Genetic evolution, plasticity and bet-hedging as adaptive responses to temporally autocorrelated fluctuating selection: A quantitative genetic model

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Abstract

Adaptive responses to autocorrelated environmental fluctuations through evolution in mean reaction norm elevation and slope and an independent component of the phenotypic variance is analysed using a quantitative genetic model. Analytic approximations expressing the mutual dependencies between all three response modes are derived and solved for the joint evolutionary outcome. Both genetic evolution in reaction norm elevation and plasticity are favored by slow temporal fluctuations, with plasticity, in the absence of microenvironmental variability, being the dominant evolutionary outcome for reasonable parameter values. For fast fluctuations, tracking of the optimal phenotype through genetic evolution and plasticity is limited. If residual fluctuations in the optimal phenotype are large and stabilizing selection is strong, selection then acts to increase the phenotypic variance (bet-hedging adaptive). Otherwise, canalizing selection occurs. If the phenotypic variance increases with plasticity through the effect of microenvironmental variability, this shifts the joint evolutionary balance away from plasticity in favor of genetic evolution. If microenvironmental deviations experienced by each individual at the time of development and selection are correlated, however, more plasticity evolves. The adaptive significance of evolutionary fluctuations in plasticity and the phenotypic variance, transient evolution, and the validity of the analytic approximations are investigated using simulations.

Introduction

Several primary mechanisms of phenotypic adaptation to fluctuating environments are widely recognized. The main mechanism envisioned by Darwin (1859) was adaptation through the continual effects of natural selection on heritable variation among individuals. Without knowledge about the mechanisms underlying heredity, Darwin thought that this would be a slow process, dependent on the continual input of new variability through mutation. For polygenic traits we now know that considerable amounts of standing genetic variation can be maintained by a balance between mutation and selection (Barton and Keightley, 2002) and that this variation is sufficient to explain observed and often high rates of contemporary evolution (Hendry and Kinnison, 1999). If the rate of change in the environment is high, however, adaptive tracking of the environment through genetic evolution is limited by the amount of standing genetic variance in the trait under selection, such that the mean phenotype may lag behind the phenotypic optimum (Lande and Shannon, 1996).

All phenotypic traits have both genetic and environmental influences. The way in which an organism responds to environmental conditions may itself have a genetic basis, however, and may thus evolve if such genotype by environment interaction is present. The resulting evolutionary response, adaptive phenotypic plasticity, represents an alternative, competing mechanism of phenotypic adaptation to changing environmental conditions. Plasticity is limited by the correlation between the environment at the time of development and at the time of selection which is necessarily less than one in continuously changing environments since selection always occurs some time after development. The model of Gavrilets and Scheiner (1993) predicts a reaction norm equal to the regression of the optimal phenotype at time of selection on the environment at the time of development. Other limitations may include possible costs of plasticity associated with the sensory, physiological and developmental machinery needed to develop a plastic phenotype (Moran, 1992; DeWitt et al., 1998). Thus, a perfect match between the optimal and mean phenotype is not expected from theoretical consideration nor seen empirically (e.g.

Charmantier et al., 2008).

A third distinct mechanism of phenotypic adaptation is diversifying bet-hedging, hereafter referred to as only bet-hedging. A well known example is the random time of germination of the seeds of many desert plant preventing the extinction of genetic lineages by increasing the likelihood that at least some individuals belonging to a particular genetic lineage germinate in good years (Cohen, 1966). Theoretical models based on the geometric mean fitness principle have shown that selection fluctuating across generations can create selection for increased values of the environmental component of the phenotypic variance provided that the fluctuations in the fitness of different phenotypes are sufficiently large. In Bull (1987) and later models (e.g. Svardal et al., 2011) based on Gaussian stabilizing selection, bet-hedging is only adaptive if the variance of the fluctuations in the optimum is larger than the squared width of the Gaussian fitness function. If this holds, the phenotypic variance evolves to the difference between these two quantities. Other models, involving asymmetric fitness functions (Einum and Fleming, 2004; Olofsson et al., 2009), reach similar conclusions.

The above three forms of adaptations are not necessarily mutually exclusive but may possibly occur simultaneously. Plasticity is known to reduce genetic evolution the reaction norm elevation, both in the case of autocorrelated temporal (Tufto, 2000b; Michel et al., 2014) and spatial (de Jong, 1999; Scheiner, 1998) fluctuation in the phenotypic optimum. With constant plasticity, this occurs as a result of the reduced mismatch between the mean and optimal phenotype, reducing selection on mean reaction norm elevation (Tufto, 2000b; Michel et al., 2014). An association between the phenotypic variance and plasticity, through developmental instability as hypothesized by DeWitt et al. (1998) or through microenvironmental variability, might additionally further reduce adaptive tracking with increasing levels of plasticity through reduced heritability. The reverse effect of genetic evolution in mean elevation on the evolution of plasticity is less well understood. Michel et al. (2014) assume that the reaction norm slope remains unaffected by adaptive tracking, contrary to previous results for deterministic cyclic fluctuations (Tufto, 2000b, eq. 23). Lande (2009) also relies on this simplification, which implicitly assumes that

environmental fluctuations are too fast for any significant amount of adaptive tracking in mean elevation to occur. The high rates of contemporary evolution observed in many traits (Hendry and Kinnison, 1999; Kinnison and Hendry, 2001; Bell, 2010), however, suggests that there is a need for theoretical models that considers the mutual evolutionary dependency between these two processes in more detail.

Similar mutual feedbacks must also exist between the evolution of bet-hedging and mechanisms contributing to adaptive tracking of the mean phenotype in response to environmental change. Bet-hedging, on the one hand, through the increase in the environmental component of the phenotypic variance will reduce heritability which in turn should reduce adaptive tracking through genetic evolution (Scheiner, 2014a). A small mismatch arising as a result of either adaptive tracking in mean elevation or a high level of plasticity, on the other hand, might have an effect similar to a reduction in the variance of fluctuations in the environment optimum in Bull's (1987) model, reducing the parts of the parameter space where bet-hedging is adaptive.

Here I model the joint evolution of the above three forms of response to fluctuating selection. Most traits important in phenotypic evolution are believed to be polygenic (Lande, 1982) or at least oligogenic (Bell, 2010). Some empirical evidence suggest genetic architectures in which large parts of the genome involved in phenotypic plasticity involve developmental switches and that these may be single genes (as assumed by Botero et al., 2014). Although such single genes, e.g. hox genes, may be involved, most authors argue that developmental switches are under polygenic control (e.g. West-Eberhard, 2005). This suggest that a quantitative genetic approach based on approximately constant genetic variances and covariances maintained by a balance between mutation, drift and selection should provide a reasonable starting point for a generic model. By assuming small genetic variances in reaction norm slope and bet-hedging such that evolutionary fluctuations in these quantities are small, several analytic approximations expressing the mutual dependency between the three different processes at the joint evolutionary equilibrium are derived, putting the various feedbacks between the processes discussed above in quantitative terms. These equations are then solved numerically or analytically to

yield values of these quantities in terms of basic model parameters.

The model also elucidates more subtle links between plasticity and bet-hedging. Most previous models assume that the environmental cue on which development depends is shared between all individuals in the population (Michel et al., 2014; Botero et al., 2014; Ezard et al., 2014). However, as shown by Donaldson-Matasci et al. (2013), using a model with discrete environmental states, if cues vary between individuals as a result of microenvironmental variability, the between-individual phenotypic variance will increase with increasing plasticity, in effect leading to diversifying bet-hedging but through another mechanism. More generally, we shall see that such microenvironmental variability also has implications for the level of plasticity that evolves and how this depends on correlations between microenvironments experienced individually at time of development and selection. Implications of the model for the overall relative magnitude of the different evolutionary responses are also discussed.

Model

RATIONALE AND ASSUMPTIONS

Previous theory has shown that the evolution of plasticity must be strongly dependent on the the correlation between the environment at time of development and at the time of selection. If the environment fluctuates according to a continuous-time stochastic process and selection occurs some fraction of the generation time after development, this correlation is necessarily less than one. Here, autocorrelated fluctuations in the macroenvironment ε_t affecting all individuals in the population is modeled by assuming that ε_t is an Ornstein-Uhlenbeck process (Karlin and Taylor, 1981), which is a continuous-time analogue of a first order autoregressive process, or, roughly speaking, a random walk with a mean-reverting tendency. Parameterizing this process in terms of the stationary variance $\mathrm{Var}(\varepsilon_t) = \sigma_\varepsilon^2$ and the autocorrelation α across one generation, the autocovariance

function can be written as

$$\mathrm{Cov}(\varepsilon_t, \varepsilon_{t+h}) = \sigma_\varepsilon^2 \alpha^h = \sigma_\varepsilon^2 e^{-h/T}. \tag{1a}$$

The alternative parameterization in terms of the temporal scale $T = -1/\ln\alpha$ of exponential decay in autocorrelation with increasing distance in time h, referred to as the autocorrelation time in Lande and Shannon (1996), will also be useful. For example, for autocorrelation $\alpha = 0.99$, $T \approx 99$ generations. The macroenvironment ε_t is assumed to have a stationary mean of zero. A useful recursion equation for the change in the state of the macroenvironment over a possibly non-integer time step of length h is

$$\varepsilon_{t+h} = \alpha^h \varepsilon_t + \delta_{t,h}, \tag{1b}$$

where $\delta_{t,h}$ is defined as a normally distributed increment independent of the state ε_t of the process at time t, with zero mean and variance $(1 - \alpha^{2h})\sigma_\varepsilon^2$.

To model the effects of microenvironmental variability I assume that the environments experienced by a particular individual at the time of development and selection are given by $\varepsilon_{t-\tau} + u$ and $\varepsilon_t + v$, respectively, where the microenvironmental deviations u and v are assumed to have a bivariate normal distribution with zero means, a common variance σ_u^2 and correlation ρ_{uv}. For organisms that are mostly sessile, such as many species of plants, development and selection may occur at the same spatial location. In this case ρ_{uv} may be close to one if variation in u and v reflect permanent spatial differences, say differences in topography and nutrient availability. Other organisms such as insects with continuous movement may be subject to selection at a spatial location different from that of development such that u and v are only weakly correlated. I assume that dispersal occurring after selection, however, is sufficiently high to make the population panmictic.

