When three traits make a line: Evolution of phenotypic plasticity and genetic assimilation through linear reaction norms in stochastic environments

Torbjørn Ergon[1*] & Rolf Ergon[2]

[1]Centre for Ecological and Evolutionary Synthesis, Department of Biosciences, University of Oslo, P.O. Box 1066 Blindern, N-0316 Oslo, Norway

[*]Corresponding author. E-mail: t.h.ergon@ibv.uio.no

[2]Telemark University College, Porsgrunn, Norway

Running title: When three traits make a line

Key words: genetic redundancy, environmental change, value of information, uncertain/imperfect cues, life-history theory, optimality models, cue perception and reliability

Number of words and equations in main text, excluding tables, figure captions and literature cited: 7500

Number of figures: 5

Number of tables: 0

Number of appendices for print: 3

Supporting Information for online publication: 3 files

Data archiving: No data used. The R code used for simulations is provided as Supporting Information S1 for online publication.

Abstract

Genetic assimilation results from selection on phenotypic plasticity, but quantitative genetics models of linear reaction norms considering intercept and slope as traits do not fully incorporate the process of genetic assimilation. We argue that intercept-slope reaction norm models are insufficient representations of genetic effects on linear reaction norms, and that defining the intercept as a trait is unfortunate. Instead we suggest a model with three traits representing genetic effects that respectively (1) are independent of the environment, (2) alter the sensitivity of the phenotype to the environment, and (3) determine how the organism perceives the environment. The model predicts that, given sufficient additive genetic variation in environmental perception, the environmental value at which reaction norms tend to cross will respond rapidly to selection, and eventually become equal to the mean environment. Hence, in this model, genetic assimilation in a new environment becomes complete without changes in genetic correlations, genetic drift or imposing any fitness costs on maintaining plasticity. The asymptotic evolutionary outcome of this three-trait linear reaction norm generally entails a lower degree of phenotypic plasticity than the two-trait model, and maximum expected fitness does not occur at the mean trait values in the population.

Introduction

All natural populations evolve in environments that are to some degree variable. Biologists have long realized that the phenotypic expression of different genotypes may respond differently to the same environmental change, and that such phenotypic plasticity may be heritable (DeWitt and Scheiner 2004, Pigliucci 2005). Depending on the effect this phenotypic plasticity has on selection (fitness), evolution may thus bring about mechanisms that either buffer the phenotypic expression against environmental variation (i.e., environmental canalization) or modify the responses to some environmental influence in an adaptive manner (Nijhout 2003). Phenotypic plasticity involves developmental, physiological and/or behavioral phenotypic responses to some component(s) of the environment (DeWitt and Scheiner 2004, Pigliucci 2005, Pigliucci et al. 2006). These environmental

components, often referred to as environmental 'cues'(DeWitt and Scheiner 2004), are often just correlated with, but not identical to, the environmental variables affecting fitness (e.g. McNamara et al. 2011, Svennungsen et al. 2011, Gienapp et al. 2014). Hence, cues do not provide perfect information about the optimal phenotypic expression, and it is usually adaptive to respond more conservatively towards information-poor cues than more informative ones (Yoccoz et al. 1993, Ergon 2007, McNamara et al. 2011). The phenotypic expression of a particular genotype as a function of environmental cues is called a reaction norm (Woltereck 1909, Pigliucci 2005). There has been considerable interest in evolutionary processes governing reaction norms as this is crucial for our understanding of how populations may respond to environmental change (e.g. Lande 2009, McNamara et al. 2011, Gienapp et al. 2014).

Waddington (1953, 1961) originally used the term 'genetic assimilation' to describe experimental selection results where qualitative phenotypes (such as lack of cross-veins in *Drosophila* wings) that are initially only expressed in response to a particular environmental stimuli (such as heat shock during a particular stage of development) becomes constitutively produced (i.e., becomes expressed independently of the environmental stimuli) after continued selection. However, 'genetic assimilation' is also used to describe similar phenomena in evolution of the mean of quantitative phenotypes that may remain plastic at equilibrium in a stochastic environment after an environmental change (Pigliucci and Murren 2003; Lande 2009). In such cases, the new equilibrium phenotypes will not be independent of the environment unless the reaction norm slope is zero. We adopt Lande's (2009) definition of genetic assimilation in an altered environment as the reduction in the plastic component of the phenotype with a concomitant genetic evolution, while maintaining the phenotype initially produced by plasticity in the altered environment. By 'plastic component' we here mean the difference between the phenotypic value and the mean phenotype in the environment where phenotypic variance is minimized (i.e., where individual reaction norms tend to cross; see Fig. 1 in Pigliucci et al. 2006). We consider this process of genetic assimilation as complete when the expected plastic component of the phenotype is zero and the phenotypic variance is minimized in the new mean environment (but both mean reaction norm slope and phenotypic variance in the mean environment may remain non-zero; see Fig. 1 in Pigliucci and Murren 2003 and Fig. 2 in Pigliucci et al. 2006).

Several experiments, mainly on the fruit fly *Drosephila melanogaster*, have demonstrated genetic assimilation in laboratory populations (Braendle and Flatt 2006), and there is substantial evidence for genetic assimilation, also for quantitative traits, from field observations and experiments (reviewed by Pigliucci and Murren 2003). The most commonly proposed mechanisms for genetic assimilation is that extraordinary environments may expose hidden genetic variation in reaction norms for subsequent selection, followed by eventual canalization of the phenotypic expression in the new environment (Braendle and Flatt 2006, Pigliucci et al. 2006, Lande 2009). The latter stage of this process is perhaps the least understood; it has been suggested that genetic drift or fitness costs of maintaining plasticity plays a part (West-Eberhard 2003, Pigliucci et al. 2006, Lande 2009, Bateson and Gluckman 2011), and changes in the genetic variances, covariances and genetic architecture of reaction norm components may be involved (Wagner et al. 1997, Steppan et al. 2002, Le Rouzic et al. 2013).

One approach to quantitative genetic analysis of phenotypic plasticity (Via et al. 1995, Rice 2004) is to consider the intercept and slope of linear reaction norms as two quantitative traits in their own right (de Jong 1990, Gavrilets and Scheiner 1993a, de Jong and Gavrilets 2000, Tufto 2000, Lande 2009). More generally, reaction norms have been modeled by considering polynomial coefficients as traits (Gavrilets and Scheiner 1993b, Scheiner 1993). In these models, the intercept trait is the phenotypic value at a reference cue designated as zero. Lande (2009) analyzed the evolution of such a linear reaction norm, assuming a stochastic environment undergoing a sudden extreme change (relative to the background fluctuations) in both the mean environmental cue and the phenotypic value where fitness is maximum. In his model the population responded by a rapid increase in mean reaction norm slope (plasticity), followed by a slow increase in reaction norm elevation at the reference cue with a concomitant decrease in plasticity. However, the genetic assimilation was not completed, as the cue value at which the phenotypic variance was at its minimum could never move away from the reference cue because the covariance between reaction norm slope and intercept was assumed to remain constant. Lande (2009) argued that further canalization would take place (e.g., due to fitness costs of maintaining plasticity), but did not include any such mechanisms in his modeling.

In this paper, we argue that the two-trait model is an insufficient representation of genetic effects on linear reaction norms, and hence fails to predict critical aspects of the evolution of phenotypic plasticity and genetic assimilation. Instead we suggest modeling linear reaction norms as being composed of three traits based on fundamental ways that gene products may alter linear reaction norms in such a way that they remain linear. These three traits are (a) effects of gene products that are independent of the cue (variation in this trait will shift the reaction norm along the phenotype axis), (b) effects of gene products that alter the sensitivity of the phenotype to the cue (variation in this trait will alter the slope of the reaction norm), and (c) effects of gene products that affect how the organism perceives the cue (variation in this trait will shift the reaction norm along the cue axis). Reanalyzing the scenarios for extreme environmental change considered by Lande (2009), we show that, under the three-trait reaction norm model, genetic assimilation in the new stochastic environment becomes complete (as defined above) without changes in genetic correlations among the defined traits, genetic drift or imposing any fitness costs on maintaining plasticity. Further, we show that the evolutionary equilibrium of this three-trait linear reaction norm under random mating entails (with certain exceptions) a shallower mean reaction norm slope than the slope of the optimal individual reaction norm and the equilibrium slope of the two-trait model. Hence, maximum fitness does not occur at the mean trait values in the population.

We start by deriving an expression for optimal linear reaction norms as a function of environmental cues in stationary stochastic environments. We then derive our three-trait linear reaction norm model, and finally we analyze the evolutionary dynamics of this model in a quantitative genetics framework, and compare it to the dynamics of the two-trait reaction norm model analyzed by Lande (2009).

Models

Optimal linear reaction norms in temporally variable environments

Models for optimal adaptations in variable environments have traditionally assumed either that individuals have no information about the relevant environmental variables, or that individuals have

exact information about the state of the environment (Yoshimura and Clark 1991, Roff 2002). Whenever the phenotype yielding highest fitness is not known exactly (i.e., the individuals do not have full information about the present and future environment), the long term success of a genotype depends not only on the expectation of fitness, but it is also adaptive to reduce the variance in mean fitness across generations (Yoshimura and Clark 1991, Starrfelt and Kokko 2012). Models that assume that individuals have no information about the environment have been used to explain risk-avoidance and bet-hedging strategies (den Boer 1968, Hopper et al. 2003, Starrfelt and Kokko 2012). On the other side of the spectrum, models that predict optimal trait values as a function of environmental variables, often assume that these variables are known to the individuals without error (e.g. Stearns 1992, Roff 2002).

The concept that phenotypic expressions are functions of more or less informative environmental cues is well established in evolutionary ecology (Tollrian and Harvell 1999, DeWitt and Scheiner 2004, Stephens et al. 2007, McNamara et al. 2011, Gienapp et al. 2014). For example, seasonal reproduction in many organisms must take place within a rather narrow time-window which often varies largely between years (Durant et al. 2007, Gienapp et al. 2014). Since such phenological events must often be prepared a long time in advance (due to acquiring resources, physiological developments and migration), seasonal reproduction may be influenced by rather information-poor cues such as temperature and food constituents weeks before reproductive success is determined (Berger et al. 1981, Korn and Taitt 1987, Lindstrom 1988, Negus and Berger 1998, Nussey et al. 2005). Examples of such obviously adaptive phenotypic plasticity to more or less informative environmental cues are ubiquitous in nature (Pigliucci 2005, Sultan 2010, Landry and Aubin-Horth 2014).

To derive an optimal norm of reaction to an imperfect cue, we may view the cue (U) and the phenotypic expression that maximize fitness (Θ) as having a joint distribution with given means, μ_U and μ_Θ, variances, σ_U^2 and σ_Θ^2, and a correlation, $\rho = \frac{\sigma_{U\Theta}}{\sigma_U \sigma_\Theta}$ (Figure 1). Note that we here define the cue (U) in a general sense as the *environmental component* that affects the phenotype, *not* how this component is perceived by the individuals (as in e.g. Tufto (2000)). Also note that U must not

134 necessarily be interpreted as a proxy for another environmental component that affects fitness (e.g.
135 Miehls et al. 2013), although this may be the case (see caption of Figure 1). Hence, following
136 McNamara et al. (2011) we focus on the information content in the cue (U) about the optimal
137 phenotypic expression (Θ) in the given environment.

