypothesis, individual differences in susceptibility.

Frequency-dependent selection

One hypothesis posits that frequency-dependent selection maintains genetic variation in plasticity (Ellis *et al.*, 2006, 2011; Wilson & Yoshimura, 1994; see also Wolf, van Doorn & Weissing, 2008, 2011). Frequency-dependent selection refers to conditions in which a phenotype's fitness is dependent on its frequency relative to other phenotypes in a population. Individual differences in plasticity can persist due to frequency-dependent selection when plastic phenotypes attain higher fitness than less-responsive phenotypes in a population composed mostly of the latter – and vice versa (Ellis *et al.*, 2006, 2011; Wilson & Yoshimura, 1994).

Parental bet-hedging

The current article focuses on the bet-hedging hypothesis of differential plasticity (Belsky, 1997, 2005; Belsky & Pluess, 2009a). This hypothesis states that it might be adaptive for parents to produce offspring with varying levels of developmental plasticity because different levels of plasticity may be optimal in different environmental states. Since the future is uncertain, natural selection might favor parents who spread their investments and associated risk by producing offspring that vary in their sensitivity to environmental influences, including parenting (Belsky, 1997, 2005; Belsky & Beaver, 2011; Ellis *et al.*, 2011; Pluess & Belsky, 2010, 2013; see also Figueredo & Wolf, 2009).

Experiential regulation of plasticity

As noted, between-individual variation in plasticity may result from between-individual differences in prior experiences (Boyce & Ellis, 2005; Del Giudice, Ellis & Shirtcliff, 2011; Ellis *et al.*, 2011; Pluess & Belsky, 2011). This view is central to Boyce and Ellis' (2005) theory of Biological Sensitivity to Context (BSC), which regards physiological reactivity as a contextually regulated plasticity factor.

Biological sensitivity to context

Boyce and Ellis (2005) argue that individuals growing up under extreme environmental conditions may benefit from developing heightened BSC (Ellis & Boyce, 2008; Ellis, Essex & Boyce, 2005; Ellis *et al.*, 2011). Such heightened reactivity can augment vigilance to dangers and threats in stressful environments and enhance the benefits derived from support and care in protective ones (Del Giudice *et al.*, 2011; Ellis *et al.*, 2006). In environments with an intermediate level of stress, individuals may down-regulate reactivity, thus avoiding the costs associated with persistently elevated levels of physiological reactivity when the benefits do not outweigh the cost (Ellis & Boyce, 2008).

Prenatal programming of plasticity

A related view, developed by Belsky (1997, 2005; Belsky & Pluess, 2009a; Belsky *et al.*, 2007), stipulates that negative emotionality in infants and young children – a known correlate of physiological reactivity – also functions as a plasticity factor. Children manifesting high levels of negativity (e.g. fear, distress, inhibition) prove not just more susceptible to the adverse effects of negative environments (e.g. harsh parenting, maternal depression), but also benefit more from supportive ones (in terms of mental health, not fitness). Children's developmental experiences, including exposure to prenatal stress (e.g. Huizink, Bartels, Rose, Pulkkinen, Eriksson *et al.*, 2008; O'Connor, Ben-Shlomo, Heron, Golding, Adams *et al.*, 2005; Pesonen, Räikkönen, Strandberg & Järvenpää, 2005), appear to influence both their physiological reactivity (e.g. Claessens, Daskalakis, van der Veen, Oitzl, de Kloet *et al.*, 2011; Ellis & Boyce, 2008; Kaiser & Sachser, 2009), even when measured in adulthood (Heim, Newport, Wagner, Wilcox, Miller *et al.*, 2002), and their negative emotionality (Belsky, Fish & Isabella, 1991).

Consistency in early experiences

Frankenhuis and Panchanathan (2011a, 2011b) recently proposed a third experiential process that may contribute to individual differences in plasticity: stochastic sampling. In some developmental domains, organisms may face a tradeoff between sampling more cues to the environmental state and tailoring their phenotypes to

local conditions. In these domains, some individuals may receive a homogeneous sample set, resulting in a confident estimate about the environmental state, leading such individuals to specialize early in life, and thereby sacrifice plasticity (assuming that phenotypic development is not fully reversible). In contrast, others may receive a heterogeneous set of cues, resulting in a less confident estimate, leading them to defer 'phenotypic commitment' (in order to avoid mismatch), keep sampling, and specialize later. As a consequence, individuals may come to differ in their levels of plasticity.

Developing the model

In building a model of Belsky's (1997, 2005; Belsky & Pluess, 2009a) bet-hedging argument of differential plasticity, our first challenge is to translate *for-better-and-for-worse* outcomes, which are typically defined in terms of mental health, quality of life, and social desirability, into biological fitness (Belsky, 2008; Ellis, Del Giudice, Dishion, Figueredo, Gray *et al*., 2012; Frankenhuis & Del Giudice, 2012; Frankenhuis & de Weerth, 2013; Manuck, 2010). In biology, traits are considered beneficial to organisms to the extent that they increase individuals' relative survival and reproductive success. In contrast, developmental psychologists tend to view distressing or socially undesirable behavior as inherently maladaptive, and behaviors enhancing well-being and social integration as inherently adaptive. These different notions of 'adaptive' are conceptually orthogonal: desirable behavior may (but need not) enhance reproduction, and fitness-enhancing behavior may (but need not) have desirable features.

Fitness payoffs

Individuals who are relatively sensitive to their environment are 'plastic'. They adaptively match their phenotypes to local conditions more than their peers, developing danger-adapted phenotypes (e.g. high stress) in dangerous environments and safe-adapted phenotypes (e.g. low stress) in safe environments. Whereas danger-adapted phenotypes are considered maladaptive from a mental-health perspective, but biologically adaptive in a dangerous environment, safe-adapted phenotypes in a safe environment are considered adaptive from an evolutionary as well as a mental-health perspective. From a fitness viewpoint, this raises a question: If plastic individuals match their phenotypes to local conditions, shouldn't they attain the same or higher fitness than individuals who do not adjust development (i.e. fixed types), hence outcompete them?

This depends on the costs to plasticity, which might include (a) phenotypic–environment mismatch resulting from prediction error (i.e. during developmental programming, cues may imperfectly indicate current or future environmental states, resulting in a suboptimal phenotype; Donaldson-Matasci *et al*., 2013; Nettle, Frankenhuis & Rickard, 2013; Rickard, Frankenhuis & Nettle, 2014), (b) constitutive costs (e.g. energy required for building and maintaining the neural-cognitive machinery required for plasticity), (c) information search costs (e.g. time spent sampling environmental cues), and (d) a lower degree of phenotypic integration (e.g. add-ons may be less effective than the same phenotypic element integrated early in development) compared with fixed phenotypes that specialize from birth to fit a particular environmental state (for reviews, see Auld, Agrawal & Relyea, 2010; DeWitt, Sih & Wilson, 1998; Relyea, 2002).

We will not consider these costs in detail here, but assume that there is *some* cost to plasticity. This cost is such that within any generation the fitness of plastic individuals is lower than that of (fixed) specialists matching the environmental state. However, the cost of plasticity is low enough for the fitness of plastic individuals to be higher than that of (fixed) specialists not matching the environmental state. We capture this idea in our model by assuming that plastic individuals accrue a fitness of 1 in each environment, which is intermediate between that of specialists who match the environmental state – and attain a fitness payoff of $1 + b$ – and that of specialists who do not match the environment and thus attain a payoff of $1-c$. We assume that all plastic individuals attain a fitness of 1 (instead of some attaining $1 + b$ and others $1-c$) because we want to ensure that plastic individuals, as a group, are always situated in between matched and mismatched specialists. Plastic individuals are thus 'generalists', sacrificing specificity for breadth (Wilson, 1994; Wilson & Yoshimura, 1994).