The optimal phenotype of a given individual is then assumed to be determined by its surrounding microenvironment at time of selection by the linear relationship

$$\theta = A + B(\varepsilon_t + v), \tag{2a}$$

where the parameters A and B are the optimal reaction norm elevation and slope.

The expressed phenotype z of the same particular individual is similarly determined by its surrounding microenvironment at the time of development, its genetic reaction norm elevation and slope a and b as well as a third genotypic value c, also subject to selection, according to the relationship

$$z = a + b(\varepsilon_{t-\tau} + u) + cy + e. \tag{2b}$$

The two last terms are explained in the next two paragraphs. Note how the inclusion of the microenvironmental deviation u in second term on the right hand side of (2b) gives rise to an environmental component of the phenotypic variance equal to $b^2\sigma_u^2$. Although having some of the same effects, this does not represent a relationship between developmental instability and plasticity as hypothesized be DeWitt et al. (1998) and supported empirically by some studies (Tonsor et al., 2013), but only reflects the fact that the environmental states experienced by different individuals deviates from the population mean.

Following Gavrilets and Hastings (1994), evolution of the environmental component of the phenotypic variance in the model, independent from changes through the reaction norm slope b, is accomodated by the third term on the right hand side of (2b) involving a white-noise variable y and a second reaction norm slope c representing the dependency of the phenotype z on y. The variable y, distinct and independent of u and v, may represent some random or pseudorandom microenvironmental variable in the surroundings of each individual suitable for generating a random phenotype or some random or pseudorandom factor of purely internal origin representing developmental instability. Under the latter interpretation, the model is in effect almost (see Discussion) equivalent to the non-pleiotropic models in (Scheiner, 2014b,a) although a variable y is not introduced explicitly in his model formulation. Without loss of generality, y is assumed to have a mean of zero and unit variance. This term thus gives rise to an environmental component of the phenotypic variance equal to c^2. A link between developmental instability and the reaction norm slope b as hypothesized by DeWitt et al. (1998) can be accomodated by a genetic correlation between b and c.

Environmental canalization or homogeneity are thought to induce costs associated

with regulatory feedback mechanisms necessary to make the phenotype less sensitive to microenvironmental external or internal conditions. Rather than modeling this cost explicitly by making individual fitness directly dependent on c as in Zhang and Hill (2005), a lower limit on the environmental component of the phenotypic variance reflecting such a cost only implicitly is introduced by including a fourth normally distributed zero mean individually varying term e with non-evolving variance σ_e^2. This term too may represent both microenvironmental variation independent from u, v and y but also internal developmental stochasticity. It follows that the total phenotypic variance of a particular genotype generated by microenvironmental variation, commonly referred to as V_E (Bulmer, 1980; Falconer and Mackay, 1996), is $b^2\sigma_u^2 + c^2 + \sigma_e^2$.

Following Bull (1987), Gavrilets and Scheiner (1993) and Lande (2009), stabilizing selection acts through the phenotype z towards the individually varying phenotypic optimum θ according to a Gaussian individual fitness function with width ω such that

$$w(z-\theta) = \exp\left\{-\frac{(z-\theta)^2}{2\omega^2}\right\}. \tag{2c}$$

I assume hard selection as defined by Christiansen (1975), that is, density regulation occurs globally after selection such that the contribution from each microenvironment is proportional to mean fitness within each microenvironment.

In any particular generation t it is assumed that the genotypic values (a, b, c) are jointly multivariate normal with mean vector $(\bar{a}_t, \bar{b}_t, \bar{c}_t)$ and genetic variance-covariance matrix

$$\mathbf{G} = \begin{bmatrix} G_{aa} & 0 & 0 \\ 0 & G_{bb} & 0 \\ 0 & 0 & G_{cc} \end{bmatrix}. \tag{2d}$$

No genetic correlation between a and b is expected if the character has been through a long period of genetic canalization (Lande, 2009). A similar argument can be made for the genetic correlation between a and c. A genetic correlation between plasticity and developmental instability resulting from pleiotropic genetic effects as considered in Scheiner (2014a) can be accomodated by a nonzero G_{bc}-element but this is deferred to the discussion.

MEAN FITNESS AND SELECTION GRADIENT

Before proceeding, note how the selection gradient becomes a stochastic variable through its dependency on the macroenvironment at development $\varepsilon_{t-\tau}$ and selection ε_t affecting all individuals in the population. Mean fitness and the selection gradient is therefore derived conditionally on these variables whereas all other individually varying quantities in (2a) and (2b) (a, b, c, u, v, y and e) are regarded as random. Under hard selection, the response to selection will be given by $\mathbf{G}\nabla \ln \bar{w}$ as in Lande and Arnold (1983) but with $\bar{w}$ defined as mean fitness across the distribution of microenvironments u and v (see Appendix S3).

Although the deviation of each individual's phenotype from its optimum experienced at the time of selection $z-\theta$ (the differences between the sums appearing in (2a) and (2b)) involves some non-normal terms (products between independent normally distributed variables), the deviation is a sum of four or five independent random terms and should thus be well approximated by a normal distribution. From the assumption of independence and zero expectations of the microenvironmental variables and formulas for the variance and covariance of products of random variables (Goodman, 1960; Bohrnstedt and Goldberger, 1969), the mean and variance of $z-\theta$ is

$$E(z-\theta) = \bar{a}_t + \bar{b}_t\varepsilon_{t-\tau} - A - B\varepsilon_t, \tag{3a}$$

$$\begin{aligned}\mathrm{Var}(z-\theta) = {} & G_{aa} + G_{bb}\varepsilon_{t-\tau}^2 + (G_{bb} + \bar{b}_t^2)\sigma_u^2 + G_{cc} \\ & + \bar{c}^2 + \sigma_e^2 + B^2\sigma_u^2 - 2\bar{b}_t B\rho_{uv}\sigma_u^2.\end{aligned} \tag{3b}$$

Note how the phenotypic variance, or more precisely, the population phenotype-optimum variance, in the following denoted $\sigma_z^2(\bar{b}_t, \bar{c}_t, \varepsilon_{t-\tau})$, is a function of both $\bar{b}_t$ and $\bar{c}_t$ and also the environment at the time of development $\varepsilon_{t-\tau}$.

Relying on the normal approximation for $z-\theta$, mean fitness is a convolution of two Gaussian functions that simplifies to

$$\begin{aligned}\bar{w}(\bar{a}_t, \bar{b}_t, \bar{c}_t) = {} & \sqrt{\frac{\omega^2}{\omega^2 + \sigma_z^2(\bar{b}_t, \bar{c}_t, \varepsilon_{t-\tau})}} \\ & \times \exp\left\{-\frac{(\bar{a}_t + \bar{b}_t\varepsilon_{t-\tau} - A - B\varepsilon_t)^2}{2(\omega^2 + \sigma_z^2(\bar{b}_t, \bar{c}_t, \varepsilon_{t-\tau}))}\right\}.\end{aligned} \tag{3c}$$

From this the selection gradient becomes

$$\begin{aligned}\boldsymbol{\beta} &= \left(\frac{\partial}{\partial \bar{a}_t}, \frac{\partial}{\partial \bar{b}_t}, \frac{\partial}{\partial \bar{c}_t}\right)^T \ln \bar{w} \\ &= -\frac{1}{\omega^2 + \sigma_z^2(\bar{b}_t, \bar{c}_t, \varepsilon_{t-\tau})} \\ &\times \begin{bmatrix} \bar{a}_t + \bar{b}_t\varepsilon_{t-\tau} - A - B\varepsilon_t \\ \\ (\bar{a}_t + \bar{b}_t\varepsilon_{t-\tau} - A - B\varepsilon_t)\varepsilon_{t-\tau} \\ \qquad +\sigma_u^2(\bar{b}_t - \rho_{uv}B)\left(1 - \frac{(\bar{a}_t+\bar{b}_t\varepsilon_{t-\tau}-A-B\varepsilon_t)^2}{\omega^2+\sigma_z^2(\bar{b}_t,\bar{c}_t,\varepsilon_{t-\tau})}\right) \\ \\ \bar{c}\left(1 - \frac{(\bar{a}_t+\bar{b}_t\varepsilon_{t-\tau}-A-B\varepsilon_t)^2}{\omega^2+\sigma_z^2(\bar{b}_t,\bar{c}_t,\varepsilon_{t-\tau})}\right) \end{bmatrix}. \end{aligned} \tag{3d}$$

The first component of the selection gradient shows that mean reaction norm elevation $\bar{a}_t$ increases whenever the mismatch between the mean and optimal phenotype ($\bar{a}_t + \bar{b}_t\varepsilon_{t-\tau} - A - B\varepsilon_t$) is negative. The third component representing selection on $\bar{c}_t$ through its effect on the phenotypic variance shows that $\bar{c}_t$ increases whenever the mismatch, in absolute value, is sufficiently large to make the Gaussian mean population fitness function convex rather than concave (mismatch larger than $\sqrt{\omega^2 + \sigma_z^2(\bar{b}_t, \bar{c}_t, \varepsilon_{t-\tau})}$). The second component includes two terms; the first representing selection on $\bar{b}_t$ through its effects on the phenotypic mean as in Lande (2009), and the second term representing selection through the effects of $\bar{b}_t$ on the phenotypic variance.

Multiplying the **G**-matrix with the above selection gradient (Lande and Arnold, 1983) and relying on the results in Appendix S3 leads to three stochastic non-linear recursions equations for $\bar{a}_t$, $\bar{b}_t$ and $\bar{c}_t$ coupled to each other and driven by the exogenous environmental process ε_t. These recursion equations can be used in stochastic simulations of the process. One realization is shown in Fig. 1.

Analytic Approximations

To gain some insights into the behaviour of this process beyond what can be learned from stochastic simulations, I derive several analytic approximations for the limiting case that the genetic variances G_{bb} and G_{cc} in the reaction norm slopes b and c are small. This has

three implications. First, a small genetic variance in the reaction norm slope b makes the dependency of the phenotype-optimum variance (3b) on the environment at the time of selection $\varepsilon_{t-\tau}$ through the term $G_{bb}\varepsilon_{t-\tau}^2$ small. We therefore approximate this term by its long term average value $G_{bb}\sigma_\varepsilon^2$ and let $\sigma_z^2(\bar{b}_t, \bar{c}_t)$ denote the resulting phenotype-optimum variance.