138 Under the assumption of no density or frequency dependence, the optimal phenotypic trait
139 values are those that maximize the geometric mean of fitness across generations (Dempster 1955,
140 Caswell 2001). This is equivalent to maximizing the expected logarithm of fitness. Hence, if fitness,
141 W, is a Gaussian function (with constant width and peak values) of the phenotype value, y, such that
142 $\ln\big(W(y)\big)$ is a quadratic function, the optimal *linear* reaction norm as a function of cue values u is

$$y_{opt}(u) = \mu_\Theta + \rho\frac{\sigma_\Theta}{\sigma_U}(u - \mu_U) \qquad (1)$$

143 (Appendix A). Note that, due to the quadratic fitness function $\ln\big(W(y)\big)$, this is the same as the least
144 squares prediction line of Θ as a function of cue values u (Battacharyya and Johnson 1977).

145 This optimal reaction norm under imperfect information (eq. (1)) may be seen as a weighted
146 average of the optimal phenotype under no information (μ_Θ) and the optimal phenotype under perfect
147 information ($\mu_\Theta + \frac{\sigma_\Theta}{\sigma_U}(u - \mu_U)$), with the weight being $|\rho|$ (Figure 1). Given that W is a Gaussian
148 function of y, this linear reaction norm is the optimal reaction norm (i.e., a non-linear reaction norm
149 would not perform better) as long as $\mathrm{E}[\Theta|U = u]$ is a linear function of u, which is the case when U
150 and Θ are bi-normally distributed (chap. 7.8 Johnson and Wichern 2007).

151 Optimality models of this kind have been central in the development of evolutionary ecology
152 (Parker and Maynard Smith 1990, Sutherland 2005, Roff 2010). McNamara et al. (2011) analyzed the
153 general optimal linear reaction norm given by equation (1) in terms of optimal phenology under
154 environmental change. Ergon (2007) used a similar approach to analyze optimal trade-offs between
155 pre-breeding survival, onset of seasonal reproduction and reproductive success in fluctuating
156 multivoltine species.

157 **Quantitative genetics models for linear reaction norms – two vs. three traits**

158 The optimal linear reaction norm given by equation (1) says nothing about the selection process and
159 does not consider genetic constraints. In the following we will consider a quantitative genetic model
160 for linear reaction norms, assuming phenotypic responses to an interval-scaled cue with an arbitrary
161 zero point (Houle et al. 2011).

162 In quantitative genetic models for the evolution of phenotypic plasticity, it is common to
163 model linear reaction norms by two traits, representing the intercept (α) and slope (β) of the reaction
164 norm (e.g. de Jong 1990, Gavrilets and Scheiner 1993a, de Jong and Gavrilets 2000, Tufto 2000,
165 Lande 2009, Scheiner 2013). I.e., the plastic phenotype is modeled as a function of an environmental
166 cue u on the form

$$y(u) = \alpha + \beta u \tag{2}$$

167 In this two-trait model, the intercept trait α is the phenotypic expression for the cue-value designated
168 as zero. Lande (2009) assumed that minimum phenotypic variation occurred in the mean environment
169 that the population had been adapted to, and hence defined the cue to have its zero point in this
170 reference environment. He then used this reaction norm model (eq. (2)) in a quantitative genetics
171 analysis of adaptations to a sudden extreme change in the mean environment when the reference
172 environment remained unchanged.

173 We will here analyze a more general linear reaction norm model based on three fundamental
174 ways that genetic effects can alter a linear reaction norm in such a way that it remains linear; a change
175 along the plastic phenotype axis, a change in slope (cue sensitivity), and a change in the reaction norm
176 along the cue axis. This leads us to consider a linear reaction model on the form

$$y(u) = z_a + z_b(u - z_c) \tag{3}$$

where z_a, z_b and z_c are considered as (latent) traits. A particular genetic effect may of course affect more than one of these traits, but any genetic effect on a linear reaction norm can be decomposed into these three components. Obviously, shifting the reaction norm along the cue-axis (a change in z_c) may have exactly the same effect on the reaction norm as shifting it along the y-axis (a change in z_a). By rearranging the reaction norm model (3) as $y(u) = \alpha + z_b u$ where $\alpha = z_a - z_b z_c$, we see that increasing z_a by one unit has the same effect on $y(u)$ as decreasing z_c by $1/z_b$ units. However, traits z_a and z_c still represent very different genetic effects within the organisms. Trait z_c may be thought of as representing genetic effects on "perception" of the environmental cue in a general sense. For example, variation in z_c may represent genetic effects affecting the sensory apparatus in such a way that different genotypes perceive the same environmental cue as different, but cue perception may not necessarily involve the sensory apparatus or a nervous system (see Discussion). The component of the reaction norm that is independent of the environment is the intercept ($z_a - z_b z_c$), although this component depends on the chosen zero-point of the interval scaled cue. However, trait z_a represents genetic effects that are invariant to which environment that has been designated (by the researcher) to have cue value zero; $z_b z_c$ is the component of the intercept that depends on the chosen zero-point of the cue variable. Variation in trait z_a may thus represent variation in gene products for which both the production of these gene products and their effect on $y(u)$ are independent of the cue. Finally, trait z_b (reaction norm slope) represents variation in gene products that affect the sensitivity of the plastic phenotype $y(u)$ to the cue. With this parameterization of the reaction norm (eq. (3)), z_c may be referred to as a "cue reference trait" although we do not suggest that there is necessarily a "template" of a specific environment that is stored genetically in the organisms; what is essential is the types of genetic variation that is represented by the three traits in the model.

Note that the two-trait model (eq. (2)) is a special case of the more general three-trait model (eq. (3)) where z_c is fixed to zero. Reaction norm slope is considered as a trait in both models (i.e., $\beta = z_b$), but we have used a separate notation in the two models for clarity.

Analysis

Basic properties of the reaction norm models

As already noted, an obvious difference between the two-trait (eq. (2)) and the three-trait (eq. (3)) reaction norm models is that the two-trait model implies a one-to-one correspondence between genotypes and reaction norms, whereas the three-trait model implies that one reaction norm can represent many genotypes. Nevertheless, as we will see below, linear reaction norms in a population will evolve very differently and reach a different equilibrium when we consider the reaction norm to result from three traits rather than two traits.

An essential difference between the two-trait and the three-trait reaction norm models relates to constraints in the evolution of the covariance between reaction norm intercept and slope in the population. To see this, it is elucidating to consider a particular rescaling of this covariance, u_0, defined as the cue value for which phenotypic variance is at a minimum and where the covariance between the plastic phenotypic value $y(u)$ and reaction norm slope is zero. Given a phenotypic covariance between intercept and slope ($P_{\alpha\beta}$) and a variance in reaction norm slope ($P_{\beta\beta}$), this cue value is

$$u_0 = -\frac{P_{\alpha\beta}}{P_{\beta\beta}} \qquad (4)$$

(Appendix B).

From equation (4) we see that, in the two-trait model, where reaction norm intercept (α) and slope (β) are considered as traits, u_0 is independent of the trait means, and directional selection on any of the traits will not affect u_0 unless the selection also changes the variance of the slope or covariance of the traits.

On the other hand, in the three-trait model, the covariance between intercept and slope depends on the mean traits $\bar{z}_b$ and $\bar{z}_c$. Under the assumption of normal traits, u_0 then becomes

$$u_0 = \bar{z}_c + \frac{\bar{z}_b P_{bc} - P_{ab}}{P_{bb}}. \quad (5)$$

where P_{bc}, P_{ab} and P_{bb} are the elements of the phenotypic variance-covariance matrix indicated by the subscripts (Appendix B). Thus, in this quantitative genetic model, u_0 may respond directly to directional selection on both trait z_b (if $P_{bc} \neq 0$) and trait z_c. If trait z_b is independent of trait z_a and z_c (i.e., $P_{bc} = P_{ab} = 0$), u_0 becomes $\bar{z}_c$. Note also that u_0 is independent of P_{ac}.

Lande (2009) defined the cue u ($\varepsilon_{t-\tau}$ in his model) to have its zero-point at u_0 as a "reference environment". Hence, one could define the two-trait model analyzed by Lande (2009) for any arbitrary interval scaled cue variable as $y(u) = \alpha' + \beta(u - u_0)$ where the genetic correlation between the traits α' and β is by necessity zero since u_0 is defined by $cov(y(u_0), \beta) = cov(\alpha', \beta) = 0$ (Appendix B; see also last paragraph on page 1438 in Lande (2009)). This model is structurally similar to our three-trait model except that the "reference environment" in our model is considered as an individual trait, z_c (reflecting individual variation in cue "perception"), which is exposed to selection. Unlike in Lande's (2009) model, where the definition of trait α' depends on u_0, there are no constraints on the phenotypic or genotypic covariances in our three-trait model (other than that the covariance matrix must be positive-definite). The two-trait model of Lande (2009) can only evolve in the same way as the three-trait model if u_0 is treated as the mean of an individual trait with variance different from zero. Hence, the three-trait quantitative genetics model and Lande's (2009) two-trait model are not alternative parameterizations of the same model. Lande's (2009) two-trait model is a constrained (nested) version of our more general three-trait model with the trait z_c fixed to u_0, which requires that $P_{cc} = P_{ac} = P_{bc} = 0$ as well as $P_{ab} = 0$ ($P_{ab} = 0$ is only required to maintain the same definition of z_a and α' and to give $\bar{z}_c = u_0$).

For further analysis, we define the 'plastic component' of the phenotype as the difference between the phenotypic value and the expected phenotype at cue value u_0, $y(u) - E[y(u_0)]$. This definition is not dependent on any particular reaction norm model or genetic architecture of the phenotypic plasticity, and the 'plastic component' can be estimated for any phenotypic observation

when it is possible to estimate $E[y(u_0)]$. The expectation of the plastic component in a random environment becomes $E\left[y(u) - E[y(u_0)]\right] = \bar{\beta}(\mu_U - u_0)$, where the mean reaction norm slope $\bar{\beta}$ is interchangeable with $\bar{z}_b$ in the three-trait model. Note that we obtain the same expected value if we instead define the plastic component as $\beta(u - u_0)$. We will later show that expected u_0 at equilibrium in the three-trait model always becomes μ_U, and hence the expected plastic component at equilibrium will always be zero.

Evolution of linear reaction norms

Environmental change may lead to changes in any of the parameters of the joint distribution of cue (U) and the best possible phenotype (Θ) (c.f., eq. (1) and Figure 1). Any such change will impose directional selection on the individual traits defining the reaction norm, and the evolutionary response to this selection will depend on the additive genetic variances and covariances of these traits. We will here compare the evolution of linear reaction norms based on the three-trait model (eq. (3)) and the more constrained two-trait model (eq. (2)) analyzed in detail by Lande (2009). Specifically, we will analyze the transient and asymptotic evolution of the reaction norm distribution after a sudden and extreme concomitant change in both μ_U and μ_Θ, while σ_U^2, $\sigma_\Theta^2$and $\sigma_{U\Theta}$ remain unchanged. We assume that all individuals in each generation experience the same environment, and that the environments in subsequent generations are independent (as also in Lande's (2009) analysis). Following Lande (2009) we also assume that trait variances and covariances remain constant under selection. Although this may be a particularly unrealistic assumption (Steppan et al. 2002), it serves the purpose of examining how reaction norms can evolve through changes in trait means only.