In contrast, specialists are less malleable, adapting their phenotypes less to context, developing relatively similar phenotypes even in different environments (compared with plastic individuals). When their phenotype matches the environmental state, they thrive because they achieve a viable phenotype–environment fit without paying a cost for plasticity; in this case, specialists attain higher fitness ($1 + b$) than plastic individuals. However, when the phenotypes of specialists are not well matched to the environment, they suffer, attaining lower fitness ($1-c$) than plastic individuals. Specialists thus sacrifice breadth for specificity (Wilson, 1994; Wilson & Yoshimura, 1994).

Spatial and temporal environmental variation

The bet-hedging hypothesis of differential plasticity (Belsky, 1997, 2005; Belsky & Pluess, 2009a) is based on the idea that parents cannot predict with certainty what environmental state their offspring will experience. However, environments can be unpredictable in different ways, and these may result in different selection pressures, hence different adaptations (in our case, different offspring compositions; see below). Here we examine two kinds of environmental variation well known in evolutionary biology: *spatial* and *temporal* environmental variation (some use the terms *individual-level* and *population-level*; e.g. Bergstrom & Godfrey-Smith, 1998; Donaldson-Matasci *et al.*, 2008); we do not analyze their combination (see, e.g. Carja, Furrow & Feldman, 2014). The appropriate fitness calculations under spatial and temporal environmental variation constitute a rich field of inquiry in biology; such calculations depend on the intricacies and degree of realism of assumptions about organisms and their physical and social environments. In this article, we will discuss just two stylized scenarios. We recommend the following works to readers seeking more information or interested in developing more refined follow-up models (Frank, 2011; Leimar, 2009; McNamara, Trimmer, Eriksson, Marshall & Houston, 2011).

Spatial environmental variation

In environments that vary exclusively spatially (and not temporally), the world is divided into different patches, each with a particular state (e.g. dangerous or safe). Within a given generation, any offspring may develop in either patch, and the associated probabilities are equal for all individuals (i.e. juveniles disperse from their natal patch and settle on a new patch at random); some offspring will develop in one patch (e.g. safe), others in another patch (e.g. dangerous), and so forth (if there are more than two patches). Across generations, however, individuals always face the same spatial distribution, and so parents attain the same expected fitness within each generation – and thus also across generations; that is, there is no variance in parents' fitness across generations.[1] In this scenario, parents' fitness can be computed as the arithmetic mean of the fitness of all their offspring[2]: the fraction of offspring in the safe patch multiplied by their fitness, *plus* the fraction of offspring in the dangerous patch multiplied by their fitness (and so forth, if there are more patches). This calculation implies that even if *some* offspring attain low fitness (e.g. they die), parents might still do well, depending on the fitness attained by their other offspring.

Temporal environmental variation

In contrast, in environments that vary only temporally (and not spatially), within a single generation all individuals confront the same environmental state (e.g. dangerous); however, in the next generation, their offspring may face a different state (e.g. safe). Thus, if the environment fluctuates temporally, the entire population experiences variation across generations. In such environments, long-term fitness depends on the fitness of one generation, multiplied by the fitness of the next generation, and so forth. The average fitness of this series will not be the arithmetic mean, but the geometric (i.e. multiplicative) mean – the n-th root of the product of n fitness values[3] (Dempster, 1955; King & Masel, 2007; Lewontin & Cohen, 1969). Unlike with spatial variation, fluctuations in success can be catastrophic when the environment varies temporally. If an entire set of offspring is mismatched to its environment in one generation and fails to reproduce, this lineage will be wiped out.

Formal definition of bet-hedging

Variance in fitness across environments lowers the geometric mean, but not the arithmetic mean. Indeed, the geometric mean is often approximated by the arithmetic mean minus a variance term – most commonly, the variance divided by two times the arithmetic mean[4] (Frank, 2011; Starrfelt & Kokko, 2012; Stearns,

[1] This expectation being identical across generations assumes that genotypes produce an infinite number of offspring. If genotypes produce a finite number of offspring (as they do in reality), they will experience variance in fitness across generations (Starrfelt & Kokko, 2012). Because this variance becomes very small at even modest population sizes (Hopper, Rosenheim, Prout & Oppenheim, 2003), we will ignore it here.

[2] We assume discrete, non-overlapping generations, which consist of a single selective life stage: organisms are born and reproduce; mature individuals die; and the cycle repeats. Parents and offspring do not coexist (see also General Discussion section).

[3] This assumes an infinitely large and well-mixed population (Starrfelt & Kokko, 2012). When populations are finite, variance in offspring number reduces fitness in proportion to the inverse of the population size (see Frank & Slatkin, 1990, Gillespie, 1974, and Proulx & Day, 2001, for discussions of geometric mean fitness in finite populations, particularly small ones; for a discussion of the evolution of bet-hedging in large, structured populations, see Lehmann & Balloux, 2007).

[4] This approximation assumes that the fitness of individuals does not deviate much from the average fitness of their genotype within a given generation (Starrfelt & Kokko, 2012). If it does, a structurally similar, albeit less succinct, approximation may be preferable.

2000; see Young & Trent, 1969, for other approximations). The long-term fitness of genotypes thus depends not only on their immediate (arithmetic) expectation for the next generation, but also on variance in their fitness across generations (Dempster, 1955). This insight formed the foundation of bet-hedging theory in biology (Slatkin, 1974). A mean–variance tradeoff exists if an increase in a strategy's short-term (i.e. next-generation) expected arithmetic mean also increases its long-term (i.e. across generations) expected fitness variance. Bet-hedging strategies are those which, when confronting this tradeoff, sacrifice short-term fitness to reduce long-term fitness variance (Seger & Brockmann, 1987; Philippi & Seger, 1989).

Diversified vs. conservative bet-hedging

We will focus on situations in which parents hedge their bets by producing diverse offspring (i.e. a mixture of types); this is known as 'diversified' bet-hedging (captured by the idiom: 'Don't put all your eggs in one basket'). Formally, 'bet-hedging' applies to any strategy that increases geometric mean fitness while sacrificing short-term arithmetic mean fitness. If, for instance, parents are selected to produce only plastic offspring in order to reduce variation in fitness, despite a reduction in short-term arithmetic mean fitness, this is bet-hedging, too; it is called 'conservative' bet-hedging (captured by the idiom: 'A bird in the hand is worth two in the bush') because individuals avoid extreme payoffs.

Diversified bet-hedging can be instantiated in different ways: by producing genetically diverse offspring, or by producing genetically homogenous offspring each of which randomly develops a phenotype (as if drawn from a fixed probability distribution). The latter is called 'adaptive coin flipping' because, metaphorically, each individual flips a coin to determine what phenotype he or she will develop: e.g. specialist or generalist (Cooper & Kaplan, 1982; Kaplan & Cooper, 1984; Salathé, Van Cleve & Feldman, 2009). We defer further discussion of instantiation to the General Discussion section.

A gambling metaphor

Before introducing the mathematical model, we introduce a gambling metaphor in order to introduce key concepts, develop intuitions, and preview results. Imagine a casino with a large number of identical roulette wheels. All wheels are spun simultaneously throughout the night (e.g. every five minutes). Each spin of the wheels corresponds to a generation and the outcome of a spin corresponds to the environmental state experienced by developing offspring in the model we later present. Each roulette wheel has a certain number of red pockets and a certain number of black pockets, where black pockets correspond to a safe environment and red pockets to a dangerous environment (we ignore numbers associated with different pockets). The fraction of black pockets is given by the probability p. For example, if $p = .75$ there are three times as many black pockets as red pockets for each and every roulette wheel in the casino.

Next to each wheel is a felt table with three squares, one black, one red, and one white. Before each spin, gamblers place their chips on these squares. A gambler represents a parent and the set of all gamblers in the casino represents an evolving population of parents. Each chip represents a child. Just as a gambler chooses to bet on black, red, or white, a parent can have a child that is a safe-specialist, a danger-specialist, or a generalist. A chip bet on the white square returns back that same chip regardless of the outcome of the spin (i.e. no gain, no loss). A chip bet on the black square returns $1 + b$ chips if the wheel comes up black (where b represents a fraction of one chip, varying between 0 and 1) and $1-c$ chips if the wheel comes up red (where c represents a fraction between 0 and 1). Similarly, a chip bet on red returns $1 + b$ chips if the outcome is red and $1-c$ if the outcome is black.