Secondly, in the limit of small genetic variances in b and c, the products bu and cy appearing as terms in the expression for the deviation $z-\theta$ tend to normal distributions. This in turn implies that $z-\theta$ is also normally distributed, and so (3c) and (3d) are exact in this limiting case.

Thirdly, and most importantly, although small G_{bb} and G_{cc} will slow down the transient phase of the evolution of $\bar{b}_t$ and $\bar{c}_t$, fluctuations in $\bar{b}_t$ and $\bar{c}_t$, once stochastic equilibrium has been reached, will be small such that these quantities can be replaced by their eventual long term equilibrium values $\bar{\bar{b}}$ and $\bar{\bar{c}}$. As we shall see, this greatly simplifies the analysis and leads to several useful approximations expressing the mutual evolutionary dependencies between the three forms of adaptations.

It is emphasized that the magnitude of G_{bb} and G_{cc} is an empirical question and that the following analytic approximations ultimately must to be validated against stochastic simulations of the process in cases also including larger genetic variances in b and c, see Appendix S4.

GENETIC EVOLUTION IN MEAN REACTION NORM ELEVATION

From (3d), for given values of $\bar{\bar{b}}$ and $\bar{\bar{c}}$, the recursion equation for fluctuations in mean elevation $\bar{a}_t$ becomes

$$\bar{a}_{t+1} = (1-s)\bar{a}_t - s\bar{\bar{b}}\varepsilon_{t-\tau} + sA + sB\varepsilon_t, \tag{4a}$$

where

$$s = \frac{G_{aa}}{\omega^2 + \sigma_z^2(\bar{\bar{b}}, \bar{\bar{c}})}. \tag{4b}$$

Note that the phenotypic variance (3b) in the denominator increases with both $\bar{\bar{b}}$ and $\bar{\bar{c}}$ and how this reduces s through a reduction in heritability.

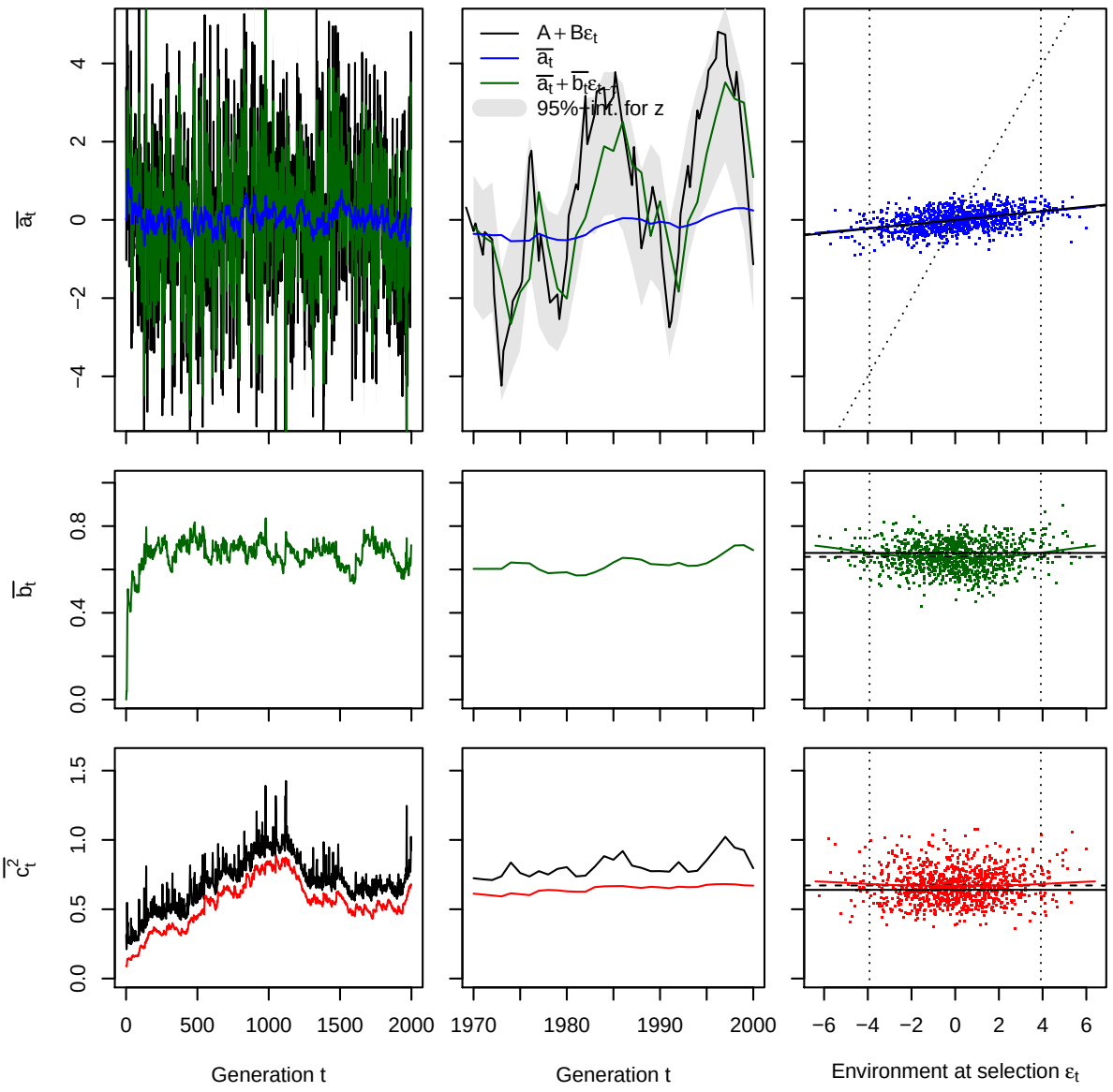

Figure 1: The evolution of $\bar{a}_t$, $\bar{b}_t$ and $\bar{c}_t^2$ (blue, green, and red curve in upper, middle and lower row, respectively) simulated over 2000 generations (left column) with a closeup of the last 30 generations shown in middle column. Also shown (left and middle column) is the mean optimal phenotype $A + B\varepsilon_t$ (black curve in upper plots), the mean phenotype $\bar{a}_t + \bar{b}_t\varepsilon_{t-\tau}$ (green curve in upper plots), a 95% phenotype interval (grey shaded area in upper plots), and the evolution of the total phenotypic variance (black curve in lower plots). Parameter values used are $\omega^2 = 1$, $\sigma_\varepsilon^2 = 4$, $\alpha = 0.7$, $\tau = 0.8$, $\alpha^\tau = 0.752$, $\sigma_u^2 = 0$, $\rho_{uv} = 0$, $G_{aa} = 0.1$, $G_{bb} = 0.01$, $G_{cc} = 0.01$, $A = 0$ and $B = 1$ and initial values $\bar{a}_1 = 0$, $\bar{b}_1 = 0$, $\bar{c}_1 = 0.3$. The right column shows a longer simulation extended to 10^5 generations excluding the first 10^4 generations with $\bar{a}_t$, $\bar{b}_t$ and $\bar{c}_t^2$ plotted againts the environment at selection (the points are a subsample). Solid black lines are based on the analytic approximations in the main text solved for the joint evolutionary outcome, dashed black lines are estimated regression slopes and mean values and solid colored lines estimated polynomial regressions. The dotted lines in the rightmost plots indicate 95% probability limits of the distribution of ε_t and additionally, in the upper plot, the optimal phenotype $A + B\varepsilon_t$.

Eqs. (1b) and (4a) define the joint dynamics of ε_t and $\bar{a}_t$ as a Gaussian stochastic process with a bivariate normal stationary distribution (see Fig. 1, upper right plot). The stationary covariance between $\bar{a}_t$ and ε_t generated by genetic evolution in mean elevation is of particular interest, both in itself and, as we shall see, in terms of its effect on the evolution of the mean reaction norm slope and the phenotypic variance. Taking the covariance between the two sums appearing in (1b) and (4a) and using (1a) leads to a recursion equation for $\mathrm{Cov}(\bar{a}_t, \varepsilon_t)$ which solved for the stationary covariance yields

$$\mathrm{Cov}(\bar{a}_t, \varepsilon_t) = \frac{s\alpha(B - \bar{\bar{b}}\alpha^\tau)\sigma_\varepsilon^2}{1 - \alpha(1-s)}. \tag{4c}$$

Note how the covariance, in the case of no plasticity, increases with increasing autocorrelation α towards its maximum limiting value of σ_ε^2 as α goes to 1 (perfect matching), and how the covariance is reduced by the fraction $\bar{\bar{b}}\alpha^\tau/B$ by any given level of plasticity $\bar{\bar{b}}$. This is somewhat different from the covariance derived by Michel et al. (2014) but their result is based on a continuous-time approximations and different model details.

Genetic evolution in mean elevation also produces a stationary variance of $\bar{a}_t$ which can be expressed as function of $\bar{\bar{b}}$ and $\bar{\bar{c}}$ (Appendix S1) of importance in the following subsection.