Quantitative genetics – modeling

Assuming that the individual traits of the reaction norm (3) have a multi-normal distribution with a constant variance-covariance matrix in a population with discrete generations, the fundamental equation describing the change in the population mean of the traits from a generation t to the next,

$$\begin{bmatrix}\bar{z}_a\\ \bar{z}_b\\ \bar{z}_c\end{bmatrix}_{t+1} - \begin{bmatrix}\bar{z}_a\\ \bar{z}_b\\ \bar{z}_c\end{bmatrix}_{t} = \begin{bmatrix}G_{aa} & G_{ab} & G_{ac}\\ G_{ab} & G_{bb} & G_{bc}\\ G_{ac} & G_{bc} & G_{cc}\end{bmatrix}\boldsymbol{\beta}_t \tag{6}$$

is the product of the additive genetic variance-covariance matrix for the traits ($\boldsymbol{G}$) and the selection gradient $\boldsymbol{\beta}_t$ defined as the sensitivity of the logarithm of population mean fitness to changes in each of the mean trait values (Lande 1979, Lande and Arnold 1983),

$$\boldsymbol{\beta}_t = \begin{bmatrix}\partial/\partial\bar{z}_{a,t}\\ \partial/\partial\bar{z}_{b,t}\\ \partial/\partial\bar{z}_{c,t}\end{bmatrix}\ln(\bar{W}_t). \tag{7}$$

We will assume a Gaussian fitness function with width ω and peak value W_{max}, and that all individuals experience the same environment in any generation.

A random individual in generation t has phenotype $y_t(u_t) = z_{a,t} + z_{b,t}(u_t - z_{c,t})$, where the traits $[z_{a,t}, z_{b,t}, z_{c,t}]$ are drawn from a multi-normal distribution with mean $[\bar{z}_a, \bar{z}_b, \bar{z}_c]_t$ and phenotypic covariance matrix $\boldsymbol{P}$. When the phenotypic expression that maximizes fitness in that generation is θ_t, this individual will have fitness

$$W_t = W(y_t(u_t), \theta_t) = W_{max}\exp\left(-\frac{(y_t(u_t)-\theta_t)^2}{2\omega^2}\right). \tag{8}$$

To find an analytical expression of the selection gradient (7), the standard approach (Lande and Arnold 1983, Lande 2009) would be to first find the population mean fitness by integrating over the phenotype distribution, $p(y_t(u_t))$,

$$\bar{W}_t = \int_{-\infty}^{\infty} W(y_t(u_t), \theta_t) p(y_t(u_t)) dy \tag{9}$$

However, because $p(y_t(u_t))$ is not normal as it involves the product of the two normally distributed traits $z_{b,t}$ and $z_{c,t}$, it is not straightforward to solve this integral analytically. Indeed, it seems that an exact analytical expression for the selection gradient (7) does not exist. We therefore initially based our analysis on simulations of the evolutionary process (6) where the selection gradient (7) is computed numerically by simulating a population of 10,000 individuals at each generation (see Supporting Information S1 for R code). These simulations are accompanied by (and compared to) a mathematical analysis presented in Supporting Information S2.

In the simulation results presented in Figure 2, we used the same parameter values as in Lande's (2009) analysis of the two-trait model except that we, for convenience, used a somewhat less extreme sudden change in the environment, with a change in μ_U and μ_Θ of 3 (instead of 5) standard deviations of the background fluctuations (σ_U and σ_Θ of equation (1)). As Lande (2009) we used a diagonal $\boldsymbol{G}$-matrix and sat G_{cc} to half the cue variance (three-trait model) or zero (two-trait model). For simplicity, in the simulations we also assumed that only trait z_a had a non-additive residual component with variance σ_e^2, such that $P_{aa} = G_{aa} + \sigma_e^2$, $P_{bb} = G_{bb}$, $P_{cc} = G_{cc}$, and $P_{ab} = P_{ac} = P_{bc} = 0$. The two-trait model is obtain simply by setting also $P_{cc} = 0$ and $\bar{z}_c = 0$.

Quantitative genetics – results

Selection according to equations (6) to (9) will find equilibrium values of the mean traits that maximize $\mathrm{E}[\ln(\bar{W})]$. Since it is not possible to find a general analytical solution, we present simulation results, in addition to approximate theoretical results and an analytical solution in the case of a constant environment (Supporting Information S2).

The simulations show that immediately after the sudden environmental change, there is a rapid increase in reaction norm slope (Figure 2B), while $\bar{z}_c$ (Figure 2C) swings back in the opposite direction of the change in mean cue μ_U (i.e., away from the new optimum). This phase of the

adaptation may be characterized as a "state of alarm" where it becomes adaptive to exaggerate the perception of the environmental change. As $\bar{z}_a$ moves towards the new optimum (Figure 2A), the reaction norm slope $\bar{z}_b$ is reduced and $\bar{z}_c$ turns towards the new optimum. Eventually, $\bar{z}_c$ stabilizes around μ_U and $\bar{z}_a$ stabilizes around μ_Θ (Figure 2D).

Since we used a diagonal phenotypic variance-covariance matrix (***P***) in the simulations, the cue value u_0 that yields minimum phenotypic variance (eq. (5)) equals $\bar{z}_c$, which stabilizes around μ_U in the simulations (Figure 2C). Hence, equilibrium u_0 appears to be $u_0^* = \mu_U$. As shown both by simulations (Supporting Figures S1-S3) and theoretical considerations (Supporting Information S2), this property of the three-trait model holds also when ***P*** is not diagonal – i.e., at equilibrium, phenotypic variance is *always* minimum in the mean environment. As a result, the three-trait model leads to complete genetic assimilation in the sense that the expected plastic component (defined above) at equilibrium becomes zero, $\bar{z}_b^*(\mu_U - u_0^*) = 0$. In contrast, in the two-trait model, the expected plastic component is zero only when $\mu_U = -P_{\alpha\beta}/P_{\beta\beta}$, and the phenotypic variance can only be minimized in this mean environment (see equation (4)). This contrast in the asymptotic state of the systems obtained from the two alternative reaction norm models is illustrated in Figure 3. Figure 4 shows the trajectories of phenotypic variation and the expected plastic component in the simulated scenario presented in Figure 2. Supporting Figures S4 and S5 show simulation results for a scenario where there is no environmental variation before and after the sudden environmental change (more similar to classic examples of genetic assimilation).

Interestingly, as seen in Figure 2B, the mean reaction norm slope $\bar{z}_b$ in the three-trait model, stabilizes at a lower level than the optimal slope yielding the highest expected fitness of an individual, $\sigma_{U\Theta}/\sigma_U^2$ (see eq. (1)), which is also the equilibrium mean slope in the two-trait model (Gavrilets and Scheiner 1993a, Lande 2009). Intuitively, this is because the optimal value of trait z_b of an individual depends on the value of trait z_c that this individual possesses, which is stochastic. As it is not straightforward to calculate equilibrium mean traits in the three-trait model (see Supplementary Information S2), we first investigated this by calculating the mean trait values that yield maximum expected logarithm of fitness, $E[\ln(W)]$, for a *random* individual in the population (Appendix C).

Under the assumption that $P_{ab} = P_{bc} = 0$, we obtain $\bar{z}_a^{(*)} = \mu_\Theta$, $\bar{z}_b^{(*)} = (\sigma_{U\Theta} + P_{ac})/(\sigma_U^2 + P_{cc})$, and $\bar{z}_c^{(*)} = \mu_U$, which is close to the stationary means in the simulations (Figure 2). For comparison, the equilibrium mean traits in the two-trait model become $\bar{\beta}^* = \sigma_{U\Theta}/\sigma_U^2$ and $\bar{\alpha}^* = \mu_\Theta - \bar{\beta}^* \mu_U$ (Gavrilets and Scheiner 1993a, Lande 2009). Note that the denominator in the expression for $\bar{z}_b^{(*)}$ is the variance of $(U - z_c)$ and not the variance of the cue U alone as in the expression for $\bar{\beta}^*$ in the two-trait model; i.e., genetic variance in the perception trait z_c inflates the variance of the perceived cue $(U - z_c)$. Hence, if $P_{ac} = 0$, $\bar{z}_b^{(*)}$ is always lower than the optimal slope in equation (1) unless $P_{cc} = 0$ (which gives the two-trait reaction norm model). This is indicated by a stippled reaction norm in Figure 1. Note again, however, that selection will maximize $\mathrm{E}[\ln(\bar{W})]$ in the population and not $\mathrm{E}[\ln(W)]$ for a random individual. Nevertheless, as shown in Figure 2 and Supporting Information S2, maximizing $\mathrm{E}[\ln(\bar{W})]$ when $P_{ab} = P_{bc} = 0$ also leads to $\bar{z}_a^* = \mu_\Theta$ and $\bar{z}_c^* = \mu_U$ (i.e., the same values that maximize $\mathrm{E}[\ln(W)]$), but the equilibrium mean slope $\bar{z}_b^*$ equals $\bar{z}_b^{(*)}$ above only when $\sigma_U^2 = \sigma_\Theta^2 = \sigma_{U\Theta} = 0$ (i.e. only in a constant environment). Note that $\bar{z}_a^*$ and $\bar{z}_c^*$ are independent of the variances and covariance of U and Θ when $P_{ab} = P_{bc} = 0$. In Supporting Information S2 we conjecture that the equilibrium mean traits $\bar{z}_a^*$ and $\bar{z}_c^*$ are affected by σ_U^2, σ_Θ^2 and $\sigma_{U\Theta}$ only indirectly through $\bar{z}_b^*$ (but when $P_{ab} = P_{bc} = 0$, $\bar{z}_a^*$ and $\bar{z}_c^*$ are independent of z_b^*, and hence also of σ_U^2, σ_Θ^2, and $\sigma_{U\Theta}$).

As seen in Figure 2B the asymptotic mean $\bar{z}_b$ in the simulations (where $P_{ac} = 0$) is close to but somewhat larger than $\bar{z}_b^{(*)} = \sigma_{U\Theta}/(\sigma_U^2 + P_{cc})$. This discrepancy is to be expected for two reasons. First, as pointed out above, the mean trait $\bar{z}_b^{(*)}$ that maximizes $\mathrm{E}[\ln(W)]$ for a random individual is not identical to the equilibrium mean trait where the selection gradient (7) is zero, i.e. where $\mathrm{E}[\ln(\bar{W})]$ is maximized (Supporting Information S2). As shown in Supporting Information S2, the equilibrium mean reaction norm slope $\bar{z}_b^*$ can be approximated analytically if we assume that the plastic phenotype $y(u)$ has a normal distribution, which is very nearly the case with the parameter values in our simulations in Figure 2. The integral (9) then has an analytical solution, and as a result an approximate equilibrium slope $\bar{z}_b^*$ can be found numerically from the equation (assuming $P_{ab} = P_{bc} = 0$)

$$\bar{z}_b^* \approx \frac{\sigma_{U\Theta} + P_{ac}}{\sigma_U^2 + P_{cc}} + \frac{\left(\sigma_\Theta^2 + \bar{z}_b^{*2}\sigma_U^2 - 2\bar{z}_b^*\sigma_{U\Theta} + P_{bb}\sigma_U^2\right)(-P_{ac} + \bar{z}_b^* P_{cc})}{(\omega^2 + P_{aa} - 2\bar{z}_b^* P_{ac} + P_{bb}P_{cc} + \bar{z}_b^{*2}P_{cc})(\sigma_U^2 + P_{cc})} \quad (10)$$

where the large values of σ_Θ^2 and especially ω^2 used in the simulations make the second term positive but small compared to the first term which is equal to $\bar{z}_b^{(*)}$ above. Note that the first and dominant term in equation (10) is found by maximization of $\mathrm{E}[\ln(W)]$, without the assumption of a normal plastic phenotype (Appendix C).