Gamblers must bet all of their chips every time the wheels spin (i.e. they cannot reserve chips, though betting on white results in the same outcome). Each gambler has a strategy she uses in placing her bets. Some strategies will, over time, do better than other strategies. And we might expect that most gamblers end up deploying similar strategies. There is, after all, a uniquely best strategy for any combination of values for p, b, and c. Note: we chose roulette as a metaphor, rather than poker, because our model assumes that the payoff of a strategy is not dependent on its frequency relative to other strategies in the population. We leave an extension of our model that incorporates frequency-dependent selection – in which parents are playing games not only against nature, but against each other as well – for a future study.

On some nights, the casino limits gamblers such that they can only bet one chip on each wheel every time the wheels spin (e.g. if a gambler has 20 chips, she must place 20 bets on 20 different roulette wheels). On other nights, the casino forces gamblers to place all of their chips on just one wheel each time the wheels spin (e.g. the gambler must now place all 20 of her chips on just one of the roulette wheels).

In the first version, in which separate bets are placed on separate wheels, a gambler does best by betting all of

her chips on just one color (i.e. red, black, or white); she should never bet on different colors. To understand why, we need to calculate the expected return in any round for betting on each of the three colors. If a gambler bets on black, her expected payoff is $1 + pb - (1-p)c$ (see Appendix for details). When this expectation is greater than 1, the gambler will, over time, make more money betting on black than betting on white, which is the safe bet. If, instead, a gambler bets on red, her expected payoff is $1 + b(1-p) - pc$. Again, if this is greater than 1, she will make more money, on average, by betting on red than on white. If both of these expectations (i.e. betting on red and betting on black) are less than 1, the gambler does poorly betting on either black or red; her best play is to bet on white and preserve her chips. If one expectation (e.g. betting on red) is greater than 1 and the other expectation less than 1 (e.g. black), she should bet on the color that has an expected rate of return greater than 1. And if both are greater than 1, she makes the most money betting on the color with the higher expected rate of return. In this game, because gamblers bet each chip on a separate wheel and because the outcomes across wheels are independent, losing at any particular wheel is no big deal, especially when betting a large number of chips each time. What matters, in the long run, is the arithmetic average across wheels. In this case, that average is maximized by betting on just one color, depending on the values of p, b, and c.

Things are less simple in the second version of the game, in which a gambler must place all of her chips on just one wheel each time the wheels spin. If, for example, she places all of her chips on the red square and black comes up, she stands to lose money; the larger the value of c, the more the gambler loses. When $c = 1$, the most it can be, the gambler is wiped out. Rather than placing all of her chips on a single color, she must be more cautious in this version of the game, especially when the value of c is large, hedging her bets to insure against catastrophe. In this type of game, a gambler seeks to maximize the geometric mean payoff, not the arithmetic mean (see above section Spatial and Temporal Environmental Variation). To maximize the geometric mean, the gambler must take into account variation in her payoffs across gambles. How to maximize the geometric mean exactly depends on the values of p, b, and c.

To preview results for this second version of the game (which we describe in more detail below), when the benefit of a correct bet exceeds the cost of an incorrect bet ($b>c$), a gambler does best by placing all her chips on the black square, all her chips on the red square, or distributing her chips between black and red squares; she should never place any chips on the white square. The precise distribution between red and black depends on the values of p, b, and c. When the cost of being wrong exceeds the benefit of being correct ($c>b$), a gambler sometimes does best by placing some chips on the white square and the remainder on either the black square (if $p > .5$) or the red square (if $p < .5$), but never both. These betting strategies ensure a good payoff without suffering catastrophic failure.

This gambling metaphor has fundamental similarities with the problem faced by parents in choosing the developmental strategies of their offspring when the environment fluctuates across time and space. The nights in which gamblers can bet only one chip per roulette wheel corresponds to a purely spatially varying ecology, whereas the nights in which gamblers must place all their chips on just one wheel corresponds to a purely temporally varying ecology.

The mathematical model

In our model, parents can produce *plastic* offspring (generalists), who can adapt their phenotype to local conditions, attaining a payoff of 1 in each of the two environmental states. Parents can also produce *fixed* offspring (safe- and danger-specialists), who are less able to adapt to local conditions, attaining high payoffs (relative to generalists) in environments matching their phenotypes ($1 + b$), and low payoffs (relative to generalists) in environments not matching their phenotypes ($1-c$). We assume that b and c range between 0 and 1; this assumption ensures that mismatched specialists do not attain negative fitness, and implies that well-matched specialists can at most attain double the fitness of generalists. This assumption is justified because empirical work shows that fitness effects of natural phenotypic variation virtually always fall within this range in humans (reviewed in Keller & Miller, 2006; Nettle & Pollet, 2008; Penke, Denissen & Miller, 2007; Stearns, Byars, Govindaraju & Ewbank, 2010), as well as other organisms (reviewed in Endler, 1986; Hoekstra, Hoekstra, Berrigan, Vignieri, Hoang *et al.*, 2001; Kingsolver, Hoekstra, Hoekstra, Berrigan, Vignieri *et al.*, 2001; Morrissey & Hadfield, 2012; Siepielski, Gotanda, Morrissey, Diamond, DiBattista *et al.*, 2013).

Optimal offspring distribution

Our goal is to compute the fraction of safe-specialists (x), danger-specialists (y), and plastic individuals (z) that natural selection favors parents to produce. Each of these

fractions ranges between 0 and 1, and all three must sum to 1. We are particularly interested in the region where $0 < z < 1$, because there natural selection favors parental bet-hedging via the production of both fixed and plastic offspring. The optimal values of x, y, and z will depend on features of the evolutionary ecology; that is, on the probabilities of a safe (p) and dangerous environment ($1-p$), and the magnitude of fitness effects (b and c). These fitness effects specify how much better or worse specialists do compared with generalists if their phenotypes match or do not match the environmental state. We assume that environmental parameters (p, b, and c) are extrinsic, meaning that parents and offspring cannot control them. We leave an extension of our model that incorporates developmental niche construction (Flynn, Laland, Kendal & Kendal, 2013) – in which parents and/or offspring can influence environmental parameters – for a future study.

For simplicity, we assume that safe-specialists in a safe environment attain the same fitness as danger-specialists in a dangerous environment (namely, $1 + b$). We also assume that safe-specialists in a dangerous environment attain the same fitness as danger-specialists in a safe environment (namely, $1-c$). This symmetry in payoffs justifies limiting our analyses to cases where $p \geq 1-p$ (with $p < 1-p$, our results would be the same, flipping x for y). However, a future study should extend our model to include asymmetric fitness payoffs, where the benefits and costs of being well matched or mismatched vary by environmental state; such asymmetries might severely reduce the scope for bet-hedging to evolve (Salathé *et al.*, 2009).

Results with spatial environmental variation

When the environment varies spatially (but not temporally), parental fitness (w) is given by equation 1 (see Appendix for details):

$$w = 1 + x[pb - c(1-p)] + y[b(1-p) - pc] \qquad (1)$$

The 1 just to the right of the equal sign represents the payoff achieved by a generalist. If parents only produce generalists, they will achieve a fixed payoff of 1. The second term, $x[pb - c(1-p)]$ represents the change in fitness when producing safe-specialists instead of generalists, in which x represents the fraction of safe-specialists among the offspring, pb the benefit of safe-specialists developing in a safe environment, and $c(1-p)$ the cost of safe-specialists developing in a dangerous environment. The third term, $y[b(1-p) - pc]$, represents the change in fitness when producing danger-specialists instead of generalists, and has a similar interpretation to the second term. The sum of these terms represents the arithmetic mean of fitness, averaging across the three types of offspring.