EVOLUTION OF THE INDEPENDENT ENVIRONMENTAL COMPONENT OF THE PHENOTYPIC VARIANCE

Next consider the evolution of the phenotypic variance through evolution in $\bar{c}_t$, again assuming that the fluctuations in $\bar{b}_t$ are small such that it can be replaced by its long term average equilibrium value $\bar{\bar{b}}$ but with $\bar{a}_t$ covarying with ε_t as described in the previous subsection. It follows that $\bar{c}_t$ must fluctuate around some value $\bar{\bar{c}}$ at which the expected value of the $\bar{c}$-component of the selection gradient (3d) is zero. This leads to

$$-\frac{\bar{\bar{c}}}{\omega^2 + \sigma_z^2(\bar{\bar{b}}, \bar{\bar{c}})}\left(1 - \frac{\mathrm{Var}(\bar{a}_t + \bar{\bar{b}}\varepsilon_{t-\tau} - A - B\varepsilon_t)}{\omega^2 + \sigma_z^2(\bar{\bar{b}}, \bar{\bar{c}})}\right) = 0, \tag{5a}$$

which solved for $\bar{\bar{c}}$ yields two solutions of opposite sign at which the total phenotypic variance is

$$\sigma_z^2(\bar{\bar{b}}, \bar{\bar{c}}) = \mathrm{Var}(\bar{a}_t + \bar{\bar{b}}\varepsilon_{t-\tau} - A - B\varepsilon_t) - \omega^2, \tag{5b}$$

and the solution $\bar{\bar{c}} = 0$ which is only stable stochastically if there are no nonzero solutions. In terms of $\bar{\bar{c}}^2$, the solution is

$$\begin{aligned}\bar{\bar{c}}^2 = &\operatorname{Var}(\bar{a}_t + \bar{\bar{b}}\varepsilon_{t-\tau} - A - B\varepsilon_t) - \omega^2 \\ &- G_{aa} - G_{bb}(\sigma_\varepsilon^2 + \sigma_u^2) - G_{cc} \\ &- \bar{\bar{b}}^2\sigma_u^2 - \sigma_e^2 - B^2\sigma_u^2 + 2\bar{\bar{b}}B\rho_{uv}\sigma_u^2.\end{aligned} \tag{5c}$$

This extends Bull's (1987) result for the evolution of phenotypic variance in three ways. First, in Bull's model, the optimal phenotypic variance is given by the difference between the variance of fluctuations in the environmental optimum (termed π^2 there) and the squared width ω^2 of the Gaussian stabilizing selection function, $\pi^2 - \omega^2$. In the present model with plasticity as well as genetic evolution in mean elevation, π^2 is replaced by the analogous variance of the mismatch between the population mean phenotype $\bar{a}_t + \bar{\bar{b}}\varepsilon_{t-\tau}$ and the populations mean optimal phenotype $E(\theta) = A + B\varepsilon_t$. An expression for this mismatch variance in terms of the solutions for the evolution of $\bar{a}_t$ and $\bar{\bar{b}}$ is given in Appendix S1.

Second and thirdly, the present model includes genetic variance components and environmental variance components associated with plasticity (as in Donaldson-Matasci et al. (2013)) (second and third line of (5c) respectively). Provided that they are sufficiently small, these components do not inflate the total phenotypic variance, but instead replace parts of the independently evolving component $\bar{\bar{c}}^2$ correspondingly, such that the overall sum remains equal to (5b).

EVOLUTION OF MEAN REACTION NORM SLOPE

Approximate solutions for the long term average reaction norm slope once the process has reached its stationary distribution can now similarly be expressed as a function of $\bar{\bar{c}}$ and the above approximate solution for the behaviour of $\bar{a}_t$. Using the same approach as in the previous subsection, it follows that $\bar{b}_t$ too must fluctuate around some long term value $\bar{\bar{b}}$ at which the expected value of the $\bar{b}$-component of the selection gradient (3d) is

zero. This leads to

$$\begin{aligned}\mathrm{Cov}(\bar{a}_t, \varepsilon_{t-\tau}) + \bar{\bar{b}}\sigma_\varepsilon^2 - B\,\mathrm{Cov}(\varepsilon_t, \varepsilon_{t-\tau}) \\ + \sigma_u^2(\bar{\bar{b}} - \rho_{uv}B)\left(1 - \frac{\mathrm{Var}(\bar{a}_t + \bar{\bar{b}}\varepsilon_{t-\tau} - A - B\varepsilon_t)}{\omega^2 + \sigma_z^2(\bar{\bar{b}}, \bar{\bar{c}})}\right) = 0.\end{aligned} \tag{6a}$$

Although we will use this equation in the overall numerical solution (see next section), it is worth considering some further approximations to provide some insights into the behaviour of the solution.

We consider two special cases. First, note that the expected values of the $\bar{c}$- and $\bar{b}$-components of the selection gradient, eqs. (5a) and (6a), share a common factor involving the ratio between the mismatch variance and the sum of ω^2 and the phenotypic variance $\sigma_z^2(\bar{\bar{b}}, \bar{\bar{c}})$. If we assume that bet-hedging is adaptive such that $\bar{c}_t$ has evolved to an optimal value given by one of the nonzero solutions of (5a), it follows that the expected value of the common factor and the expected value of last term of the $\bar{b}$-component of the selection gradient is zero. Using (1a), the remaining part can be solved for $\bar{\bar{b}}$ to give

$$\bar{\bar{b}} = \alpha^\tau B - \frac{\mathrm{Cov}(\bar{a}_t, \varepsilon_{t-\tau})}{\sigma_\varepsilon^2}. \tag{6b}$$

This result expresses the optimal reaction norm slope $\bar{\bar{b}}$ in the presence of genetic evolution in mean elevation, assuming that the environmental component of the phenotypic variance has been free to evolve through changes in $\bar{c}_t$ towards some optimal nonzero value $\bar{\bar{c}}$. Note how the optimal slope $\bar{\bar{b}}$ equals the difference between two regression slopes, the first being the slope of the regression of the population mean optimal phenotype $\bar{\theta}_t = A + B\varepsilon_t$ on the macroenvironment at the time of development $\varepsilon_{t-\tau}$ (as in Gavrilets and Scheiner, 1993), and the second being the slope of the regression of the mean reaction norm elevation $\bar{a}_t$ on the macroenvironment at the time of development.

The second case is the limiting case of the mismatch variance being much smaller than $\omega^2 + \sigma_z^2(\bar{\bar{b}}, \bar{\bar{c}})$. This will arise if fluctuations in the environment are slow (large autocorrelation time T) such that adaptive tracking through either plasticity or through genetic evolution in mean elevation or both are favored. The common factor shared with the expected gradient for $\bar{c}$ is then close to one and the last term in the (6a) can be

approximated by $\sigma_u^2(\bar{\bar{b}} - \rho_{uv}B)$. Solving for $\bar{\bar{b}}$, the small mismatch limit optimal slope becomes

$$\bar{\bar{b}} = \frac{\alpha^\tau \sigma_\varepsilon^2 + \rho_{uv}\sigma_u^2}{\sigma_\varepsilon^2 + \sigma_u^2} B - \frac{\mathrm{Cov}(\bar{a}_t, \varepsilon_{t-\tau} + u)}{\sigma_\varepsilon^2 + \sigma_u^2}. \tag{6c}$$

This is again the difference between two regression slopes but now between variables at the individual instead of at the population level. The first is the slope of the regression of the random phenotypic optimum in the microenvironment at time of selection $\theta = A + B(\varepsilon_t + v)$ experienced by a randomly chosen individual (2a) on the microenvironment of the same individual at the time of development $\varepsilon_{t-\tau} + u$. The second is the slope of the regression of the reaction norm elevation a, also on the microenvironment at time of development.

The difference between predictions (6b) and (6c) can be interpreted as follows. In the case when bet-hedging is adaptive (6b), the increase in the phenotypic variance with increasing levels of plasticity $\bar{b}$ caused by the microenvironmental variability has no fitness consequences as long as the total phenotypic variance can evolve to its optimal value given by (5c) through joint evolution of $\bar{c}_t$. More generally, a steeper slope may also lead to a smaller phenotypic variance, or more precisely a smaller phenotype-optimum variance $\sigma_z^2(\bar{b}_t, \bar{c}_t)$, if the correlation ρ_{uv} is positive, but again, since the total phenotypic variance will evolve freely towards its optimal value, the pattern of microenvironmental variation does not influence the mean reaction slope produced. In contrast, in the small mismatch limit (6c), bet-hedging is maladaptive, and stabilizing selection will make $\bar{c}_t$ fluctuate around zero while also creating additional selection against large values of the slope of the reaction norm $\bar{b}_t$ through its effect on the phenotypic variance. However, if the correlation ρ_{uv} between the individual level microenvironmental deviations at development and selection u and v is sufficiently high, a higher mean reaction norm slope $\bar{b}$ will evolve. For intermediate values of the mismatch variance, we shall see that the mean reaction norm slope in general evolves towards an intermediate value between the predictions of (6b) and (6c). The solutions obtained in the above two special cases should thus be seen as upper and lower bounds on the mean reaction norm slope.

Joint Evolutionary Outcome

Employing (1a), (4b), (S1.2) and (S1.4), eqs. (4c), (S1.3), (5c), and (6a) constitute a non-linear system of four equations expressing the mutual dependencies between $\mathrm{Cov}(a_t, \varepsilon_t)$, $\mathrm{Var}(a_t)$, $\bar{\bar{b}}$ and $\bar{\bar{c}}^2$. In general the solution can only be computed numerically. The numerical solution yields values for these quantities as functions of the basic model parameters $\mathbf{G}$, α, σ_ε^2, σ_u^2, ρ_{uv}, σ_e^2, τ, ω, A and B. This solution, shown in Figs. 2 to 5 as function of different parameter values, represents the joint evolutionary response taking into account a number of feedback loops between the three forms of evolutionary response, in the limiting case considered in the previous section.

Figs. 2 to 5 are, without loss of generality, based on $A = 0$ and $B = 1$ and $\omega^2 = 1$ such that all parameters with dimensions involving that of the phenotype are measured in units of the width ω of the Gaussian fitness function. It can also be noted that the effect of σ_e^2 is confounded with ω^2 since these parameters always appear through their sum in the selection gradient. Thus, we may also without loss of generality set $\sigma_e^2 = 0$. For the time lag between development and selection we choose a value of $\tau = 0.5$, which should be a reasonable average value for most organisms.

In the following, genetic evolution and plasticity are quantified by their associated components in the slope of the regression of the mean phenotype on the environment at selection

$$E(\bar{z}_t|\varepsilon_t) = E(\bar{a}_t + \bar{\bar{b}}\varepsilon_{t-\tau}|\varepsilon_t) = A + \left(\frac{\mathrm{Cov}(\bar{a}_t, \varepsilon_t)}{\sigma_\varepsilon^2} + \bar{\bar{b}}\alpha^\tau\right)\varepsilon_t, \tag{7}$$

relative to the slope B of the relationship between the mean optimal phenotype and ε_t, $E(\theta_t|\varepsilon_t) = A + B\varepsilon_t$.