The second reason for the discrepancy between the asymptotic mean $\bar{z}_b$ in the simulations and $\bar{z}_b^{(*)}$ is that when the population under directional selection based on equation (6) evolves towards a stationary state, the mean traits will fluctuate around the equilibrium because of the influence from the random inputs u_t and θ_t (as seen in Figure 2). In stationarity this leads to $\bar{z}_a = E[\bar{z}_a] + v_a$ etc. (where $E[\bar{z}_a] = \lim_{N\to\infty} \frac{1}{N}\sum_{t=1}^{N} \bar{z}_{a,t}$ and $E[v_a] = 0$ etc.), and, as shown in Supporting Information S2, the variances and covariances of v_a, v_b and v_c then enter into equation (10). Note that we assume that u_t and θ_t have zero autocorrelation, such that the covariances between the mean reaction norm parameters and the environment caused by adaptive tracking (Tufto 2015) can be neglected.

Because the reaction norm slope $\bar{z}_b^*$ is influenced by the phenotypic variance of the cue reference trait z_c (and its covariance with the other traits; eq. (10)), and hence deviates from the slope that maximizes fitness (eq. (1)), the expected fitness at equilibrium will be lower than the expected fitness of the optimal individual reaction norm in equation (1) (Figure 5, lower right panel). As a consequence a proportion of the population will have a higher expected fitness than an individual with mean trait values. Nevertheless, mean fitness in the population after the environmental change stabilizes around a higher level in the three-trait model than in the two-trait model (Figure 5, left panels), despite a lower expected fitness at mean trait values (right panels). The reason for this is that the three-trait model gives a lower phenotypic variance in the new environment (Figure 4A). Mean fitness in the two-trait model thus stabilizes around the optimum *only* when the mean cue is zero because phenotypic variance will not be minimized in other environments (Figure 5, left panels).

Discussion

Quantitative genetics models are theoretical models for the joint evolution of population means of quantitative individual phenotypic traits, where the researchers define traits that they find most meaningful in the context they are studied. In quantitative genetics models of reaction norms where plastic phenotypes are modeled as a linear function of an interval scaled environmental cue, the reaction norm intercept and slope are often considered as individual traits subjected to selection (Gavrilets and Scheiner 1993b, Scheiner 1993, de Jong and Gavrilets 2000, Tufto 2000, Lande 2009, Scheiner 2013, Tufto 2015). The intercept of such a reaction norm (i.e., the reaction norm value at cue value zero) is often not very biologically meaningful since this trait, as well as its variance and covariance with other traits, depend on the defined zero-point, or "reference cue", of the (arbitrary) interval scaled cue variable. One may, however, as in Lande (2009), define the zero-point of the cue to be the mean cue value for which the population is adapted to. This ensures that the variance of the plastic phenotype is minimized in the mean environment, which is theoretically plausible (Bürger 2000, Lande 2009, Le Rouzic et al. 2013), but it is not clear how this "reference cue" may evolve (in Lande's (2009) analysis it is assumed to remain constant; see however de Jong and Gavrilets 2000).

We have here suggested that the "reference cue" can be considered as an individual trait that reflects genetic variation in cue "perception" in a general sense, and hence considered a linear reaction norm on the form $y(u) = z_a + z_b(u - z_c)$. In this model, the biological meaning of all the traits, and their variances and covariances, is not modified when redefining the zero-point of the cue variable u (which is not the case for the intercept $\alpha = z_a + z_b z_c$, $var(\alpha)$ and $cov(\alpha, z_b)$). The three traits in this model reflect three fundamentally different genetic effects on linear reaction norms. While z_b represents genetic effects on cue sensitivity, z_c reflects genetic effects on cue "perception" (in the general sense discussed below) and has the same scale as the environmental cue, and z_a represents genetic effects that are both independent of the cue value and are invariant to its defined zero-point (the latter is not the case for the intercept). These structural differences in the reaction norm models matter for the equilibrium mean reaction norms (and distributions) because the traits do not have independent effects on the plastic phenotype ($y(u)$) (note the product $z_b z_c$ in the three-trait model).

In our analysis, we have shown that the cue value where variance of the plastic phenotype is minimized (where reaction norms "tend to cross"; u_0) always evolves to equal the mean environment at equilibrium. This means that the expected plastic component of the phenotype defined as the difference between the value of the plastic phenotype and the phenotype at u_0 always becomes zero at equilibrium in a stationary stochastic environment (i.e., $E[y(u) - y(u_0)] = 0$ at equilibrium). Hence, genetic assimilation, as defined in the Introduction, will always be completed at equilibrium in our three-trait model regardless of the mean environment, and without assuming any cost of maintaining plasticity (DeWitt et al. 1998, West-Eberhard 2003, Pigliucci et al. 2006, Lande 2009, Bateson and Gluckman 2011, Svennungsen et al. 2011) or any change in the variances or covariances of our defined traits (de Jong and Gavrilets 2000). Even though u_0 may be interpreted as '$-cov(\text{intercept}, \text{slope})/var(\text{slope})$' we find u_0 biologically more meaningful than the covariance between reaction norm slope and a somewhat arbitrarily defined intercept trait. Note that u_0 is a population level parameter that does not depend on any quantitative genetic model for the linear reaction norm, and which can easily be estimated (as discussed below). Further, our analysis also demonstrate that the equilibrium mean reaction norm slope in the three-trait model will deviate from the optimal slope yielding the highest expected fitness of a hypothetical individual that can tune reaction norm intercept and slope accurately and independently (eq. (1)), which is also the equilibrium mean slope of the two-trait model (Gavrilets and Scheiner 1993a, Lande 2009). At least when there is weak correlation between z_a and z_c (i.e., P_{ac} is sufficiently small), the mean slope should be lower than the optimal individual slope. Intuitively, this is because the optimal slope is lower when the cue reference trait of a random individual, in addition to the environmental cue, is stochastic (see Appendix C). As a consequence, maximum expected fitness does not occur at the mean trait values in the population.

In the three-trait model, phenotypic variance in a given environment increases with both $\bar{z}_b$ and the distance between $\bar{z}_c$ and the environmental cue (u), at least when the traits are independent (see equation S2-3 in Supporting Information S2), whereas in the two-trait model, phenotypic variance is independent of the trait means. In our simulations, after the sudden environmental change, there is a

436 rapid initial increase in both $\bar{z}_b$ and the distance between $\bar{z}_c$ and the new mean cue value (i.e., $\bar{z}_c$
437 initially evolves rapidly in the *opposite* direction of the change in the environmental cue, such that the
438 perception of the environmental change is exaggerated). Hence, due to the positively interacting
439 (epistatic) effects of $\bar{z}_b$ and $\bar{z}_c$ on the plastic phenotype $y(u)$, this efficiently increases phenotypic
440 variance in the new environment which enhances the evolvability of the plastic phenotypic character
441 and acts to restore population mean fitness (see Figure 2 and Figure 5). The subsequent process of
442 assimilation whereby reaction norm slope $\bar{z}_b$ is reduced, $\bar{z}_c$ moves towards the mean cue value, and $\bar{z}_a$
443 evolves towards mean Θ, is a much slower process.

444 **Genetic effects on linear reaction norms**

445 Although a shift in the reaction norm along the cue-axis (through trait z_c) can have exactly the same
446 effect on the individual reaction norm as a shift along the phenotype-axis (through trait z_a), the genetic
447 bases for these effects are fundamentally different, and, as explained above, changes in the means of
448 these two traits have different effects on the population. It also seems obvious that there will often be
449 genetic variation on both these traits.

450 Phenotypic plasticity involves complex pathways, at both organismal and cell levels, from
451 perception of environmental cues and physiological transduction to phenotypic expression (reviewed
452 in Sultan and Stearns 2005). Depending on the type of organism and the nature of the phenotypic
453 characters and the environmental cues, these pathways may, to varying degrees, involve sensory
454 systems, neuroendocrine and metabolic systems, cellular reception, gene regulation networks, and
455 other developmental, physiological and behavioral processes. Environmental conditions may directly
456 affect any of these systems and processes, not just the sensory systems (e.g., temperature may directly
457 affect metabolism and gene regulation in ectothermic organisms (Gillooly et al. 2002, Ellers et al.
458 2008), and various processes may be affected by food constituents (Sanders et al. 1981, Meek et al.
459 1995, Krol et al. 2012) and nutritional state (Lõmus and Sundström 2004, Rui 2013, Mueller et al.
460 2015)). Genetic variation in upstream (i.e., close to the cue perception) regulatory processes, which
461 may involve cue activation thresholds for transduction elements, may affect the way the environment
462 is "perceived" (in a general sense) by the organism, and hence the cue reference trait (trait z_c) in our

model. Genetic variation in downstream processes close to the phenotypic expression of quantitative characters, on the other hand, may affect the degree of up/down regulation in response to given levels (and types) of transduction elements and hence the slope of linear reaction norms (trait z_b in our model). Finally, some genetic variation may have the same additive effect on the phenotype irrespective of the environmental cue (trait z_a in our model). The importance of differentiating between these three traits may be better appreciated when considering the effects of the mean traits on the population; A change in $\bar{z}_c$ will change the cue value at which different genotypic reaction norms tend to cross (u_0), whereas a change $\bar{z}_a$ will not.

While there is ample evidence for widespread genetic variation for reaction norms in natural populations (Falconer and Mackay 1996, Sultan and Stearns 2005, Sengupta et al. 2015), there are not many examples where the full pathway of phenotypic plasticity from cue perception to phenotype expression is known in great detail (Sultan 2010, Morris and Rogers 2014), and even less is known about the genetic variation of the different elements of these pathways. It seems, however, obvious that there may be substantial genotypic variation in perception of (and not just responses to) environmental cues (i.e., variation in trait z_c in our model). Examples indicating genetic variation in environmental perception include, among other examples, substantial among-population variation in the signal transduction pathway of induced plant defense in *Arabidopsis thaliana* (Kliebenstein et al. 2002), and individual variation in systemic stress responses has likely components of individual variation in what is perceived as stressful (Hoffmann and Parsons 1991, Badyaev 2005, Dingemanse et al. 2010). There is also considerable variation and "fine tuning" in light (and shading) perception systems involving phytochromes that are sensitive to different wave lengths in plants (Smith 1990, 1995, Schlichting and Smith 2002).

Predictions and empirical evaluations

Parameters in a reaction norm function considered as quantitative traits are always latent in the sense that one cannot measure their phenotypic value by a single measurement of an individual (except for traits that are defined for a particular environment, such as an intercept). While one may estimate reaction norm intercept and slope from multiple measurement of the same genotype or related

individuals with known genealogy (Nussey et al. 2007; Martin et al. 2011), such data alone does not provide enough information to separate the traits z_a and z_c (from a statistical point of view, the three-trait model fitted to such data is over-parameterized, which may be one of the reasons it has not previously been considered; note however that the three-trait model predicts a different phenotypic distribution than the two-trait model due to the product $z_b z_c$). Nevertheless, if one have a detailed understanding of the physiological (or developmental) mechanisms of the plastic response one may still be able to estimate meaningful reaction norm traits beyond a phenomenological 'intercept' and 'slope', including traits associated with cue perception (trait z_c) in the sense discussed above. Time-series data from selection experiments may also provide information about the genetic architecture of the reaction norms (Fuller et al. 2005).

The cue value that gives minimum phenotypic variation in the population (u_0), may be estimated by fitting data on genotype specific phenotypic measurements to mixed-effects linear models with random individual slopes and intercepts (Martin et al. 2011, Bates et al. 2014), or from a random regression "animal model" building on a known relatedness among individuals (Nussey et al. 2007). Our three-trait quantitative genetics model gives certain predictions about the evolution of u_0 under environmental change. Our analysis shows that the mean cue reference trait ($\bar{z}_c$), and hence u_0 (eq. (5)), will respond rapidly to changes in the mean environment (provided sufficient additive genetic variation). Whenever there is selection for increased plasticity (i.e., selection for higher $|\bar{z}_b|$), it also becomes adaptive to exaggerate the perception of the environmental change, and u_0 will swing away in the *opposite* direction of the change in the mean cue during a "phase of alarm" (see Figure 2). Later, u_0 will move towards, and eventually fluctuate around, the new cue value. In contrast, under the two-trait model u_0 will not change in response to changes in the mean cue values.