If both the second and third terms are negative, safe- and danger-specialists do worse, on average, than generalists. In this case, parents maximize fitness by producing all generalists ($z = 1,\ x = 0,\ y = 0$). If the second term is positive and the third negative, safe-specialists do better, on average, than generalists, who do better, on average, than danger-specialists. In this case, parents should produce all safe-specialists ($z = 0$, $x = 1$, $y = 0$). If the second term is negative and third term positive, danger-specialists do best and parents should only produce them ($z = 0$, $x = 0$, $y = 1$). Finally, if both the second and third terms are positive, generalists do worse than safe- and danger-specialists. If one of these specialists has even a slight edge over the other, parents should exclusively produce the specialist with the edge ($x = 1$, $y = 0$ or $x = 0$, $y = 1$).

As with the version of roulette in which gamblers can only bet one separate chip on each roulette wheel, when environmental variation is only spatial, parents maximize their fitness by either producing all safe-, all danger-specialists, or all generalists, and never a mixture.[5] With spatial variation, we expect no variation in plasticity due to bet-hedging.

Results with temporal environmental variation

When the environment varies temporally (but not spatially), selection never favors a mixture of all three types (see Appendix for details). Instead, selection favors the production of either only one type (safe-specialist, $x = 1$; danger-specialist, $y = 1$; or generalist, $z = 1$) or a mixture of two types (safe-specialists and danger-specialists, $x+y = 1$; safe-specialists and generalists, $x+z = 1$; or danger-specialists and generalists, $y+z = 1$). Because we assume that $p \geq 1-p$, the mixture between danger-specialists and generalists is never favored. So, for mixtures, we have just two strategies: safe-specialists and generalists (henceforth, 'differential plasticity'), or safe-specialists and danger-specialists (henceforth, 'mixture of specialists').

[5] Levene (1953) showed mathematically that spatial environmental variation with local population regulation maintains genetic polymorphisms (for related theory, see Frank & Slatkin, 1990; Maynard Smith & Hoekstra, 1980; Seger & Brockmann, 1987; for related empirical work, see Kawecki & Ebert, 2004; Savolainen, Lascoux & Merilä, 2013). We do not consider this case here, because the genetic polymorphisms that evolve with local population regulation do not result from a mean–variance tradeoff, and therefore do not qualify as bet-hedging (see the section on Formal Definition of Bet-hedging).

With temporal variation, differential plasticity can sometimes be favored. In the Appendix, we provide analytical results. Figure 1 depicts the regions of parameter space in which differential plasticity is uniquely favored; that is, where it attains higher, not lower or equal, fitness than all of the other strategies (see also figures in Appendix).

Consistent with previous findings from biology (Donaldson-Matasci *et al.*, 2008; Moran, 1992; Starrfelt & Kokko, 2012), natural selection only favors bet hedging when fitness effects are large (i.e. the variation in fitness associated with different types must be large), in order to reduce costly (or even catastrophic) variance in fitness. We also report a novel result: Natural selection only favors differential plasticity when the cost of being mismatched exceeds the benefit of being well adapted ($c>b$; Figure 1; see also figures in Appendix). We reflect on these results below.

When benefits exceed costs

When the benefit of being well matched is larger than the cost of being mismatched ($b>c$), selection never favors differential plasticity ($x+z = 1$). Instead, selection favors producing either all safe-specialists ($x = 1$) or a mixture of specialists ($x+y = 1$). Producing all safe-specialists is favored when the benefit of being well matched and the cost of being mismatched are small ($b,c << 1$) or the probability of experiencing the safe environment is high. With small fitness effects, fitness variance is also small, so we are back to a world that is approximately like spatial variation; selection favors producing only the type that has the highest arithmetic mean fitness. When p is high, there is little point in producing a second type to capture some benefits in the rare environment; individuals do best by producing only the type that is well matched to the common environment, despite suffering the occasional loss. When the benefit and cost are large ($b,c >> 0$) and p is not that high, selection favors producing a mixture of specialists. A mixture of specialists does better than only safe-specialists because the mixture experiences much less variation in fitness across time. And when the environment varies temporally, what matters is geometric mean fitness, not arithmetic mean fitness. A high variance in payoff reduces the geometric mean.

Focusing on the region in which selection favors a mixture of specialists, we can ask why a mixture of specialists beats differential plasticity (see the Appendix for a proof). In this region, when p is not that high and b, $c >> 0$, producing only safe-specialists results in too much exposure to risk. When the environment is dangerous, safe-specialists do very poorly. To mitigate this risk, selection favors mixing safe-specialists with another type, either danger-specialists or generalists. When the benefit of being well matched exceeds the cost of mismatch, this other type should be danger-specialists.

To understand why, we can think about the effect of the two kinds of mixing on the variance in fitness across environments. With a mixture of specialists, there is always a mix that results in the same or nearly identical payoffs in each environment (see figures in Appendix); this is true even if the environmental state is highly variable (e.g. $p = .6$). With differential plasticity, there is no way of mixing safe-specialists and generalists to eliminate variance in payoff across environments; the

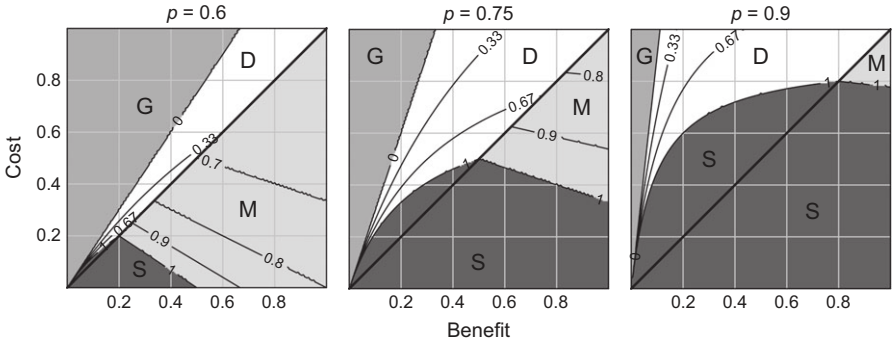

Figure 1 *The x-axis represents the additional benefit (b) fixed specialists obtain, compared with plastic generalists, if their phenotype matches the environmental state. The y-axis represents the additional cost (c) fixed specialists incur, compared with plastic generalists, if their phenotype does not match the environmental state. Contour lines indicate the optimal fraction of safe-specialists, when the probability of a safe environment equals 0.6, 0.75, and 0.9 (from left to right). In regions denoted 'S' (shaded dark gray), only safe-specialists are favored; if 'G' (shaded medium gray), pure generalists; if 'M' (shaded light gray), mixtures of specialists (i.e. safe- and danger-specialists); if 'D' (not shaded), differential plasticity (i.e. mixtures of generalists and safe-specialists). If b > c, differential plasticity is never favored. If c > b, differential plasticity can be favored, especially if c is large.*

only way to eliminate fitness differences in the two environments is to produce all generalists. Mixtures of specialists can always reach lower variance in payoffs than differential plasticity, which all else being equal (e.g. the arithmetic mean) results in higher geometric mean fitness.

When costs exceed benefits

When the cost of mismatch exceeds the benefit of being well matched ($c>b$), selection sometimes favors differential plasticity. When the magnitude of benefit and cost are small ($c,b << 1$), selection favors producing only generalists when p is below some threshold (the precise value of this threshold depends on the values of b and c). When the magnitude of fitness effects is large ($c,b >> 0$), selection often favors differential plasticity over a mixture of specialists.

To understand why, we can think about gains and losses. With differential plasticity, the safe-specialists do somewhat better than generalists in the more common safe environment and a lot worse in the more rare dangerous environment. With a mixture of specialists, the safe-specialists do reasonably well in the safe environment and a lot worse in the dangerous environment; the danger-specialists do reasonably well in the dangerous environment and very poorly in the safe environment. Because the cost exceeds the benefit, differential plasticity is exposed to smaller losses than a mixture of specialists. As a result, differential plasticity experiences less variance in payoffs across environments compared to a mixture of specialists, thereby achieving higher geometric mean fitness.