NO MICROENVIRONMENTAL VARIABILITY

We begin by considering the model without any microenvironmental variability ($\sigma_u^2 = 0$). Fig. 2 shows the three evolutionary responses as functions of increasing autocorrelation time T for three different values of the genetic variance in reaction norm elevation G_{aa}. Large environmental fluctuations ($\sigma_\varepsilon^2 = 3$) relative to the squared width $\omega^2 = 1$ of the

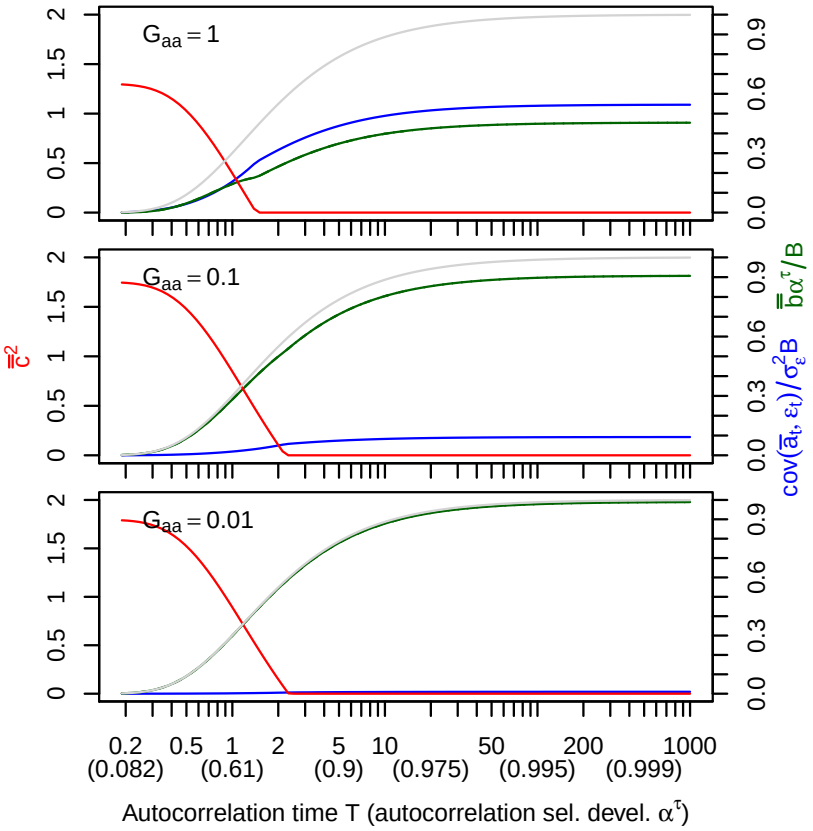

Figure 2: Joint evolutionary responses to the autocorrelation time T in the environmental optimum ε_t (generations) and the corresponding correlation between environments at development and selection α^τ in the absense of microenvironmental variation ($\sigma_u^2 = 0$), for genetic variance in reaction norm elevation G_{aa} equal to 1, 0.1 and 0.01 (upper, middle and lower plot). The red curve represents the level of bet-hedging (quantified by the component $\bar{c}^2$ of the phenotypic variance, left axis). Other curves (right axis) represents regression slopes of the mean phenotype on the environment at time of selection resulting from genetic evolution in mean elevation ($\mathrm{Cov}(\bar{a}_t, \varepsilon_t)/\sigma_\varepsilon^2$), blue curve), plasticity ($\bar{b}\alpha^\tau$, darkgreen curve), and their sum (light grey curve). Other parameter values used are $\sigma_\varepsilon^2 = 3$, $\sigma_u^2 = 0$, $\sigma_e^2 = 0$, $\tau = 0.6$, $G_{bb} = 0.05$, $G_{cc} = 0.05$, $A = 0$ and $B = 1$.

stabilizing selection function are assumed such that bet-hedging is adaptive in some parts of the parameter space shown.

The assumption that a single environmental variable determines both development and selection makes the autocorrelation between the macroenvironments at development and selection α^τ coupled to time scale of temporal fluctuations in the phenotypic optimum $T \approx 1/(1-\alpha)$. This creates a joint evolutionary balance between the evolving reaction norm slope and the amount of genetic evolution in mean elevation since both evolutionary responses are favored by increasing autocorrelation. Although a partial closed form solution for the joint evolutionary outcome is available in the case considered here ($\sigma_u^2 = 0$) (Appendix S2), the relative contributions of plasticity and genetic evolution in mean elevation to overall phenotypic adaptation is well described by the limiting values (see Appendix S2) in the case of slow fluctuations in the environment. As $\alpha \to 1$,

$$\frac{\bar{\bar{b}}\alpha^\tau}{B} \to (1 - 2s\tau), \tag{8a}$$

$$\frac{\mathrm{Cov}(\bar{a}_t, \varepsilon_t)}{\sigma_\varepsilon^2 B} \to 2s\tau, \tag{8b}$$

as also indicated by Fig. 2, with no mismatch between the mean and optimal phenotype (light grey curves). The proportion of overall phenotypic adaptation attributable to genetic evolution in mean elevation thus increases with G_{aa}, the strength of stabilizing selection ω^2 and increasing time lag τ through the quantity $2s\tau$ where s simplifies to

$$s = \frac{G_{aa}}{\omega^2 + G_{aa} + G_{bb}\sigma_{\bar{\varepsilon}}^2 + G_{cc} + \sigma_e^2}. \tag{8c}$$

Conversely, plasticity is favored by a short delay between development and selection (τ small), weak selection (ω^2 large) and small genetic variance in reaction norm elevation G_{aa}. Empirical studies suggest typical G_{aa}/ω^2 values less than 0.1 (Kingsolver and Hoekstra, 2001) which would correspond to $s = 0.091$ if other genetic variances are small. For an intermediate time lag of $\tau = 0.5$, 9% of overall adaptation would thus be attributable to genetic evolution in mean elevation and 91% to plasticity (Fig. 2, middle plot)

Turning to the leftmost region of the parameter space shown in Fig. 2, for autocorrelation times less than about 3 generations, as adaptive tracking through plasticity and

genetic evolution in mean elevation becomes sufficiently small and the mismatch variance sufficiently large, diversifying bet-hedging (red curves) begins to occur as a joint evolutionary outcome. In the limiting case of $\alpha = 0$ the environment simplifies to a white noise process that prevents any adaptive tracking from occurring. As in Bull (1987), for small G_{aa}, the phenotypic variance then evolves to a maximum value of almost $\sigma_\varepsilon^2 - \omega^2 = 2$ out of which a component equal to $\bar{\bar{c}}^2 = 1.8$ is attributable to evolution in $\bar{c}_t$ (lower plot in Fig. 2) with the remaining part being genetic and non-evolving environmental components (see (3b) and the figure caption for details of all parameter values used). Increasing G_{aa} in this limiting case, however, has both a direct negative and an indirect somewhat obscure positive effect on $\bar{\bar{c}}^2$. First, as the genetic variance in reaction norm elevation increases from $G_{aa} = 0.01$ up to $G_{aa} = 1$ (leftmost part of Fig. 2), this increases the total phenotypic variance by 0.99, which is compensated by a decrease by the same amount in $\bar{\bar{c}}^2$, see equation (5c). However, increasing G_{aa} also leads to maladaptive genetic evolution in mean elevation, an effect noted by Lande and Shannon (1996), because any response in mean elevation to past selection will be uncorrelated with the current phenotypic optimum. The resulting fluctuations in $\bar{a}_t$ creates an additional increase in the mismatch variance beyond σ_ε^2 given by (S1.3) which in the white noise case simplifies to $\mathrm{Var}(\bar{a}_t) = s\sigma_\varepsilon^2/(2-s) = 0.5000245$ for the parameter values used. The net effect, $0.99 - 0.5000245$, reduces $\bar{\bar{c}}^2$ from its value of 1.8 (lower plot in Fig. 2) to a value of 1.3100245 for $G_{aa} = 1$ (upper plot).

UNCORRELATED MICROENVIRONMENTAL DEVIATIONS

Next we consider the effect of microenvironmental variation (Fig. 3) assuming that the variance of microenvironmental deviations u and v affecting development and selection is $\sigma_u^2 = 1$, that is, one third of the variance in the macroenvironment $\sigma_\varepsilon^2 = 3$. We also begin by assuming that microenvironmental deviations u and v are uncorrelated. This may be realistic for organisms with continuous movement experiencing spatially heterogeneous environments over spatial scales much shorter than typically dispersal distances such as many insects (Clobert et al., 2012). Other parameter values remain unchanged from the

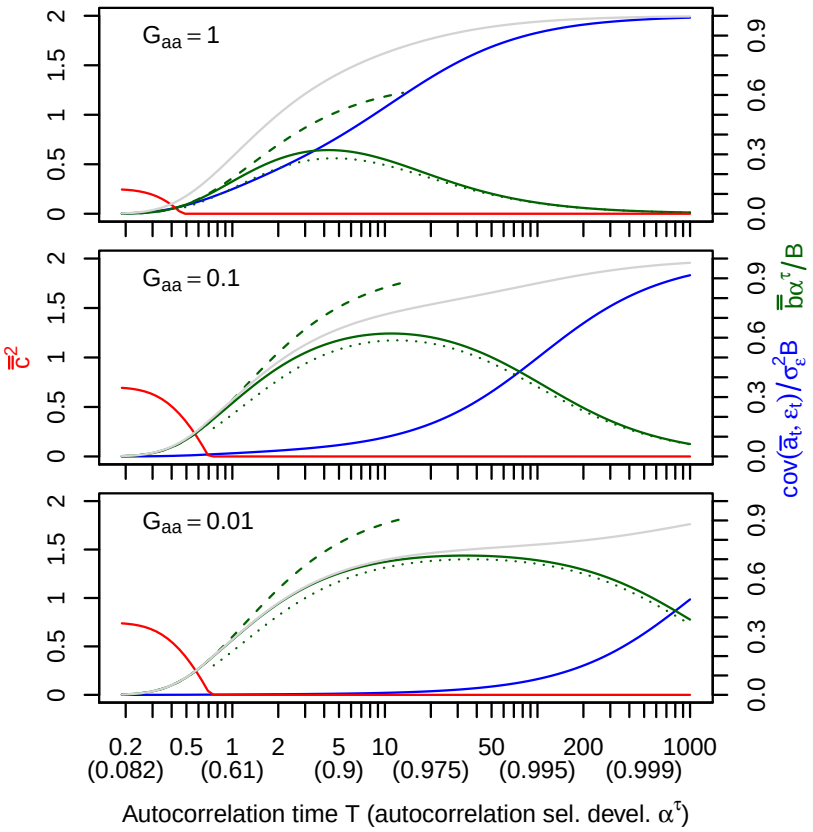

Figure 3: Same as Fig. 2 but with uncorrelated microenvironmental deviations affecting development and selection with variance $\sigma_u^2 = 1$. Also shown are joint solutions for the levels of plasticity predicted by approximations (6b) (bet-hedging jointly adaptive, dashed darkgreen curve) and (6c) (small mismatch variance limit, dotted darkgreen curve).

previous subsection.