Future directions

In this paper we have made a number of simplistic, but quite standard, assumptions, including interval scaled cues and phenotypes, Gaussian fitness with constant width and peak, lack of density and frequency dependence, random mating, discrete generations where all individuals are exposed to the same environment (e.g. no spatial heterogeneity), and uncorrelated environments from one generation

to the next. These assumptions may be modified or relaxed in future developments. In particular, the two-trait model has been used in theoretical studies involving within-generation heterogeneity (de Jong and Gavrilets 2000, Tufto 2000, Scheiner 2013, Tufto 2015). We suggest that these studies may be developed by including a cue reference trait in the linear reaction norms (our three-trait model). The models may also be modified by incorporating different reaction norm shapes. Notably, de Jong and Gavrilets (2000) allowed the genetic covariance between reaction norm intercept and slope, as well as their variances, to evolve through selection on allelic pleiotropy. It would be interesting to repeat their approach on our three-trait model to investigate the relative contributions (and synergies) of the evolution of trait means and trait variances and covariances.

Several authors have assumed flexible polynomial reaction norms with the polynomial coefficients considered as traits (Gavrilets and Scheiner 1993a, b, Scheiner 1993, Via et al. 1995). We suggest that such rather phenomenological reaction norm models may be modified by basing the polynomial expressions on how the environmental cues are perceived by the individuals ($u - z_c$) rather than on the environmental cue variable itself (u), although the developmental or behavioral mechanistic basis for higher order terms may not be clear.

Regardless of the reaction norm shape, we argue that it is essential to distinguish between genetic variation in how the environmental cues are perceived from other genetic variation affecting the reaction norm distribution in the population. We suggest that future developmental and behavioral studies pay more attention to genetic variation in environment perception and transduction, and that the contributions of such genetic variation to phenotypic variation in natural environments are evaluated.

Acknowledgements

We thank Arnaud Le Rouzic, Samuel M. Scheiner, Thomas F. Hansen and Øistein H. Holen for comments on an earlier version of the manuscript; we greatly appreciate their input and advice but may still disagree on some issues. This work made use of the Abel computing cluster, owned by the University of Oslo and the Norwegian meta-centre for High Performance Computing (NOTUR). We

543 are thankful for technical support from the Research Computing Services group at USIT, University of

544 Oslo.

Appendix A: Optimal reaction norms

The aim is here to find the optimal reaction norm that maximizes $\mathrm{E}[\ln(W)]$, irrespective of any genetic model. We assume that fitness W for a given phenotype y is Gaussian with constant width ω and peak value W_{max}, such that

$$\ln(W) = \ln(W_{max}) - \frac{1}{2\omega^2}(y(U) - \Theta)^2$$

where U is the environmental cue and Θ is the best possible phenotype under perfect information. We thus maximize $\mathrm{E}[\ln(W)]$ by minimizing the criterion function $J = E[(y(U) - \Theta)^2]$, where both U and Θ are random variables with a joint distribution (not necessarily normal).

A linear reaction norm, may be described as $y(U) = \alpha + \beta U$. To find the intercept (α) and slope (β) that minimize J, we first develop J and then solve $\left\{\frac{\partial J}{\partial \alpha} = 0, \frac{\partial J}{\partial \beta} = 0\right\}$ for α and β. Using $E[y(U)^2] = var(y(U)) + E[y(U)]^2$, etc., we get

$$J = E[(\mathrm{y}(U) - \Theta)^2]$$
$$= var(y(U)) + E[y(U)]^2 - 2(cov(y(U), \Theta) + E[y(U)]E[\Theta]) + var(\Theta) + E[\Theta]^2$$

Substituting $var\big(y(U)\big) = \beta^2\sigma_U^2$ and $cov(y(U), \Theta) = \beta\sigma_{U\Theta}$, and further solving $\frac{\partial J}{\partial \alpha} = 0$ and $\frac{\partial J}{\partial \beta} = 0$ for α and β, we find the optimal intercept and slope as $\alpha^* = \mu_\Theta - \frac{\sigma_{U\Theta}}{\sigma_U^2}\mu_U$ and $\beta^* = \frac{\sigma_{U\Theta}}{\sigma_U^2}$ which correspond to equation (1) in the main text.

Appendix B: Cue value u_0 where phenotypic variance is minimum and covariance between reaction norm slope and phenotype is zero

Assume a population of linear reaction norms on the form $y(u) = \alpha + \beta u$, where α and β are traits with phenotypic variances $P_{\alpha\alpha}$, $P_{\beta\beta}$ and covariance $P_{\alpha\beta}$. The variance in the plastic phenotype at a given cue value, u, is then

$$var\big(y(u)\big) = P_{\alpha\alpha} + u^2 P_{\beta\beta} + 2u\, P_{\alpha\beta}$$

Minimization by setting $\frac{\delta}{\delta u} var\big(y(u)\big) = 2u\, P_{\beta\beta} + 2\, P_{\alpha\beta} = 0$ gives the result

$$u_0 = -\frac{P_{\alpha\beta}}{P_{\beta\beta}}$$

We find the same from

$$cov(y(u), \beta) = \mathrm{E}\big[(y - \bar{y})\big(\beta - \bar{\beta}\big)\big] = E\big[\big(\alpha + \beta u - \bar{\alpha} - \bar{\beta} u\big)\big(\beta - \bar{\beta}\big)\big] = \; P_{\alpha\beta} + u\, P_{\beta\beta} = 0$$

In the three-trait genetic model (3), on the other hand, the reaction norm intercept and slope are $\alpha = z_a - z_b z_c$ and $\beta = z_b$, respectively. Since $\mathrm{E}[z_a - z_b z_c] = \bar{z}_a - \mathrm{E}[(z_b - \bar{z}_b + \bar{z}_b)(z_c - \bar{z}_c + \bar{z}_c)] = \bar{z}_a - P_{bc} - \bar{z}_b \bar{z}_c$, and since normal distributions give $\mathrm{E}[(z_b - \bar{z}_b)^2 (z_c - \bar{z}_c)] = 0$ (Isserlis' (1918) theorem), we then have

$$P_{\alpha\beta} = \mathrm{E}[(z_a - z_b z_c - \bar{z}_a + P_{bc} + \bar{z}_b\bar{z}_c)(z_b - \bar{z}_b)]$$

$$= P_{ab} - \mathrm{E}[(z_b - \bar{z}_b + \bar{z}_b)(z_c - \bar{z}_c + \bar{z}_c)(z_b - \bar{z}_b)] = P_{ab} - \bar{z}_c P_{bb} - \bar{z}_b P_{bc}$$

Since $\beta = z_b$ and thus $P_{\beta\beta} = P_{bb}$, the result above applied to the three-trait model thus gives

$$u_0 = \bar{z}_c + \frac{\bar{z}_b P_{bc} - P_{ab}}{P_{bb}}$$

Appendix C: Mean traits that maximize $\mathrm{E}[\ln(W)]$ for a random individual

We here apply the same procedure as in Appendix A to find the mean trait values in the population that maximize $\mathrm{E}[\ln(W)]$ for a random individual for which the traits are stochastic quantities. Starting with our linear three-trait model, $y = z_a + z_b(U - z_c)$ where the traits z_a, z_b and z_c are normally distributed with phenotypic covariance matrix $\boldsymbol{P}$, we develop the criterion function J by using $z_a = z_a - \bar{z}_a + \bar{z}_a$ etc., and $\mathrm{E}[(z_a - \bar{z}_a)(z_a - \bar{z}_a)] = P_{aa}$ etc. According to Isserlis' (1918) theorem (given normal traits) we have $\mathrm{E}[(z_a - \bar{z}_a)(z_b - \bar{z}_b)(z_c - \bar{z}_c)] = 0$, $\mathrm{E}[(z_b - \bar{z}_b)^2(z_c - \bar{z}_c)] = 0$ and $\mathrm{E}[(z_b - \bar{z}_b)(z_c - \bar{z}_c)^2] = 0$. Further assuming that the traits are uncorrelated with both Θ and U we find

$$J = \mathrm{E}[(z_a + z_b(U - z_c) - \Theta)^2]$$

$$= \mathrm{E}\left[\begin{pmatrix} z_a - \bar{z}_a + \bar{z}_a + (z_b - \bar{z}_b)(U - \mu_U) + (z_b - \bar{z}_b)(\mu_U - \bar{z}_c) - (z_b - \bar{z}_b)(z_c - \bar{z}_c) \\ +\bar{z}_b(U - \mu_U) + \bar{z}_b(\mu_U - \bar{z}_c) - \bar{z}_b(z_c - \bar{z}_c) - (\Theta - \mu_\Theta) - \mu_\Theta \end{pmatrix}^2\right]$$

$$= P_{aa} + 2P_{ab}(\mu_U - \bar{z}_c) - 2\bar{z}_b P_{ac} + {\bar{z}_a}^2 - 2\bar{z}_a P_{bc} + 2\bar{z}_a\bar{z}_b(\mu_U - \bar{z}_c) - 2\bar{z}_a\mu_\Theta + P_{bb}\sigma_U^2$$

$$+ P_{bb}(\mu_U - \bar{z}_c)^2 - 2P_{bc}\bar{z}_b(\mu_U - \bar{z}_c) + P_{bb}P_{cc} - 2P_{bc}\bar{z}_b(\mu_U - \bar{z}_c) + 2P_{bc}\mu_\Theta$$

$$+ {\bar{z}_b}^2\sigma_U^2 - 2\bar{z}_b\sigma_{U\Theta} + {\bar{z}_b}^2(\mu_U - \bar{z}_c)^2 - 2\bar{z}_b(\mu_U - \bar{z}_c)\mu_\Theta + {\bar{z}_b}^2 P_{cc} + \sigma_\Theta^2 + \mu_\Theta^2$$

In order to find the mean traits that maximize $\mathrm{E}[\ln(W)]$, we now minimize J by finding the solution to the equations

$$\frac{\partial J}{\partial \bar{z}_a} = 2\bar{z}_a - 2\mu_\Theta - 2P_{bc} + 2\bar{z}_b(\mu_U - \bar{z}_c) = 0$$

$$\frac{\partial J}{\partial \bar{z}_b} = 2(\bar{z}_a - \mu_\Theta - 2P_{bc})(\mu_U - \bar{z}_c) + 2\bar{z}_b(\sigma_U^2 + P_{cc}) + 2\bar{z}_b(\mu_U - \bar{z}_c)^2 - 2\sigma_{U\Theta} - 2P_{ac} = 0$$

$$\frac{\partial J}{\partial \bar{z}_c} = -2(\bar{z}_a - \mu_\Theta - 2P_{bc})\bar{z}_b - 2(P_{bb} + \bar{z}_b{}^2)(\mu_U - \bar{z}_c) - 2P_{ab} = 0$$

With $P_{bb} > 0$ the solution is

$$\bar{z}_a{}^{(*)} = \mu_\Theta + P_{bc} - \bar{z}_b \frac{\bar{z}_b P_{bc} - P_{ab}}{P_{bb}}$$

$$\bar{z}_b{}^{(*)} = \frac{P_{bb}(\sigma_{U\Theta} + P_{ac}) - P_{ab}P_{bc}}{P_{bb}(\sigma_U^2 + P_{cc}) - P_{bc}^2}$$

$$\bar{z}_c{}^{(*)} = \mu_U - \frac{\bar{z}_b P_{bc} - P_{ab}}{P_{bb}}$$

With $P_{bb} = P_{ab} = P_{bc} = 0$ and hence constant z_b (i.e., "Baldwin effect" sensu Lande (2009)) we can only show that $\bar{z}_a{}^{(*)} + \bar{z}_b{}^{(*)}\left(\mu_U - \bar{z}_c{}^{(*)}\right) = \mu_\Theta$, i.e., that the point $[\bar{z}_c{}^{(*)}, \bar{z}_a{}^{(*)}]$ is located on the straight line with slope $\bar{z}_b{}^{(*)} = (\sigma_{U\Theta} + P_{ac})/(\sigma_U^2 + P_{cc})$ going through the point $[\mu_U, \mu_\Theta]$. The point $[\bar{z}_c{}^{(*)}, \bar{z}_a{}^{(*)}]$ is thus forced towards the solution above only when $P_{bb} > 0$.