General discussion

We presented a mathematical model in order to examine whether individual differences in plasticity could result from parental bet-hedging. Our results support the hypothesis' logical coherence in that there are ecological scenarios in which natural selection might favor differential plasticity. However, three conditions must simultaneously hold. First, environmental variation must occur temporally, not exclusively spatially. Second, fitness effects must be large (i.e. variation in individuals' plasticity is correlated with substantial variation in fitness). Both of these conditions are consistent with other biological models of bet-hedging (Donaldson-Matasci *et al.*, 2008; Moran, 1992; Starrfelt & Kokko, 2012). Third, the costs of being mismatched must exceed the benefits of being well matched. To our knowledge, this result is novel and may be specific to the evolution of differential plasticity. If these three conditions are met, our model can account for differential plasticity (i.e. the coexistence of plastic generalists and fixed specialists). However, our model cannot account for a coexistence of plastic generalists and multiple fixed specialists (i.e. different specialists adapted to different environments). Including density-dependence in the model will likely change this result (see Kawecki & Ebert, 2004; Savolainen *et al.*, 2013; Wilson & Yoshimura, 1994); however, a future study should formally examine this.

Empirical predictions

Before discussing the plausibility of large fitness effects, we will first derive empirical predictions. We will focus on two predictions, which apply to contemporary populations only to the extent that individuals inhabit environments that share properties with those environments in which parental bet-hedging may have originally evolved (i.e. no evolutionary disequilibrium).

First, if parental bet-hedging explains differential plasticity, we should expect large fitness consequences of variation in plasticity. For example, at a given level of environmental harshness, danger-specialists should attain much higher fitness than plastic generalists who should attain much higher fitness than safe-specialists. Measuring fitness (even proxies) is difficult in long-lived organisms such as humans, but it is not impossible. For instance, fieldwork on Yanomamö Indians of Amazonas indicates that 88% of *unokai* (men who have killed) sired at least one offspring, compared with 49% of non-*unokai* (Chagnon, 1988; see also Walker & Bailey, 2013). Accordingly, among the Yanomamö, an individual who develops a danger-adapted phenotype (e.g. physical strength, high levels of vigilance) might be more likely (than conspecifics who develop safe-adapted phenotypes) to become *unokai*, and less likely to become a victim, thus garnering fitness benefits. However, for selection to potentially favor differential plasticity (as opposed to only danger-specialists), at other times safe-specialists would need to have a large advantage. In those periods, selection pressures may resemble those faced by the Waorani of Ecuador, among whom more aggressive men have fewer children surviving to reproductive age (Beckerman, Erickson, Yost, Regalado, Jaramillo *et al.*, 2009). Moreover, for differential plasticity to beat mixtures of specialists, in times when aggression is favored, the benefits of aggression must be smaller than its costs in times when it is disfavored.

Second, we expect the extent to which individuals differ in their plasticity levels to depend on: (a) environmental variance and (b) the cost–benefit ratio (i.e. the extent to which the costs of being mismatched exceed the

benefits of being well matched). Specifically, populations experiencing larger environmental variance should include more plastic generalists, and fewer specialists, than populations experiencing smaller environmental variance (Figure 1; for evidence of bet-hedging plants producing proportions of offspring types that track frequencies of environmental states, see Graham, Smith & Simons, 2014, and Rajon, Desouhant, Chevalier, Débias & Menu, 2014; for evidence of bet-hedging plants producing proportions of offspring types that track *both* frequencies of environmental states *and* ontogenetic information, see Sadeh, Guterman, Gersani & Ovadia, 2009, and Simons, 2014). Further, we expect the proportion of plastic generalists to be larger, and the proportion of specialists to be smaller, in those populations in which the cost–benefit ratio is greater (Figure 1), as larger mismatch costs penalize specialists. Having stated predictions, we now turn to the question whether the conditions our results point to plausibly pertain to human evolutionary history.

Plausibility of large fitness effects

Our results indicate that selection can favor differential plasticity, provided fitness effects are large (i.e. variation in individuals' plasticity is correlated with substantial variation in fitness). Larger fitness effects increase fitness variance (lowering geometric mean fitness), which can be reduced by producing mixtures of types – including, if $c>b$, differential plasticity. Meta-analyses of studies of wild animal populations show that frequencies of fitness effects are exponentially distributed, with smaller effects being much more common than larger ones (reviewed in Endler, 1986; Hoekstra *et al.*, 2001; Kingsolver *et al.*, 2001; for studies of humans, see Keller & Miller, 2006; Nettle & Pollet, 2008; Penke *et al.*, 2007; Stearns *et al.*, 2010). Moreover, recent analyses suggest that large fitness effects are even less common than earlier surveys indicated (Morrissey & Hadfield, 2012; Siepielski *et al.*, 2013). The plausibility of large fitness effects in the present case depends, among other things, on the extent to which 'plasticity levels' are correlated across different developmental domains. If an individual's plasticity level in one domain (e.g. metabolic adaptation) predicts her plasticity levels in other domains (e.g. reproductive development, stress responsivity), then the effects on overall fitness could be large. In contrast, if plasticity levels are narrowly trait-specific, their effects will be restricted to single traits as well, reducing their impact on overall fitness. The extent to which plasticity levels are correlated across domains is an open and interesting question, which has recently come into focus in studies of humans (Aron *et al.*, 2012; Belsky & Pluess, 2013; Del Giudice *et al.*, 2011) and other animals (Dingemanse, Kazem, Réale & Wright, 2010; Sih & Del Giudice, 2012; Stamps & Groothuis, 2010). Initial evidence suggests that plasticity levels are correlated across domains, but it would be premature to draw firm conclusions (Belsky & Pluess, 2013). Future research is needed to clarify this important issue.

Limitations and future directions

Models are by design simplified, idealized versions of reality, the goal of which is to capture some essential components of a process or system. Moreover, models may serve different kinds of purposes. One distinction is that between general and specific models (Parker & Maynard Smith, 1990): 'General models promote understanding of qualitative features. The parameters of such models may not be easy to measure. Specific models are based on a particular system and have parameters that can be measured so that predictions can be made' (Houston & McNamara, 2005, p. 934). We presented a general model whose goal is to: (a) explicate assumptions, (b) test their theoretical consequences and thus the logical cogency of hypotheses, and (c) understand interactions between variables that are difficult to intuit, if not impossible, without the help of formalizations (see Fawcett, Hamblin & Giraldeau, 2013; Frankenhuis, Panchanathan & Barrett, 2013). We have provided one way of capturing and analyzing the bet-hedging argument of differential plasticity (Belsky, 1997, 2005; Belsky & Pluess, 2009a, 2009b). However, our work is imperfect and incomplete, and we hope that future research will address these limitations.

First, our model does not address how mechanistic instantiation might constrain optimality (McNamara & Houston, 2009). We assume that parents can produce optimal proportions of offspring types, unhindered by genetic, developmental, physiological, or cognitive constraints. Such an assumption of unbounded optimality is sometimes called the 'phenotypic' or 'behavioral' gambit (Grafen, 1984; Fawcett *et al.*, 2013; Frankenhuis *et al.*, 2013). In real organisms, however, constraints abound. To give one example: we assume that offspring can produce exactly the same distributions of offspring types as their parents did ('like begets like'). This assumption is realistic in haploid asexual organisms, where selection happens among competing clones, but not in humans, who have two sets of chromosomes. Diploid genetic systems (a) constrain the offspring distributions that parents can produce (i.e. some types of offspring may not be producible), and (b) limit the extent to which parents can control the distribution of those offspring types that they can in fact produce (e.g. due to random shuffling of

alleles, recombination, and other processes); this is why heterozygotes, who are less susceptible to sickle-cell anemia than homozygotes, are not universal in populations exposed to malaria.