This microenvironmental variability increases the phenotypic variance, or more precisely, the individual level phenotype-optimum variance $\sigma_z^2(\bar{\bar{b}}, \bar{\bar{c}})$ by $(B^2 + \bar{\bar{b}}^2)\sigma_u^2$, see (3b). This in turn lead to selection for smaller values of $\bar{\bar{c}}^2$ (5c). The individual level variance in the phenotypic optimum $B^2\sigma_u^2$ can alternatively be interpreted as weaker stabilizing selection changing ω^2 to $\omega^2 + B^2\sigma_u^2$. Both interpretations imply a reduction in the value of $\bar{\bar{c}}^2$ in Fig. 3 relative to the corresponding values in Fig. 2, in some cases all the way down to zero, reducing the region of the parameter space in which bet-hedging is adaptive and enlarging the region in which selection for reduced phenotypic variance (environmental canalization) is present.

Turning to the reaction norm slope $\bar{\bar{b}}$, microenvironmental variability does not always have an effect. Within the region in which bet-hedging is adaptive, with little or no genetic evolution in mean elevation (lower plot in Fig. 3), the mean reaction norm slope remain unchanged from Fig. 2 and equal to the value $\bar{\bar{b}} = \alpha^\tau$ as predicted by (6b) for small G_{aa} (Fig. 3, lower plot). In the vicinity of this region, the level of plasticity predicted by (6a) (solid dark green curve) remains close to the solution based on approximation approximation (6b) (dashed green curve), but then gradually transition towards the value predicted by the small mismatch variance limiting approximation (6c) (dotted green curve).

For autocorrelation times of 10 generations or more, adaptive tracking through both plasticity and genetic evolution becomes extensive and the mismatch variance small, generating a penalty on the phenotypic variance and stronger selection against high reaction norm slopes. This shifts the joint evolutionary balance between these two evolutionary responses in favor of genetic evolution, making this the predominant evolutionary response for autocorrelation times T greater than about 100 generations for $G_{aa} = 0.1$.

CORRELATED MICROENVIRONMENTAL DEVIATIONS

For more sessile organisms such as many species of plants, development and selection may occur at the same spatial location. This may give rise to a high correlation between

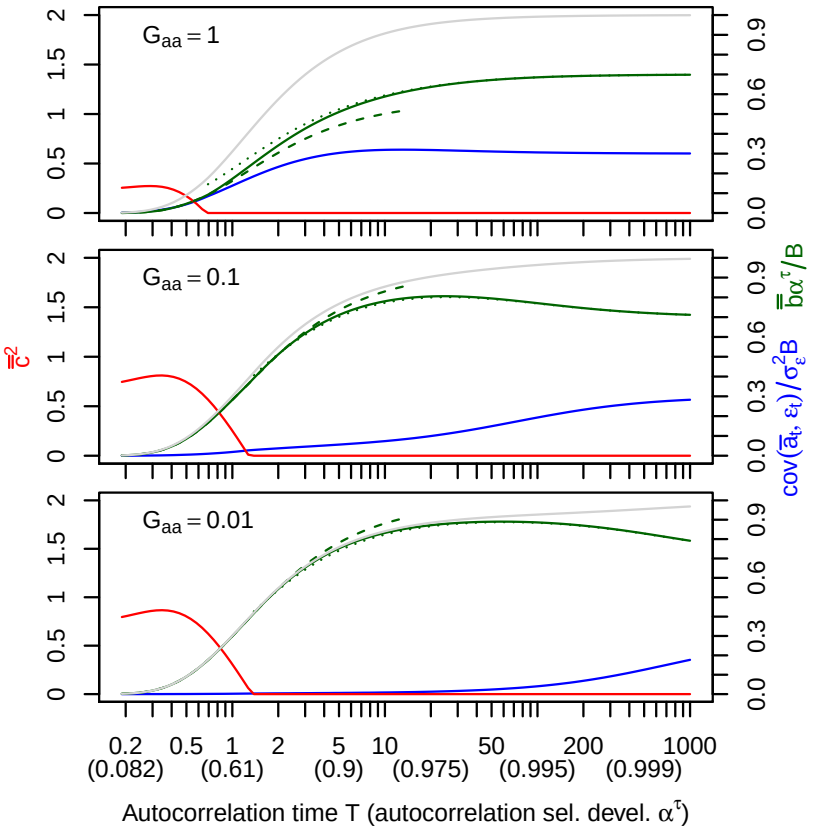

Figure 4: Same as Fig. 3 but with an added correlation $\rho_{uv} = 0.7$ between microenvironmental deviations u and v.

the microenvironmental deviations u and v at the time of development and selection. Fig. 4 shows the joint evolutionary response given a value of $\rho_{uv} = 0.7$ for this parameter, again, as function of the macroenvironmental autocorrelation time T (or autocorrelation between the macroenvironments at the time of development and selection) and for the same genetic variances in reaction norm elevation G_{aa}.

Not surprisingly, compared to Fig. 3, the same or a greater amount of plasticity evolves in all parts of the parameter space. For moderate or low values of G_{aa} such that the amount of tracking through mean elevation is limited, there is first an increase in the reaction norm slope caused by increasing macroenvironmental autocorrelation but again, as in Fig. 3, for high levels of plasticity (greater than $\rho_{uv}B$), the phenotypic variance increase with increasing values of the slope which shifts the joint evolutionary balance in favor of genetic evolution. As apparent from Fig. 3 and as shown in Appendix S2, in the limit of slow fluctuations ($T \to \infty$, $\alpha \to 1$), the mean reaction norm slope and the slope of the regression of mean elevation on the environment at the time of selection evolves to

$$\bar{\bar{b}} = \rho_{uv}B, \tag{9a}$$

$$\frac{\mathrm{Cov}(\bar{a}_t, \varepsilon_t)}{\sigma_\varepsilon^2} = (1 - \rho_{uv})B. \tag{9b}$$

This result has a simple intuitive explanation. When fluctuations in the macroenvironment are very slow the model is essentially equivalent to a model with a constant macroenvironment with the population mean phenotype $\bar{z}_t$ always matching the constant mean phenotypic optimum $\bar{\theta}_t$. The evolving level of plasticity is then a result of the pattern of microenvironmental variability only. Setting σ_ε^2 and $\mathrm{Cov}(\bar{a}_t, \varepsilon_{t-\tau})$ in (6c) both equal to zero we then arrive at (9a).

Does increased plasticity, through a reduction in the mismatch variance, also reduce the region of the parameter space in which bet-hedging is adaptive? Somewhat surprisingly, comparison of the region where the red curves are positive in Fig. 4 to those in Fig. 3 shows that this region is enlarged by the positive correlation ρ_{uv} between the microenvironmental deviations u and v. This results follows from the dependency of the individual level phenotype-optimum variance $\sigma_z^2(\bar{b}_t, \bar{c}_t)$ on $\bar{b}_t$. For positive ρ_{uv}, the vari-

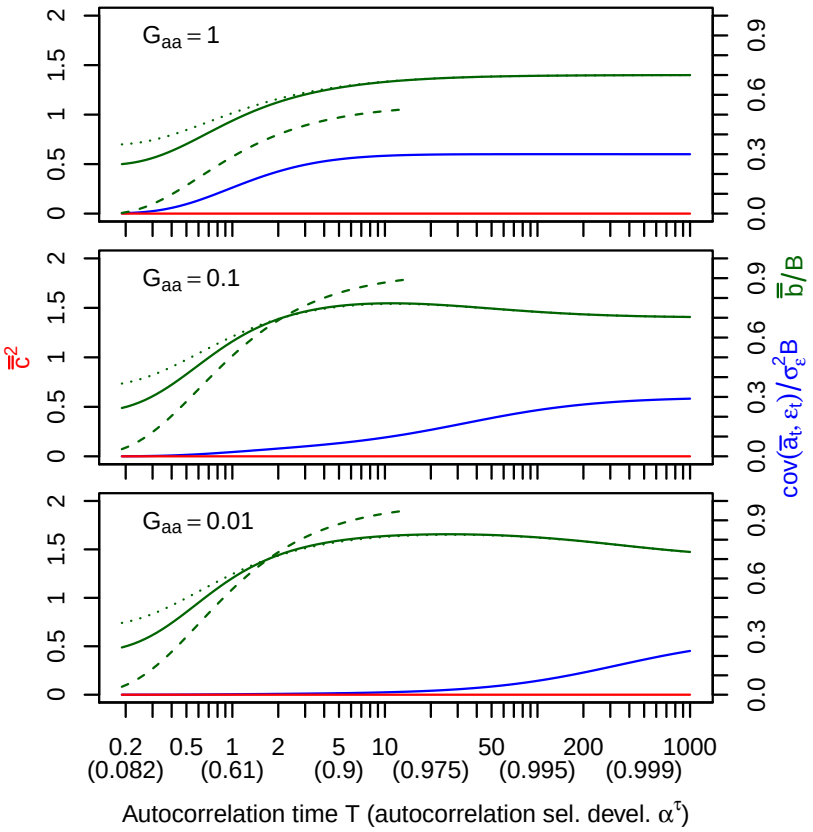

Figure 5: Same as Fig. 4 but with the variance of macroenvironmental fluctuations reduced from $\sigma_\varepsilon^2 = 3$ to $\sigma_\varepsilon^2 = 1$ such that diversifying bet-hedging is no longer adaptive for short macroenvironmental autocorrelation times. Note also that the darkgreen green curves represents $\bar{\bar{b}}$ only.

ance is at its minimum for $\bar{b}_t = \rho_{uv}$ (see (3b)). Therefore, the steeper slope resulting from higher macroenvironmental autocorrelation translates to a smaller phenotype-optimum variance such that the net effect of increased plasticity is increased selection for higher values of $\bar{c}^2$ and an enlargement of the region of adaptive bet-hedging.