The traditional two-trait model is a special case of the three-trait model where $\bar{z}_c = 0$, $\bar{z}_a = \alpha$, $\bar{z}_b = \beta$, $P_{cc} = 0$, $P_{ac} = 0$ and $P_{bc} = 0$. Solving $\frac{\partial J}{\partial \alpha} = 0$ and $\frac{\partial J}{\partial \beta} = 0$ gives the solution $\alpha^{(*)} = \mu_\Theta - \frac{\sigma_{U\Theta}}{\sigma_U^2}\mu_U$ and $\beta^{(*)} = \frac{\sigma_{U\Theta}}{\sigma_U^2}$, which is identical to the optimal intercept and slope derived in Appendix A, as well as the asymptotic trait means found by Lande (2009).

Literature cited

Badyaev, A. V. 2005. Role of stress in evolution: from individual adaptability to evolutionary adaptation. Pages 277 - 294 *in* B. Hallgrímsson and B. K. Hall, editors. Variation; a central concept in biology. Elsevier Academic Press.

Bates, D., M. Maechler, B. M. Bolker, and S. Walker. 2014. lme4: Linear mixed-effects models using Eigen and S4. ArXiv e-print; submitted to Journal of Statistical Software.

Bateson, P. P. G., and P. D. Gluckman. 2011. Plasticity, robustness, development and evolution. Cambridge University Press, Cambridge.

Battacharyya, G. K., and R. A. Johnson. 1977. Statistical concepts and methods. John Wiley & Sons.

Berger, P. J., N. C. Negus, E. H. Sanders, and P. D. Gardner. 1981. Chemical triggering of reproduction in *Microtus montanus*. Science **214**:69-70.

Braendle, C., and T. Flatt. 2006. A role for genetic accommodation in evolution? Bioessays **28**:868-873.

Bürger, R. 2000. The Mathematical Theory of Selection, Recombination, and Mutation. Wiley, New York.

Caswell, H. 2001. Matrix Population Models; Construction, analysis and interpretation. Sinauer Associates, Sunderland, Massachusetts.

de Jong, G. 1990. Quantitative Genetics of reaction norms. Journal of Evolutionary Biology **3**:447-468.

de Jong, G., and S. Gavrilets. 2000. Maintenance of genetic variation in phenotypic plasticity: the role of environmental variation. Genet. Res. **76**:295-304.

Dempster, E. R. 1955. Maintenance of genetic heterogeneity. Cold Spring Harbor Symposia on Quantitative Biology **20**:25-32.

629 den Boer, P. 1968. Spreading of risk and stabilization of animal numbers. Acta Biotheoretica
630 **18**:165-194.
631 DeWitt, T. J., and S. M. Scheiner, editors. 2004. Phenotypic plasticity; Functional and
632 Conceptual Approaches. Oxford University Press, New York.
633 DeWitt, T. J., A. Sih, and D. Sloan Wilson. 1998. Costs and limits of phenotypic plasticity.
634 Trends in Ecology & Evolution **13**:77- 81.
635 Dingemanse, N. J., A. J. Kazem, D. Reale, and J. Wright. 2010. Behavioural reaction norms:
636 animal personality meets individual plasticity. Trends Ecol Evol **25**:81-89.
637 Durant, J. M., D. O. Hjermann, G. Ottersen, and N. C. Stenseth. 2007. Climate and the match
638 or mismatch between predator requirements and resource availability. Climate
639 Research **33**:271-283.
640 Ellers, J., J. Marien, G. Driessen, and N. M. van Straalen. 2008. Temperature-induced gene
641 expression associated with different thermal reaction norms for growth rate. Journal of
642 Experimental Zoology **310**:137-147.
643 Ergon, T. 2007. Optimal onset of seasonal reproduction in stochastic environments: When
644 should overwintering small rodents start breeding? Ecoscience **14**:330-346.
645 Falconer, D. S., and T. F. C. Mackay. 1996. Introduction to quantitative genetics. Fourth
646 edition. Longman, Essex.
647 Fuller, R. C., C. F. Baer, and J. Travis. 2005. How and when selection experiments might be
648 useful. Integrative and comparative biology 45:391-404.
649 Gavrilets, S., and S. M. Scheiner. 1993a. The genetics of phenotypic plasticity. V. Evolution
650 of reaction norm shape. Journal of Evolutionary Biology **6**:31-48.
651 Gavrilets, S., and S. M. Scheiner. 1993b. The genetics of phenotypic plasticity. VI.
652 Theoretical predictions for directional selection. Journal of Evolutionary Biology
653 **6**:49-68.

654 Gienapp, P., T. E. Reed, and M. E. Visser. 2014. Why climate change will invariably alter
655 selection pressures on phenology. Proceedings of the Royal Society B-Biological
656 Sciences **281**:8.
657 Gillooly, J. F., E. L. Charnov, G. B. West, V. M. Savange, and J. H. Brown. 2002. Effects of
658 size and temperature on developmental time. Nature **417**:70-73.
659 Hoffmann, A. A., and P. A. Parsons. 1991. Evolutionary genetics and environmental stress.
660 Oxford University Press, New York.
661 Hopper, K. R., J. A. Rosenheim, T. Prout, and S. J. Oppenheim. 2003. Within-generation bet
662 hedging: a seductive explanation? Oikos **101**:219-222.
663 Houle, D., C. Pélabon, G. P. Wagner, and T. F. Hansen. 2011. Measurement and meaning in
664 biology. The quarterly review of biology **86**:3-34.
665 Isserlis. 1918. On a formula for the product-moment coefficient of any order of a normal
666 frequency distribution in any number of variables. Biometrika **12**:134–139.
667 Johnson, R. A., and D. W. Wichern. 2007. Applied multivariate statistical analysis. sith
668 edition. Pearson Prentice Hall.
669 Kliebenstein, D. J., A. Figuth, and T. Mitchell-Olds. 2002. Genetic Architecture of Plastic
670 Methyl Jasmonate Responses in Arabidopsis thaliana. Genetics **161**:1685-1696.
671 Korn, H., and M. J. Taitt. 1987. Initiation of early breeding in a population of *Microtus*
672 *townsendii* (Rodentia) with the secondary plan compound 6-MBOA. Oecologia
673 (Berlin) **71**:593-596.
674 Krol, E., A. Douglas, H. Dardente, M. J. Birnie, V. van der Vinne, W. G. Eijer, M. P.
675 Gerkema, D. G. Hazlerigg, and R. A. Hut. 2012. Strong pituitary and hypothalamic
676 responses to photoperiod but not to 6-methoxy-2-benzoxazolinone in female common
677 voles (Microtus arvalis). General and Comparative Endocrinology **179**:289-295.

678 Lande, R. 1979. Quantitative genetic analysis of multivariate evolution, applied to brain:body
679 size allometry. Evolution **33**:402-416.
680 Lande, R. 2009. Adaptation to an extraordinary environment by evolution of phenotypic
681 plasticity and genetic assimilation. Journal of Evolutionary Biology **22**:1435-1446.
682 Lande, R., and S. J. Arnold. 1983. The measurement of selection on correlated characters.
683 Evolution **37**:1210-1226.
684 Landry, C. R., and N. Aubin-Horth. 2014. Ecological Genomics. Springer.
685 Le Rouzic, A., J. M. Álvarez-Castro, and T. F. Hansen. 2013. The Evolution of Canalization
686 and Evolvability in Stable and Fluctuating Environments. Evolutionary Biology
687 **40**:317-340.
688 Lindstrom, E. 1988. Reproductive effort in the red fox, vulpes vulpes, and future-supply of a
689 fluctuating prey. Oikos **52**:115-119.
690 Lõmus, M., and L. F. Sundström. 2004. Leptin and social environment influece the risk-
691 taking and feeding behaviour of Asian blue quail. Animal Behaviour 68:607-612.
692 Martin, J. G. A., D. H. Nussey, A. J. Wilson, and D. Réale. 2011. Measuring individual
693 differences in reaction norms in field and experimental studies: a power analysis of
694 random regression models. Methods in Ecology and Evolution **2**:362-374.
695 McNamara, J. M., Z. Barta, M. Klaassen, and S. Bauer. 2011. Cues and the optimal timing of
696 activities under environmental changes. Ecology Letters **14**:1183-1190.
697 Meek, L. R., T. M. Lee, and J. F. Gallon. 1995. Interaction of maternal photoperiod history
698 and food type on growth and reproductive development of laboratory meadow voles
699 (microtus-pennsylvanicus). Physiology & Behavior **57**:905-911.
700 Miehls, A. L. J., A. G. Mcadam, P. E. Bourdeau, and S. D. Peacor. 2013. Plastic response to a
701 proxy cue of predation risk when direct cues are unreliable. Ecology **94**:2237-2248.

Morris, M., and S. M. Rogers. 2014. Integrating phenotypic plasticity within an ecological genomics framework: Recent insights from the genomics, evolution, ecology, and fitness of plasticity. Pages 73 - 105 *in* C. R. Landry and N. Aubin-Horth, editors. Ecological Genomics. Springer, Dordrecht.

Mueller, C. A., J. Eme, W. W. Burggren, R. D. Roghair, and S. D. Rundle. 2015. Challenges and opportunities in developmental integrative physiology. Comparative Biochemistry and Physiology a-Molecular & Integrative Physiology **184**:113-124.

Negus, N. C., and P. J. Berger. 1998. Reproductive strategies of Dicrostonyx groenlandicus and Lemmus sibiricus in high-arctic tundra. Canadian Journal of Zoology **76**:391-400.

Nijhout, H. F. 2003. Development and evolution of adaptive polyphenisms. Evolution & Development **5**:9–18.

Nussey, D. H., E. Postma, P. Gienapp, and M. E. Visser. 2005. Selection on heritable phenotypic plasticity in a wild bird population. Science **310**:304-306.

Nussey, D. H., A. J. Wilson, and J. E. Brommer. 2007. The evolutionary ecology of individual phenotypic plasticity in wild populations. Journal of Evolutionary Biology **831-844.**:831-844.

Parker, G., and J. Maynard Smith. 1990. Optimality theory in evolutionary biology. Nature **348**:27-33.

Pigliucci, M. 2005. Evolution of phenotypic plasticity: where are we going now? Trends in Ecology & Evolution **20**:481-486.

Pigliucci, M., C. J. Murren, and C. D. Schlichting. 2006. Phenotypic plasticity and evolution by genetic assimilation. Journal of Experimental Biology **209**:2362-2367.

Roff, D. A. 2002. Life history evolution. Sinauer Associates, Inc., Sunderland, MA.

Roff, D. A. 2010. Modelling evolution: : an introduction to numerical methods. Oxford University Press.