Given the randomness inherent to sexual reproduction, it seems implausible that in species producing small numbers of offspring, like humans, parents will diversify their offsprings' levels of plasticity using genetic means – for instance, by mating with multiple, genetically diverse partners (e.g. serial monogamy, promiscuity). Instead, it is more likely that parental bet-hedging, if it occurs in humans, is instantiated via epigenetic mechanisms, such as pre- or postnatal programming (Belsky, 2005; Belsky & Pluess, 2009a, 2011; Ellis *et al*., 2011; for evidence in non-human animals, see Crean & Marshall, 2009). In this scenario, natural selection would favor parents who transmit to their offspring, not so much different genetic variants, but rather variation in epigenetic settings that determines the extent to which experience shapes phenotypic development (i.e. epigenetic variation in sensitivity to developmental programming). An alternative possibility is that natural selection favors adaptive 'coin-flipping', in which parents produce offspring that stochastically vary in their levels of plasticity, irrespective of offspring experience (Bull, 1987; Cooper & Kaplan, 1982; Kaplan & Cooper, 1984; Salathé *et al*., 2009). Regardless, it would be valuable to formally examine how diploid genetics might influence scope for differential plasticity to evolve.

A second limitation is that our model assumes discrete, non-overlapping generations, which consist of a single selective life stage, as these assumptions are implicit in the way we computed geometric mean fitness: organisms are born and reproduce; mature individuals die; and the cycle repeats. Parents and offspring do not coexist. This assumption does not hold for humans. Therefore, future work should explore variants of our model that include overlapping generations. Classical biological models show that temporally fluctuating selection – in which the relative fitnesses of different phenotypes vary over time – is ineffective in maintaining genetic variation if generations are non-overlapping (e.g. Frank & Slatkin, 1990; but see Svardal, Rueffler & Hermisson, 2011). However, as Del Giudice (2012) notes, this result changes 'in species with (a) overlapping generations in which juveniles and adults coexist, and (b) multiple life stages, at least [one] of which is temporarily "shielded" from the [selective] effects of environmental change. When these conditions are met, temporally fluctuating selection becomes extremely effective in maintaining genetic variation, as multiple life stages store genetic variation and maintain it as the environment changes' (p. 55; for supporting references, see Del Giudice, 2012). Although maintenance of genetic variation in fluctuating environments does not normally result from bet-hedging (i.e. from strategies that sacrifice short-term mean fitness in order to reduce long-term fitness variance), in some cases it might (Svardal *et al*., 2011). It will thus be interesting to examine how including overlapping generations affects scope for differential plasticity to evolve.

A third limitation is that we assume discrete types of offspring (one plastic type, and two fixed types); our model does not consider continuous variation in the degree of plasticity. It is uncertain whether between-individual variation in plasticity would also evolve if individuals could develop such continuous levels. Conceivably, in that case, parents might produce all offspring with the same, intermediate level of plasticity. This is especially worth exploring because human G×E interaction research suggests that variation in plasticity may be better characterized in terms of a gradient than in typological terms (Belsky & Pluess, 2009a, 2009b; Belsky & Beaver, 2011).

Fourth, our model makes specific assumptions about rates of environmental change. It assumes that, first, the environmental state (e.g. safe or dangerous) is stable enough within generations for developmental programming to evolve, but not perfectly stable, resulting in maladaptive developmental programming in some individuals. Second, the environmental state is variable between generations to an extent that parents cannot predict the environment their offspring will experience any better than the long-term average probabilities of environmental states. In other words, our model assumes high environmental auto-correlation *within* generations, and no environmental auto-correlation *between* generations. An extension of the current model would be to consider between-generation auto-correlation in the environment. Recent evidence suggests that there have been periods in human history characterized by climatic fluctuations on the scale of decades to millennia, which is very rapid over evolutionary timescales but rather slow over one or even several individual lifetimes (Potts, 1998; Richerson, Boyd & Bettinger, 2001). This means that being born at a time of nutritional stress, or abundance, would have predicted – albeit imperfectly – a lifetime of such conditions for oneself and one's offspring. There might have been between-generation autocorrelation in dimensions of the social environment as well. For instance, within-society differences in social status (determining access to resources) may have been moderately stable across lifetimes in ancestral societies as they are in many extant societies (Borgerhoff Mulder, Bowles, Hertz, Bell, Beise *et al*., 2009) and in some non-human primates (Cheney, 1977). Formal models show

that moderate degrees of environmental stability across generations provide favorable conditions for the evolution of systems of epigenetic inheritance (Jablonka, Oborny, Molnár, Kisdi, Hofbauer *et al.*, 1995; Lachmann & Jablonka, 1996). With between-generation autocorrelation, uncertainty is reduced across generations. It is not clear what effect this type of uncertainty reduction will have on the scope for the evolution of bet-hedging.

Conclusions

Belsky's bet-hedging hypothesis (Belsky, 1997) is widely cited and is having interdisciplinary impact, inspiring research not only in observational and experimental studies in developmental psychology, but also in related fields, such as clinical science, pedagogy, and public policy (White, Li, Griskevicius, Neuberg & Kenrick, 2013). Despite its success, however, the bet-hedging hypothesis has never been formalized, even though related models have long existed in the biological sciences. Here we have provided such an analysis. Results support the argument's logical coherence, but only under restrictive conditions. We hope that future research will extend and modify our work, resulting in a family of models, with each examining the consequences of particular assumptions.

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Appendix

Bet-hedging model

Model set up

Parents can produce *plastic* offspring, who are adaptable, achieving a payoff of 1, irrespective of the environmental state. Parents can also produce *fixed* offspring (specialists), who are less adaptable, achieving a high payoff in a safe environment ($1+b$), and a low payoff in a dangerous environment ($1-c$).

Our goal is to compute the fraction of safe-specialists (x), danger-specialists (y), and plastic individuals (z) that natural selection favors parents to produce. Each of these fractions ranges between 0 and 1. We are particularly interested in the region where $0<x+y<1$, because there natural selection favors parental bet-hedging via the production of both fixed and plastic offspring. The optimal value of x and y will depend on features of the evolutionary environment; that is, on the probabilities of a safe (p) and dangerous environment ($1-p$), and the magnitude of fitness effects (b and c). These fitness effects specify how much better or worse specialists do compared with generalists if their phenotypes match or do not match the environmental state.

We assume that safe-specialists in a safe environment attain the same fitness as danger-specialists in a dangerous environment (namely, $1+b$). We also assume that safe-specialists in a dangerous environment attain the same fitness as danger-specialists in a safe environment (namely, $1-c$). This symmetry in payoffs justifies limiting our analyses to cases where $p>1-p$ (with $p<1-p$, our results would be a mirror image). Note that the higher p is, the more predictable the environment is across generations from the parents' viewpoint; if $p=1-p$, the environmental state is completely unpredictable from one generation to the next.

Parameters

$w\equiv$ parental fitness
$x\equiv$ fraction of safe-specialists (fixed type adapted to safe environment)
$y\equiv$ fraction of danger-specialists (fixed type adapted to dangerous environment)
$z\equiv 1-x-y\equiv$ fraction of plastic type
$p\equiv$ probability of a safe environment (range: 0–1)
$1-p\equiv$ probability of a dangerous environment
$b\equiv$ benefit to a fixed individual when fitting the local ecology (range: 0–1)
$c\equiv$ cost to a fixed individual when mismatched to local ecology (range: 0–1)

Intragenerational spatial variation

In spatially varying environments, the environment consists of different patches, each with a particular state (e.g. safe or dangerous). Within any generation, some offspring are born in one environmental state and other offspring in another. In such environments, parental fitness is given by the arithmetic mean across patches.

Fitness function

w = p(payoff in safe environment) + (1-p)(payoff in dangerous environment)

$$\begin{aligned} w = {} & p[(1-x-y)(1)+x(1+b)+y(1-c)] \\ & +(1-p)[(1-x-y)(1)+x(1-c)+y(1+b)] \end{aligned} \quad (2)$$

A basic and simple calculation leads to a reformulation:

$$w = 1 + x[pb - c(1-p)] + y[b(1-p) - pc] \quad (3)$$

This equation has a straightforward interpretation: In the second term, pb represents parents' expected benefit

of well-matched safe-specialists (i.e. the probability of a safe environment multiplied by the benefit that safe-specialists obtain in this environment), and $c(1-p)$ represents the expected cost of mismatched safe-specialists (i.e. the probability of a dangerous environment multiplied by the cost that safe-specialists incur in this environment). In the third term, $b(1-p)$ represents parents' expected benefit of well-matched danger-specialists (i.e. the probability of a dangerous environment multiplied by the benefit that danger-specialists obtain in this environment), and pc represents the expected cost of mismatched danger-specialists (i.e. the probability of a safe environment multiplied by the cost that danger-specialists incur in this environment).