The level of plasticity is again, within the region of adaptive bet-hedging, not influenced by the pattern of microenvironmental variability but only by the macroenvironmental autocorrelation between development and selection. This somewhat counterintuitively includes no influence of ρ_{uv}.

MODERATE ENVIRONMENTAL FLUCTUATIONS

Diversifying bet-hedging requires that the variance of environmental fluctuations in the selective optimum, or as shown here, that the variance of the mismatch between the mean

phenotype and the mean optimum is larger than the squared width ω^2 of the stabilizing selection function. In many situations, this condition is not likely to be fulfilled. In Fig. 5 we consider the same parameter values as in Fig. 4 but with the macroenvironmental variance σ_ε^2 reduced from 3 to 1. Given $\omega^2 = 1$, diversifying bet-hedging is then no longer adaptive for any value of the macroenvironmental autocorrelation α. This also makes evolution of the mean reaction norm slope $\bar{b}_t$ dependent on the pattern of microenvironmental variation everywhere. Consequently, a positive slope evolves as a result of the microenvironmental correlation $\rho_{uv} = 0.7$ also when the macroenvironmental correlation between development and selection is 0. The reaction norm slope in this case takes an intermediate value of 0.22, in-between zero as predicted by (6b) (dashed green curve) and $\rho_{uv}\sigma_u^2/(\sigma_\varepsilon^2 + \sigma_u^2) = 0.35$ as predicted by (6c) (dotted dark green curve).

For higher macroenvironmental autocorrelation α, the joint evolutionary outcome is qualitatively similar to that in Fig. 4 with $\bar{\bar{b}}$ again tending to the limiting value $\rho_{uv}B = 0.7$ and $\mathrm{Cov}(\bar{a}_t, \varepsilon_t)/\sigma_\varepsilon^2$ to $1 - 0.7$.

Discussion

MAIN FINDINGS

Here, genetic evolution, plasticity and the evolution of diversifying bet-hedging have been considered jointly within a unified quantitative genetic framework. Simple analytic approximations expressing the mutual dependency between genetic evolution, plasticity and the evolving phenotypic variance have been obtained, containing previous results (Lande and Shannon, 1996; Gavrilets and Scheiner, 1993; Bull, 1987) as special cases. As in Michel et al. (2014), a central quantity is the covariance between $\bar{a}_t$ and ε_t representing Darwinian genetic evolution in mean reaction norm elevation. This covariance is reduced by plasticity by a fraction $\bar{\bar{b}}\alpha^\tau/B$ (4c), and by reduced heritability through any increase in the environmental component of the phenotypic variance resulting from plasticity, diversifying bet-hedging or both ((3b) and (4b)). Genetic evolution in mean elevation and the evolving phenotypic variance similarly feeds back on the evolving level of plasticity. In

parts of the parameter space with decanalizing selection on the phenotypic variance (fast and large environmental fluctuations), the reaction norm slope is reduced below Gavrilets and Scheiner's (1993) prediction by an amount equal to the slope of the regression of mean elevation on the environment at development at the population level (6b). Elsewhere, through the effects of selection for reduced phenotypic variance, the reaction norm slopes evolves towards the difference between the same regression slopes but between random quantities at the individual level (6c). Bet-hedging arising through selection for increased phenotypic variance depends not on the the variance of the phenotypic optimum as in Bull's (1987) model but instead on the variance of the mismatch between the optimal and mean phenotype. This mismatch variance is reduced by adaptive tracking generated by the other responses. Solving for the joint evolutionary outcome, there is considerable overlap between the different responses (Figs. 2 to 5) with plasticity emerging as the predominant evolutionary response in large parts of the parameter space value and with bet-hedging restricted to environmental fluctuations with autocorrelation times less than two generations.

These theoretical predictions appear largely consistent with more verbal arguments for the notion that plasticity plays a more important role in evolution than previously realized (West-Eberhard, 2003), as well as empirical findings. Merilä and Hendry's (2014) meta-review summarizes 11 reviews of the role of plasticity and genetic evolution in response to climate change in different taxonomic groups. An overall conclusion from these reviews is that plasticity often appear to make a strong contribution to phenotypic trends whereas genetic contributions seems weaker and less common. In birds (Charmantier and Gienapp, 2014), plasticity was documented to be involved in observed phenotypic changes in timing of breeding or timing of migration in 18 out of 18 studies in which this was tested, 12 of which also documented an adaptive value of the changes. Genetic changes, on the other hand, did not contribute significantly in 3 of the studies that tested for this. In mammals, observed responses in 12 studies were primarily due to plasticity with only one study documenting genetic changes (Boutin and Lane, 2014). In plants, in contrast, genetic evolution appear to make a stronger contribution, with both plasticity and genetic

evolution contributing significantly in 26 out of the 38 studies reviewed by Franks et al. (2014). Given that movement between development and selection in many plants should be limited, creating a stronger correlation ρ_{uv} between the microenvironments at development and selection, this is surprising in light of the predictions of the present model (see Fig. 4 and 5). Seventeen of the studies reviewed by Franks et al. (2014) are based on space for time substitutions, however, so in reality, many of these studies document local genetic adaptations which are outside the scope of the present panmictic model, see 'Spatial Model Extensions' for further discussion below. Other possible explanations are costs or other limitations to plasticity or autocovariance with non-exponential decay (see below) which can both shift the joint evolutionary balance away from plasticity in favor of temporal genetic evolution.

Few empirical studies make an attempt at estimating the relative contribution of genetic evolution and plasticity in quantitative terms. A notable exception is Réale et al. (2003) who found plasticity and genetic evolution to account for an advancement in breeding date in red squirrels through changes in food abundance equal to 3.7 and 0.8 days per generation, respectively. As highlighted by Merilä and Hendry (2014), new inferential methods such as Crozier et al. (2011) disentangling these contribution from the pattern of fluctuations seen in phenotypic times series, perhaps combined with methods for estimating fluctuating selection as in Calsbeek (2012); Engen et al. (2012); Engen and Saether (2014); Chevin et al. (2015), are clearly needed.

The limited role of diversifying bet-hedging predicted here also appear consistent with the limited amount of conclusive empirical evidence restricted to a few studies of Chordata species and some semelparous plant species (Childs et al., 2010; Simons, 2011). However, the lack of empirical evidence may also reflect the arduous requirements of any empirical test of whether observed levels of phenotypic variation reflects the observed level of fluctuating selection. Interactions with other response modes adds to the difficulties of such empirical tests.

SIMILAR THEORETICAL MODELS

Focusing on the same overall question as here but also including reversible plasticity and conservative bet-hedging as response modes, Botero et al. (2014), using individual-based simulations, reached conclusions resembling results derived here but with the different adaptive responses showing little overlap across different parts of the parameter space. These authors also investigated transitions between different points in parameter space corresponding to different patterns of environmental fluctuations, perhaps resulting from anthropogenic climate change (Tebaldi et al., 2006; Hansen et al., 2012). Worryingly, although such transitions could be accomodated by evolution when occurring within the region of a given single response mode, most transitions across boundaries between regions of different response modes resulted in rapid population collapse and often extinction. These results may easily be a result of the model assumptions used, however, including the step function used to model a cost of plasticity, a single modifier locus s switching plasticity on and off at a threshold genotypic value of 0.5, and the assumption that the allelic values at the locus determining the reaction norm slope are somehow reset to zero whenever s goes below the 0.5-threshold in any given individual.

Also using individual-based simulations, Scheiner (2014a,b) found developmental instability being disfavored by increasing autocorrelation, possibly through genetic evolution at non-plastic loci or through closer tracking as a result of plasticity. Scheiner models an evolving phenotypic variance by a normally distributed random term representing developmental instability, where the standard deviation is a polygenic trait influenced additatively by 5 loci. Although in effect being similar to the approach used here and in Gavrilets and Hastings (1994), the standard deviation is constrained to be non-negative by truncating the distribution of mutational effects. Combined with a mutation rate set to 10% per loci per generation, this in effect produces a strong directional mutational force possibly explaining why Scheiner (2014a) observed considerable developmental instability also in cases where the variance of temporal fluctuations is below the Bull (1987) threshold.

The list of evolutionary response modes considered here is not exhaustive. If selec-

tion is strong and the autocorrelation across generations is high, the phenotype of a surviving parent will be informative about the selective environment of a given offspring. Maternal effects are then predicted to evolve, in particular, if within-generation plasticity is somehow limited through costs (Ezard et al., 2014; Kuijper and Hoyle, 2015). Maternal effects can be thus be seen as a response mode in-between genetic evolution and within-generational plasticity in efficiency, having mutual dependencies with other response modes similar to the ones considered here. For a large sudden shift in environmental conditions, however, both positive maternal effects and increased plasticity are predicted to contribute during transient phenotypic evolution (Kuijper and Hoyle, 2015), both slowing down genetic evolution as in Lande (2009). In terms of evolution of the phenotypic variance, an interesting empirical finding is negative maternal effects (*sensu* the single trait model in Kirkpatrick and Lande, 1989), as predicted by theory in slowly changing environments through its canalizing effect on the phenotypic variance (see Hoyle and Ezard, 2012; Ezard et al., 2014, and references therein).