727 Rui, L. Y. 2013. Brain regulation of energy balance and body weight. Reviews in Endocrine
728 & Metabolic Disorders **14**:387-407.
729 Sanders, E. H., P. D. Gardner, P. J. Berger, and N. C. Negus. 1981. 6-
730 Methoxybenzoxazolinone: A plant derivative that stimulates reproduction in *Microtus*
731 *montanus*. Science **214**:67-69.
732 Scheiner, S. M. 1993. Genetics and evolution of phenotypic plasticity. Annual Review of
733 Ecology and Systematics **24**:35-68.
734 Scheiner, S. M. 2013. The genetics of phenotypic plasticity. XII. Temporal and spatial
735 heterogeneity. Ecology and Evolution **3**:4596-4609.
736 Schlichting, C. D., and H. Smith. 2002. Phenotypic plasticity: linking molecular mechanisms
737 with evolutionary outcomes. Evolutionary Ecology **16**:189-211.
738 Sengupta, S., T. Ergon, and H. P. Leinaas. 2015. Microevolutionary differences in embryonic
739 life history traits of the Collembola Folsomia quadrioculata (Tullberg 1871) across a
740 wide geographic range. Ecological Entomology DOI: 10.1111/een.12270.
741 Smith, H. 1990. Signal perception, differential expression within multigene families and the
742 molecular basis of phenotypic plasticity. Plant, Cell and Environment **13**:585-594.
743 Smith, H. 1995. Physiological and ecological function within the phytocrome family. Annu.
744 Rev. Plant Physiol. Plant Mol. Biol. **46**:289-315.
745 Starrfelt, J., and H. Kokko. 2012. Bet-hedging - a triple trade-off between means, variances
746 and correlations. Biological Reviews **87**:742-755.
747 Stearns, S. C. 1992. The Evolution of Life Histories. Oxford University Press, Oxford.
748 Stephens, D. W., J. S. Brown, and R. C. Ydenberg, editors. 2007. Foraging: Behavior and
749 Ecology. The University of Chicago Press.
750 Steppan, S. J., P. C. Phillips, and D. Houle. 2002. Comparative quantitative genetics:
751 evolution of the G matrix. Trends in Ecology & Evolution **17**:320-327.

752 Sultan, S. E. 2010. Plant developmental responses to the environment: eco-devo insights. Curr
753 Opin Plant Biol **13**:96-101.
754 Sultan, S. E., and S. C. Stearns. 2005. Environmentally contingent variation: phenotypic
755 plasticity and norms of reaction.*in* B. Hallgrímsson and B. K. Hall, editors. Variation;
756 a central concept in biology. Elsevier Academic Press.
757 Sutherland, W. J. 2005. The best solution. Nature **435**:569-569.
758 Svennungsen, T. O., O. H. Holen, and O. Leimar. 2011. Inducible Defenses: Continuous
759 Reaction Norms or Threshold Traits? American Naturalist **178**:397-410.
760 Tollrian, R., and C. D. Harvell, editors. 1999. The ecology and evolution of inducible
761 defences. Princeton University Press, Princeton, NJ.
762 Tufto, J. 2000. The Evolution of Plasticity and Nonplastic Spatial and Temporal Adaptations
763 in the Presence of Imperfect Environmental Cues. The American Naturalist **156**:121-
764 130.
765 Tufto, J. 2015. Genetic evolution, plasticity, and bet-hedging as adaptive responses to
766 temporally autocorrelated fluctuating selection: A quantitative genetic model.
767 Evolution 69:2034-2049.
768 Via, S., R. Gomulkiewicz, G. Dejong, S. M. Scheiner, C. D. Schlichting, and P. H.
769 Vantienderen. 1995. Adaptive phenotypic plasticity - consensus and controversy.
770 Trends in Ecology & Evolution **10**:212-217.
771 Waddington, C. H. 1953. Genetic assimilation of an acquired character. Evolution 7:118-126.
772 Waddington, C. 1961. Genetic assimilation. Advances in Genetics 10:257–293.
773 Wagner, G. P., G. Booth, and H. Bagheri-Chaichian. 1997. A Population Genetic Theory of
774 Canalization. Evolution **51**:329-347.
775 West-Eberhard, M. 2003. Developmental plasticity and evolution. Oxford University Press,
776 Oxford.

777 Woltereck, r. 1909. Weitere experimentalle Untersuchungen uber Artveranderung, speziell
778 uber das Wesen quantitativer Artenunterschiede bei Daphniden. Zool. Gesell.:110-
779 172.
780 Yoccoz, N. G., S. Engen, and N. C. Stenseth. 1993. Optimal foraging: The importance of
781 environmental stochasticity and accuracy in parameter estimation. American
782 Naturalist **141**:139-157.
783 Yoshimura, J., and C. W. Clark. 1991. Individual adaptations in stochastic environments.
784 Evolutionary Ecology **5**:173-192.
785

Figure legends

Figure 1. Conceptual overview of optimal linear reaction norms in stochastic environments. The environmental component U (cue) that determines the mean phenotype and the environmental component E determining the phenotypic expression that maximize fitness (Θ) have a bivariate distribution with correlation ρ (central 95% of a bi-normal distribution with $\rho = 0.5$ is indicated by the ellipses in the lower right panel). This leads to a bivariate distribution of U and Θ with means μ_U and μ_Θ, variances σ_U^2 and σ_Θ^2, and a correlation $\rho = \sigma_{U\Theta}/(\sigma_U\sigma_\Theta)$ (top right panel). The shaded areas show the conditional probability distributions of E and Θ given a cue value u (with $\rho = 0.5$). If fitness, W, is a Gaussian function of the plastic phenotype value $y(u)$, the optimal reaction norm as a function of cue values u is the same as the least squares prediction of Θ given u, $y_{opt}(u) = \mu_\Theta + \rho\frac{\sigma_\Theta}{\sigma_U}(u - \mu_U)$, Appendix A. Some authors refer to U in this context as a "proxy cue" of environmental component E. However, it is sufficient to only consider U and Θ as two correlated components of a temporally varying environment. Blue line represents the optimal reaction norm under perfect information ($\rho = 1$) (when the ellipses collapse to a line), and green line represents the optimal reaction norm when U and Θ are uncorrelated ($\rho = 0$). Solid red line represents the optimal reaction norm when $\rho = 0.5$ (corresponding to the drawn ellipses). Thick stippled red line is referred to in the Analysis section. Note that in Lande's (2009) notation, ε_t corresponds to a random value of E in generation t, and $\varepsilon_{t-\tau}$ corresponds to a random U in the same generation.

Figure 2. Evolution of linear reaction norms after a sudden environmental change. Panels A-C: Trajectories of the population mean trait values with a sudden environmental change at generation 5000 (see text). Panel D: Phase plane diagram showing $\bar{z}_a$ plotted against $\bar{z}_c$ through all generations (this is the point in the cue-phenotype plane where reaction norms "tend to cross" (see Fig. 3), since the phenotypic variance-covariance matrix here is diagonal (see eq. (5)). Solid blue lines represent the three-trait model (3) and the stippled red lines represent the two-trait model (2). The trajectories were calculated as the mean of 1000 independent simulations. Grey lines show the realization of a single simulation. Solid green lines show μ_Θ (panel A), the optimal slope when reaction norm slope and

intercept can be tuned independently, $\sigma_{U\Theta}/\sigma_U^2$ (eq. (1)) (panel B), and μ_U (panel C). In panel A, the dotted blue line is the mean intercept $(\bar{z}_a - \bar{z}_b\bar{z}_c - P_{bc})$ in the three-trait model for comparison with the intercept trait in the two-trait model (stippled red line). In panel B, stippled green line shows the mean slope that gives maximum expected logarithm of fitness of a random individual (Appendix C). Parameter values in the initial environment were $\mu_U = 0$, $\mu_\Theta = 0$, $\sigma_U = 2$, $\sigma_\Theta = 4$, and $\rho = \frac{\sigma_{U\Theta}}{\sigma_U\sigma_\Theta} = 0.25$. At generation 5000, μ_U jumps to 6 and μ_Θ jumps to 12 while the other parameters remain unchanged. Diagonal $\boldsymbol{G}$ and $\boldsymbol{P}$ matrices were used with $G_{aa} = 0.5$, $P_{aa} = G_{aa} + 0.5$, $P_{bb} = G_{bb} = 0.045$, and $P_{cc} = G_{cc} = 2$ (three-trait model) or $P_{cc} = G_{cc} = 0$ (two-trait model). Initial mean trait values were $\bar{z}_a = 0$, $\bar{z}_b = \rho\frac{\sigma_\Theta}{\sigma_U} = 0.5$, and $\bar{z}_c = 0$.

Figure 3. Reaction norm distribution when the populations have reached a stationary dynamics in the two-trait model (A) and the three-trait model (B) under the scenario presented in Figure 2. The distribution of the environmental cue (U) in the new environment is indicated by the shaded areas on the x-axes, and the central 95% of the joint distribution of U and Θ is shown with the ellipses with an '×' at the mean. For each model, 50 random reaction norms (genotypes) are plotted. In the two-trait model, the cue value u_0 where phenotypic variation is minimal will always be at zero when reaction norm slope and intercept are independent (indicated with a white, crossed, symbol plotted at the mean plastic phenotype for this cue value). In contrast, in the three-trait model genetic assimilation becomes complete and u_0 moves to μ_U with a mean plastic phenotype at μ_Θ.

Figure 4: Phenotypic standard deviation, $SD(y)$ (**A**), and the expected plastic component of the phenotype, $\bar{z}_b(\mu_U - u_0)$ (**B**) in the simulations represented in Figure 2. Blue solid lines represent the three-trait model, while the red stippled lines represent the two-trait model. Horizontal grey lines are drawn at the mean values of the last 3000 generations prior to the sudden environmental change at generation 5000. Lines show the mean of 1000 independent simulations plotted at every 100th generation.

840 Figure 4. Fitness trajectories in the simulation example (Figure 2). Left panels: Population mean
841 fitness relative to maximum fitness (W_{max}). Right panels: Expected fitness at the mean trait values.
842 The lower panels show the same values plotted with a narrower range on the y-axis. Thick blue line
843 represents the three-trait model and the thin red line represents the two-trait model. Horizontal stippled
844 green line shows the fitness of the optimal reaction norm (1). Only every 20^{th} generation is plotted in
845 the left panels and every 100^{th} generation is plotted in the right panels. Plotted values are the mean of
846 the same 1000 independent simulations used for Figure 2.
847