If both of these expectations are negative, then parents maximize fitness (w) by producing $x+y=0$ (i.e. all plastic children).

If the former expectation is positive and latter negative, then parents maximize fitness by producing $x=1$ (i.e. all safe-specialists).

If the former expectation is negative and latter positive, then parents maximize fitness by producing $y=1$ (i.e. all danger-specialists).

If both expectations are positive, then parents maximize fitness by producing all offspring of the type that has the highest expectation (i.e. either $x=1$ or $y=1$).

Thus, if environmental variation is exclusively spatial, parents always maximize their fitness by producing either all safe- or danger-specialists, or all plastic individuals, and never a mixture (of both specialist types, or of one or both of the specialist types and the plastic type); thus, in this scenario, we would expect no variation in plasticity due to bet-hedging.

Intergenerational temporal variation

In environments that vary temporally between generations (but which remain stable within generations), the state of the environment changes simultaneously for all individuals across time. For instance, in one generation all individuals may develop in a safe patch, their offspring in a dangerous patch, and so forth. We assume that environmental states are independent and identically distributed (IID) across generations. In this case, if generations are discrete and non-overlapping, parental fitness is given by the average of sequential payoffs across generations. The average of n values multiplied is the nth root of their product, their geometric mean.

Fitness function

$$\begin{aligned} \mathrm{w} = &[(1-x-y)(1)+x(1+b)+y(1-c)]^{p} \\ &[(1-x-y)(1)+x(1-c)+y(1+b)]^{(1-p)} \end{aligned} \tag{4}$$

We ask what values of x and y maximize parents' fitness. Normally, we would find these values where the derivative of the fitness function w equals zero. However, the current case is exceptional, as shown below. Taking the natural log:

$$\begin{aligned} ln(w) = &\, p * ln[1-x-y+x(1+b)+y(1-c)]+ \\ &(1-p) * ln[1-x-y+x(1-c)+y(1+b)] \end{aligned} \tag{5}$$

Simplifying within brackets:

$$ln(w) = p * ln(1+xb-yc)+(1-p) * ln(1-xc+yb) \tag{6}$$

Taking the partial derivative with respect to x:

$$\frac{\partial ln(w)}{\partial x} = \frac{pb}{1+xb-yc} - \frac{c(1-p)}{1-xc+yb} \tag{7}$$

Taking the partial derivative with respect to y:

$$\frac{\partial ln(w)}{\partial y} = \frac{b(1-p)}{1-xc+yb} - \frac{pc}{1+xb-yc} \tag{8}$$

Both partial derivatives equal zero when:

$$\frac{pb}{1+xb-yc} - \frac{c(1-p)}{1-xc+yb} = 0 \tag{9}$$

$$\frac{b(1-p)}{1-xc+yb} - \frac{pc}{1+xb-yc} = 0 \tag{10}$$

Rewrite as: $u=1+xb-yc$ and: $v=1-xc+yb$:

$$\frac{pb}{u} - \frac{c(1-p)}{v} = 0 \tag{11}$$

$$\frac{b(1-p)}{v} - \frac{pc}{u} = 0 \tag{12}$$

Reorganizing:

$$pbv = (1-p)cu \tag{13}$$

$$pcv = (1-p)bu \tag{14}$$

Solve for v:

$$v = \left(\frac{1-p}{p}\right)\left(\frac{c}{b}\right)u \tag{15}$$

$$v = \left(\frac{1-p}{p}\right)\left(\frac{b}{c}\right)u \tag{16}$$

These conditions simultaneously hold when:

$$\left(\frac{1-p}{p}\right)\left(\frac{c}{b}\right)u = \left(\frac{1-p}{p}\right)\left(\frac{b}{c}\right)u \tag{17}$$

Dividing out $(\frac{1-p}{p})$, multiplying both sides by bc, and reorganizing:

$$c^2u = b^2u \tag{18}$$

$$u(b^2 - c^2) = 0 \tag{19}$$

$$u(b+c)(b-c) = 0 \tag{20}$$

Formally, this equation has four solutions: u=0, b=c, b=$-c$, and c=$-b$. However, the last two of these are not biologically meaningful, so we ignore them. The second solution is an implausible knife edge (b=c), and we will show later that differential plasticity is never uniquely favored in this condition. Thus, we will focus on the first solution:

$$u = 1 + xb - yc = 0 \tag{21}$$

$$v = 1 - xc + yb = 0 \tag{22}$$

Solving each for x:

$$xb = yc - 1 \Longrightarrow x = \frac{yc-1}{b} \tag{23}$$

$$xc = yb + 1 \Longrightarrow x = \frac{yb+1}{c} \tag{24}$$

These conditions simultaneously hold when:

$$\frac{yc-1}{b} = \frac{yb+1}{c} \tag{25}$$

Multiplying both sides by bc, and reorganizing:

$$y = \frac{-1}{b-c} \tag{26}$$

We obtain the corresponding x:

$$xb = \frac{-c}{b-c} - 1 \Longrightarrow x = \frac{-1}{b-c} \tag{27}$$

This equilibrium $x = y = \frac{-1}{b-c}$ cannot be attained. If b>c, then x<0 and y<0; if c>b, then x>1 and y>1. These results show that selection never simultaneously favors the optimal x and y (unless, perhaps, when b=c, discussed later). If $b \neq c$, selection will take x and y to the edges of a triangle, bounded by points: (x=0, y=0), (x=1, y=0), and (x=0, y=1). Within this triangle, the fitness function is monotonically increasing or decreasing, depending on the values of p, b and c.

Exploring the edges

For each combination of parameter values (i.e. p, b, and c), we want to know the associated fitness of the edge solutions. The edge solution that yields the highest fitness for a given set will be favored by natural selection. We find the optimal edge solution for x between points (x=0, y=0) and (x=1, y=0) by entering y=0 into our fitness equation $ln(w)$.

$$ln(w_{y=0}) = p * ln(1 + xb - 0c) + (1-p) * ln(1 - xc + 0b) \tag{28}$$

Taking the partial derivative with respect to x:

$$\frac{\partial ln(w_{y=0})}{\partial x} = \frac{pb}{1+xb} - \frac{(1-p)c}{1-xc} \tag{29}$$

Setting this partial derivative to zero and solving for x:

$$\hat{x}_{y=0} = \frac{pb - c(1-p)}{bc} \tag{30}$$

When $0 < \hat{x}_{y=0} < 1$, differential plasticity is favored over only plastic individuals, and over only one type of specialist. This is when:

$$\frac{c}{b+c} < p < \frac{c}{b+c} + \frac{bc}{b+c} \tag{31}$$

Next,we find the optimal edge solution for y between points (x=0, y=0) and (x=0, y=1) by entering x=0 into $ln(w)$, and taking the partial derivative with respect to y:

$$\frac{\partial ln(w_{x=0})}{\partial y} = \frac{-cp}{1-yc} + \frac{(1-p)b}{1+yb} \tag{32}$$

Setting this derivative to zero, and solving for y:

$$\hat{y}_{x=0} = \frac{b(1-p) - pc}{bc} \tag{33}$$

When $0 < \hat{y}_{x=0} < 1$, a mix of y and z is favored over only plastic individuals, and over only one type of specialist:

$$\frac{b}{b+c} - \frac{bc}{b+c} < p < \frac{b}{b+c} \tag{34}$$

Finally, we find the optimal edge solution for x between points (x=1, y=0) and (x=0, y=1), by entering z=1$-x-y$=0 (i.e. y=1$-x$) into $ln(w)$, and taking the partial derivative with respect to x:

$$\frac{\partial ln(w_{z=0})}{\partial x} = \frac{p(b+c)}{1+xb-c(1-x)} - \frac{(1-p)(b+c)}{1-xc+b(1-x)} \tag{35}$$