CONSTRAINTS ON b AND c

Using a simulation model similar to Scheiner (2014a,b), Scheiner and Holt (2012) and Scheiner (2013) observed that strong fluctuating selection theoretically may select for reaction norm slopes seemingly steeper than the optimal value predicted by theory, so called hyperplasticity, in effect a form of diversifying bet-hedging. This appears to be a result of not accommodating independent evolution of the phenotypic variance as we have done here. Without such a component, increased phenotypic variance can only be generated by steeper-than-optimal, hyper-plastic reaction norms. Adding loci influencing developmental instability without pleiotropic effects on plasticity in their model, a shallower reaction norm slope evolved (Scheiner, 2014b). If the same loci influence both plasticity and developmental instability through pleiotropy, however, hyperplasticity again evolved (Scheiner, 2014b). The realism of such genetic architectures is an open question. Given the chaotic behaviour of nature, pseudorandom microenvironmental variables in the surroundings of most organisms or pseudorandom variables internal to the organism

as defined in (2b) should be abundant as should mutations with effects depending on at least one such variable. This suggests that the assumption of G_{bc} being small should be reasonable. A nonzero genetic covariance between b and c again also appear to only affect the transient evolutionary trajectory, with hyper-plasticity being present only during a transient phase, but not at the joint evolutionary outcome once the process has reached its stationary distribution (Fig. S4.7).

This form of link to developmental instability is more conventionally thought to reduce the evolving level of plasticity, however (DeWitt et al., 1998; Tonsor et al., 2013). It is distinctly different from the relationship between the phenotypic variance and plasticity introduced here through the effect of variation in the microenvironment $\varepsilon_{t-\tau} + u$ surrounding each individual at the time of development in that developmental instability involves independent variability at the individual level possibly of purely internal origin. Although both mechanisms leads to similar reduction in the reaction norm slope, a genetic correlation between developmental instability and plasticity, would only reduce and slow down evolution of the reaction norm slope transiently, provided that the genetic correlation is not perfect (Fig. S4.8).

Alternatively, constraints can be in the form of costs. If including a cost of plasticity modeled by an additional factor $\exp\{-b^2/(2\omega_b^2)\}$ in (2c) as in Chevin et al. (2010), this would reduce the evolving mean reaction norm slope $\bar{\bar{b}}$ through the addition of ω_b^2 to the denominator in (6c) (results not included here). Similarly, as in Zhang and Hill (2005), a cost of canalization (reduced developmental instability) could be modeled through an additional factor $\exp\{-C/V_E\}$ where V_E (corresponding to cy here) in the usual way is the environmental component of the phenotypic variance. Within such a framework, a link between plasticity and developmental instability as discussed in (DeWitt et al., 1998) could instead be imposed through a joint cost function such that the cost of canalization depend on plasticity.

GENETIC VARIANCES OF b AND c

The analytic approximations derived here are only exact in the limit obtained as the genetic variances in b and c tend to zero (as verified in Appendix S4, Fig. S4.1). Empirical evidence, however, mostly from animal and plant breeding, suggests that genotype by environment interactions are ubiquitous (see e.g. Schlichting, 1986) but typically account for a moderate proportion of the total phenotypic variance. For example, in salt marsh cord grass, averaging over 8 different traits, genotype and genotype by environment interactions accounted for 19 and 5% of the total phenotypic variance, respectively (Silander, 1985). In two studies reviewed by de Jong and Gavrilets (2000), genotype by environment interaction (with temperature) accounted for about 5 to 24% of the total genetic variance in wing/thorax ratio in *Drosophila buzzati*, *D. aldrichi* and *D. melanogaster*. These values are similar to the relative magnitude between components of the phenotypic variance accounted for by plasticity $G_{bb}\sigma_\varepsilon^2$ and additive genetic variance G_{aa} used in Fig. 1 for which the analytic approximations are in close agreement with the results of stochastic simulations.

That plasticity accounts for a smaller proportion of the total phenotypic variance than the genetic variance in elevation also seems reasonable given that genetic variation in plasticity requires mutations with effects depending on a specific environmental variable. In contrast to genetic variance in non-plastic traits which may evolve to a higher value in fluctuating autocorrelated environments (Bürger, 1999), current theory for the maintenance of genetic variance in reaction norm slopes by mutation-selection balance (de Jong and Gavrilets, 2000) predicts a reduction in both variances with increasing environmental variability. Interestingly, results here (Fig. S4.3C, Appendix S4) suggest that the net effect of increasing genetic variance in reaction norm slopes is mostly maladaptive within the usual range of environmental conditions. Further simulation results (Appendix S4 and Fig. S4.6) suggest that the same holds for the genetic variance in c.

COUPLING

Several authors (Moran, 1992; Tufto, 2000b; Botero et al., 2014; Michel et al., 2014) have assumed that a reduced correlation between the environments at development and selection arise as a result of the phenotype and the selective optimum being determined by different environmental variables, say daily precipitation and daily mean temperature. This evolutionary outcome, however, would require either strong cross-correlations between the different environmental variables involved in development and selection over time lags of length τ, significant costs precluding developmental sensitivity to specific environmental variables, or genetic constraints slowing down evolution of specific sensitivities. A reasonable evolutionary argument therefore suggests that development and selection in most cases should be determined be approximately the same variable or linear combination of variables. For example, in birds where the optimal timing of breeding depends on early spring temperature, it seems likely that the cue on which the reaction norm depends would be early spring temperature rather than, say early spring precipitation which would likely be less correlated with the phenotypic optimum. This is not inconsistent with the finding that a variable such as photoperiod is involved in the timing of many phenological traits such as breeding and hibernation in diverse groups of organisms (Bradshaw and Holzapfel, 2001, 2008). Photoperiod varies deterministically and hence does not carry information about current or future environmental conditions at a given time of the year and can instead only serve as a pivotal variable against which the timing of seasonal events is aligned. Further empirical studies may warrant the development of more explicit theoretical models involving multivariate environmental fluctuations and multivariate norms of reactions. Modelling costs as some general function of the coefficients of a multivariate norm of reaction, say by extending the cost function in Chevin et al. (2010) to a multivariate Gaussian, however, would rapidly lead to a huge number of additional parameters. Here, I have therefore restricted the analysis to the univariate model of micro- and macroenvironmental variation expected from the above evolutionary argument, with a single environmental variable determining development and selection.

This assumption leads to coupling of the conditions selecting for increased plasticity

(a high correlation α^τ between the environments at selection and development) and the conditions favoring adaptive genetic evolution in mean reaction norm elevation (slow environmental fluctuations). For realistic values of G_{aa}/ω^2 of the order of 0.1 as suggested by empirical studies (Kingsolver and Hoekstra, 2001), the evolutionary mutual dependency between the two forms of phenotypic adaptations result in plasticity being the predominant evolutionary response (Fig. 2).

The relative magnitude of plasticity (including different forms of transgenerational plasticity) versus genetic evolution in mean elevation must depend on the exact functional form of the autocovariance function, however. Ezard et al. (2014) modeled varying degrees of unpredictability by adding fast autocorrelated fluctuations with negligible autocorrelation across generations on top of slower deterministic, sinusoidal fluctuations. Although termed microenvironmental and generating some of the same effects as microenvironmental variability in the model presented here, these fast fluctuations, experienced identically by all individuals of a given generation in Ezard et al.'s (2014) model, constitute a macroenvironmental temporal autocovariance function of a particular form with a sharp rise in autocovariance as the lag approaches zero. This is markedly different from the admittedly simple autoregressive model exhibiting exponential decay assumed here and elsewhere (Lande and Shannon, 1996; Michel et al., 2014). Other possibilities autocovariance functions exhibiting, say, Gaussian decay would make the correlation between development and selection even tighter, shifting the joint evolutionary balance in favor of plasticity. The realism of either of these models is an open empirical question.

SPATIAL MODEL EXTENSIONS

The evolutionary implication of coupling is complicated by the the pattern of microenvironmental variation (represented by u and v) which can be interpreted as permanent spatial environmental heterogeneity influencing each individual at the time of development and selection. Here, the analysis has been restricted to the case of variation over spatial scales sufficiently short relative to typical dispersal distances such that spatial genetic differentiation is negligible and the population can be treated as panmictic. In

addition, I have assumed hard selection (see Appendix S3), that is, global density regulation occurring after dispersal such the contribution from each microenvironment is proportional to local mean fitness. For the more general case of heterogeneity over spatial scales longer than typical dispersal distances (see e.g. de Jong, 1999; Scheiner, 2013) it seems likely that plasticity and spatial local adaptations through genetic differentiation in mean reaction norm elevation would evolve jointly in many of the same ways as in the case of temporal variability alone treated here. Just like the temporal delay τ between development and selection being some fraction of the generation length, the distance between development and selection, being some fraction of lifetime dispersal, would again couple the conditions favoring plasticity to the conditions favoring local adaptation. If dispersal occurs over distances shorter than the scale of spatial heterogeneity, this would again lead to high correlation between the environment at development and selection while at the same time favoring local adaptation by limiting gene flow between localities with divergent phenotypic optima. Given the dependency of local adaptation on ω and G_{aa}, it thus seems plausible, by the same argument as for the temporal explicit model treated here, that the joint evolutionary outcome in a more general spatio-temporal explicit model would be dominated by plasticity too. Within such a model, however, the roles of temporal and spatial variability assumed here might be reversed, with fast temporal fluctuations playing a role similar to that of microenvironmental variability in the current model (Fig. 3) through the penalty it would impose on plasticity. If the scale of spatial variability is large, this would shift the joint evolutionary balance away from plasticity in favor of local genetic differentiation, possibly resolving some of the apparent discrepancy between predictions of the present model and empirical patterns in plants (Franks et al., 2014) discussed earlier.

In terms of possible effects of spatial variation on bet-hedging, it is useful to consider of the case of soft selection, that is, population density regulated to a constant number locally in each microenvironment after selection, arguably more realistic in the non-panmictic than in the panmictic case. The effect of spatial variation then becomes identical to that of temporal variation in the present model, producing an increase rather

a decrease in the phenotypic variance (Appendix S3), an effect also noted in other models (Frank and Slatkin, 1990; Starrfelt and Kokko, 2012). Analysis of a spatially explicit stochastic model beyond numerical or individual-based simulation (e.g. Chevin and Lande, 2011; Scheiner, 2013) and the simplified cases of hard and soft selection is difficult, however, as the evolutionary process in general becomes coupled to the dynamics of local population density and changes in genetic variances generated by dispersal (Tufto, 2000a).

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