Setting this derivative to zero, and solving for x:

$$\hat{x}_{z=0} = \frac{p(1+b)-(1-p)(1-c)}{b+c} \tag{36}$$

When $0<\hat{x}_{z=0}<1$, a mix of specialists is favored over either pure specialist:

$$\frac{1-c}{2+b-c} < p < \frac{1+b}{2+b-c} \tag{37}$$

Differential plasticity can be favored if c>b

We know that natural selection will favor edge solutions; hence, we will consider differential plasticity (DP) as producing safe-specialists and plastic individuals only (not danger-specialists). DP beats Mixtures of Specialists (MS) if:

$$(1+xb)^p(1-xc)^{1-p} > (1+xb-yc)^p(1-xc+yb)^{1-p} \tag{38}$$

Dividing by DP:

$$1 > \left(\frac{1+xb-yc}{1+xb}\right)^p \left(\frac{1-xc+yb}{1-xc}\right)^{1-p} \tag{39}$$

Taking the natural log:

$$0 > p * ln\left(\frac{1+xb-yc}{1+xb}\right) + (1-p) * ln\left(\frac{1-xc+yb}{1-xc}\right) \tag{40}$$

Assume DP produces $x_{dp} = \hat{x}_{ms} - \hat{y}_{ms}$ (it can, because $\hat{x}_{ms} > \hat{y}_{ms}$):

$$0 > p * ln\left(\frac{1+xb-yc}{1+(x-y)b}\right) + (1-p) * ln\left(\frac{1-xc+yb}{1-(x-y)c}\right) \tag{41}$$

Distributing terms in the denominators:

$$0 > p * ln\left(\frac{1+xb-yc}{1+xb-yb}\right) + (1-p) * ln\left(\frac{1-xc+yb}{1-xc+yc}\right) \tag{42}$$

The left log term is negative because $-yc<-yb$. The right one because $yb<yc$. This proof ignores (1) that the slope of log terms <1 are steeper than that of log terms >1, and (2) the left log term is weighted by p, which $>1-p$. Thus, the proof holds for all values of p. If taken into account, (1) and (2) strengthen DP's superiority over MS. Barring the strategies converging on production of only Specialists, DP beats MS.

Differential plasticity is never favored if b>c

We can show that when $b>c$, DP never beats MS:

$$(1+xb)^p(1-xc)^{1-p} \le (1+xb-yc)^p(1-xc+yb)^{1-p} \tag{43}$$

If MS attains equal or higher fitness than DP in either environment, its geometric mean fitness across environments will also equal or exceed that of DP (in which case DP is never uniquely favored if $b>c$):

$$1+(\hat{x}_{dp})b \le 1+(x_{ms})b-(y_{ms})c \tag{44}$$

$$1-(\hat{x}_{dp})c \le 1-(x_{ms})c+(y_{ms})b \tag{45}$$

Deducting 1 from all sides:

$$(\hat{x}_{dp})b \le (x_{ms})b-(y_{ms})c \tag{46}$$

$$-(\hat{x}_{dp})c \le -(x_{ms})c+(y_{ms})b \tag{47}$$

Solving for x_{ms} (note: the sign flips in the bottom equation):

$$\frac{(\hat{x}_{dp})b+(y_{ms})c}{b} \le (x_{ms}) \tag{48}$$

$$\frac{(\hat{x}_{dp})c+(y_{ms})b}{c} \ge (x_{ms}) \tag{49}$$

Combined in one line:

$$\frac{(\hat{x}_{dp})b+(y_{ms})c}{b} \le (x_{ms}) \le \frac{(\hat{x}_{dp})c+(y_{ms})b}{c} \tag{50}$$

$$(\hat{x}_{dp})+(y_{ms})\left(\frac{c}{b}\right) \le (x_{ms}) \le (\hat{x}_{dp})+(y_{ms})\left(\frac{b}{c}\right) \tag{51}$$

$$\frac{c}{b} \le \frac{(x_{ms})-(\hat{x}_{dp})}{(y_{ms})} \le \frac{b}{c} \tag{52}$$

Where $x_{ms}>y_{ms}$. Consider $\hat{x}_{dp} = 0+e$, where e is tiny:

$$\frac{c}{b} \le \frac{x_{ms}-(0+e)}{y_{ms}} \le \frac{b}{c} \tag{53}$$

$$\frac{c}{b} \le \frac{x_{ms}-e}{y_{ms}} \le \frac{b}{c} \tag{54}$$

If $b>c$, MS can always fulfill this condition (note: since $x_{ms}>y_{ms}$, the left side of the equation always holds). Now consider $\hat{x}_{dp} = 1-e$:

$$(1-e)+(y_{ms})\left(\frac{c}{b}\right) \leq (x_{ms}) \leq (1-e)+(y_{ms})\left(\frac{b}{c}\right) \quad (55)$$

Now y_{ms} can be taken down to a very small number such that x_{ms} will be between the two ends. Note that y_{ms} must be smaller than $0+e$. To see this, consider $y_{ms}=0+e$ (meaning that $x_{ms}=1-e$):

$$(1-e)+(0+e)\left(\frac{c}{b}\right) \leq (1-e) \leq (1-e)+(0+e)\left(\frac{b}{c}\right) \quad (56)$$

$$(e)\left(\frac{c}{b}\right) \leq 0 \leq (e)\left(\frac{b}{c}\right) \quad (57)$$

If, however, $y_{ms}=0+d$, where $d<e$, then:

$$(1-e)+(0+d)\left(\frac{c}{b}\right) \leq (1-d) \leq (1-e)+(0+d)\left(\frac{b}{c}\right) \quad (58)$$

Subtracting 1, multiplying by -1, and simplifying (note: the signs flip):

$$e-d\left(\frac{c}{b}\right) \geq d \geq e-d\left(\frac{b}{c}\right) \quad (59)$$

Dividing by d:

$$\left(\frac{e}{d}\right)-\left(\frac{c}{b}\right) \geq 1 \geq \left(\frac{e}{d}\right)-\left(\frac{b}{c}\right) \quad (60)$$

Because $\frac{b}{c} > \frac{c}{b}$, the right term will always be smaller than the left term. MS can always produce a value of d that ensures the right term is smaller than one, and the left term larger than one. Thus, if $b>c$, MS can always match or beat DP.

Arithmetic mean fitness, variance in fitness, and geometric mean fitness

In the article, we present the regions of parameter space in which differential plasticity is uniquely favored in a temporally fluctuating environment (Figure 1). Here, we present the arithmetic mean fitness, variance in fitness, and geometric mean fitness, for three strategies (in order): differential plasticity, mixtures of specialists, and only safe-specialists. We do not present a separate figure for pure generalists: its arithmetic and geometric mean fitnesses are 1, and its fitness variance is 0, in the entire parameter space.

In the following three figures, the x-axis represents the additional benefit (b) fixed specialists obtain, compared with plastic generalists, if their phenotype matches the environmental state. The y-axis represents the additional cost (c) fixed specialists incur, compared with plastic generalists, if their phenotype does not match the environmental state. The top row depicts a strategy's arithmetic mean fitness when the probability of a safe environment equals 0.6, 0.75, and 0.9 (from left to right); here, contour lines indicate the value of arithmetic mean fitness. The middle row depicts a strategy's variance in fitness when the probability of a safe environment equals 0.6, 0.75 and 0.9 (from left to right); here, contour lines indicate the value of variance in fitness. The bottom row depicts a strategy's geometric mean fitness when the probability of a safe environment equals 0.6, 0.75, and 0.9 (from left to right); here, contour lines indicate the value of geometric mean fitness. In all of the figures, in regions denoted 'S' (shaded dark gray), only safe-specialists are favored; if 'G' (shaded medium gray), pure generalists; if 'M' (shaded light grey), mixtures of specialists; if 'D' (not shaded), differential plasticity. If $b>c$, differential plasticity is never favored. If $c>b$, differential plasticity can be favored, especially if c is large.