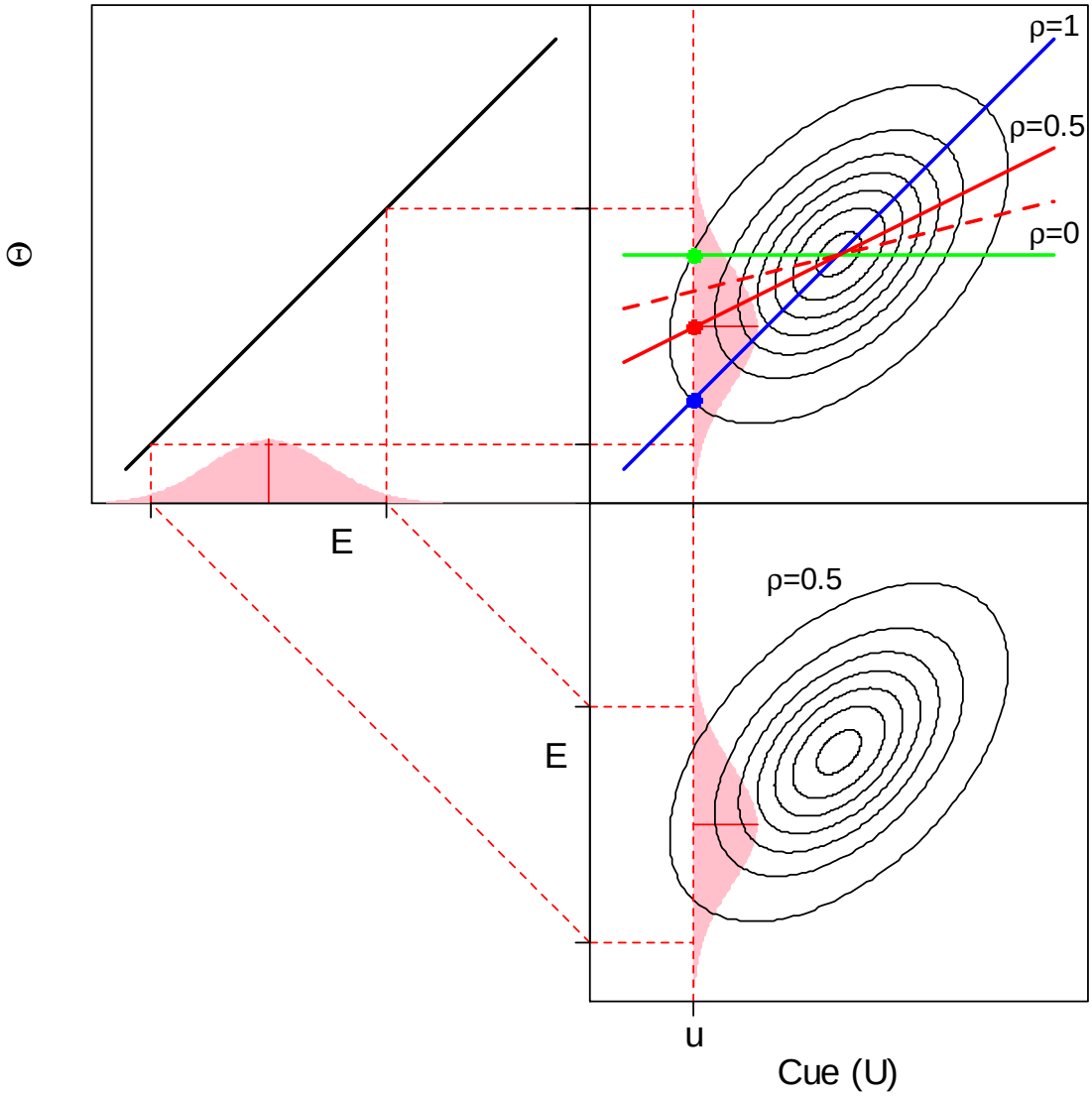
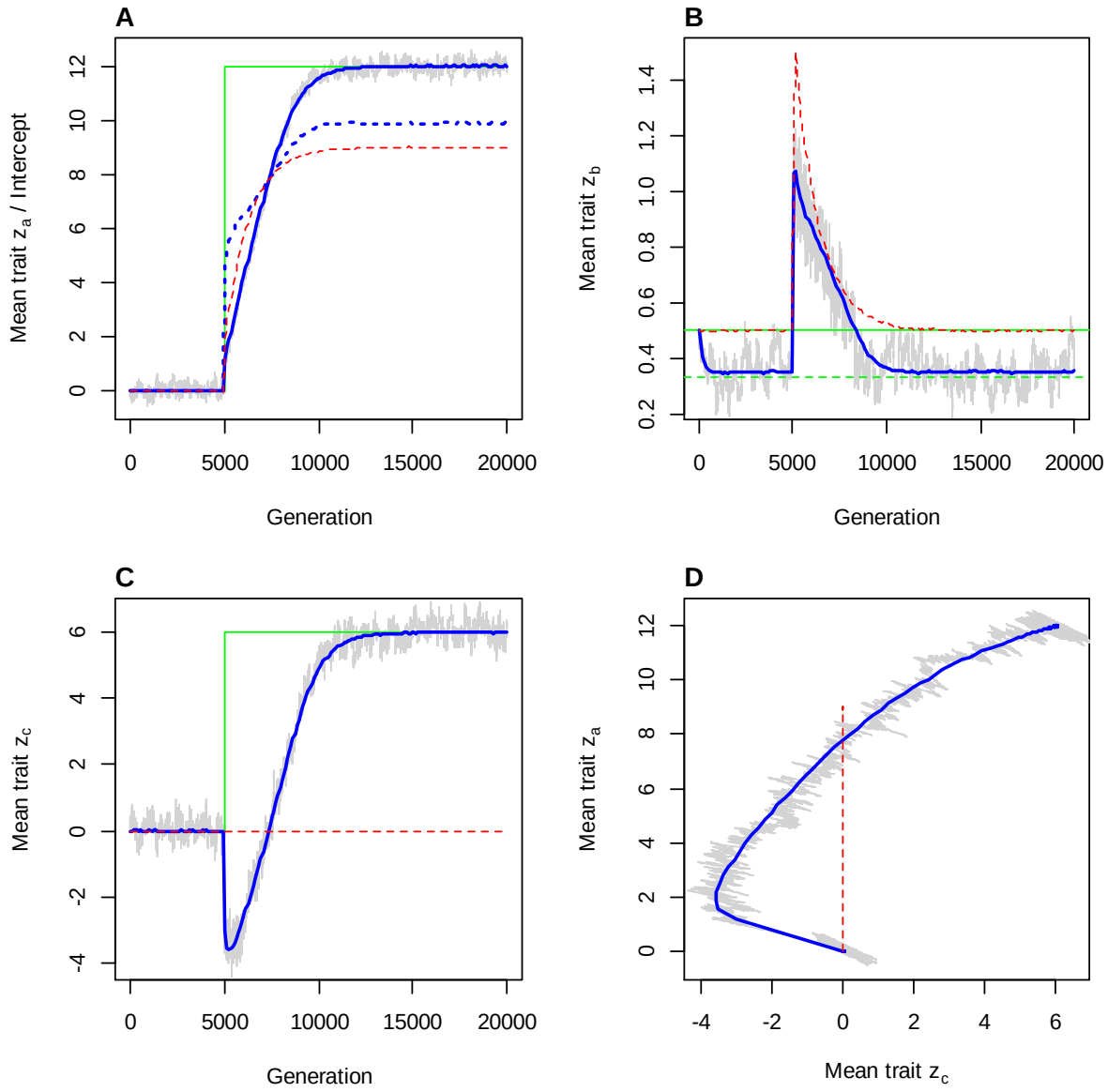


Figure 1



851

852 **Figure 2**

853

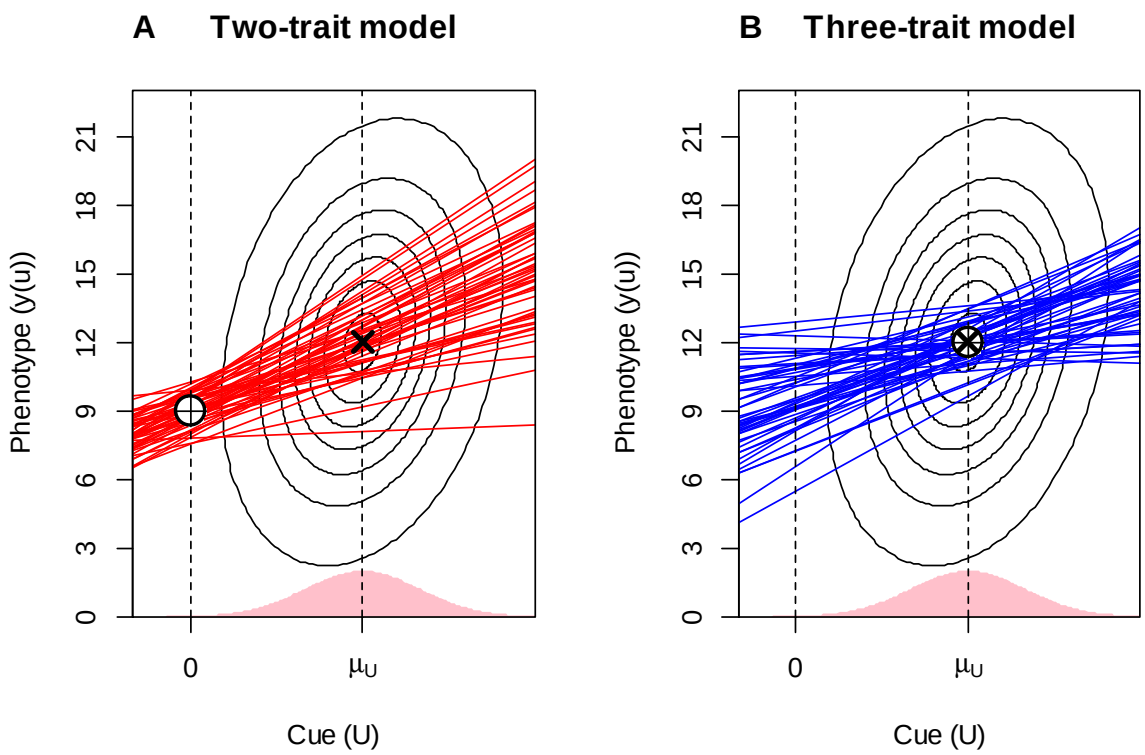


Figure 3

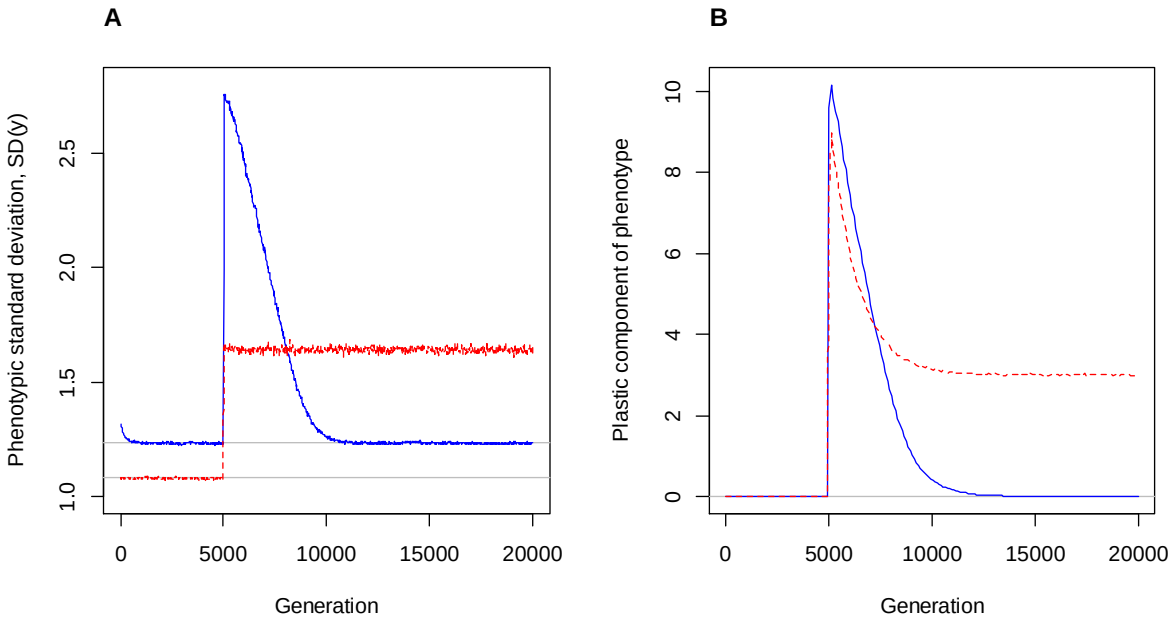


Figure 4

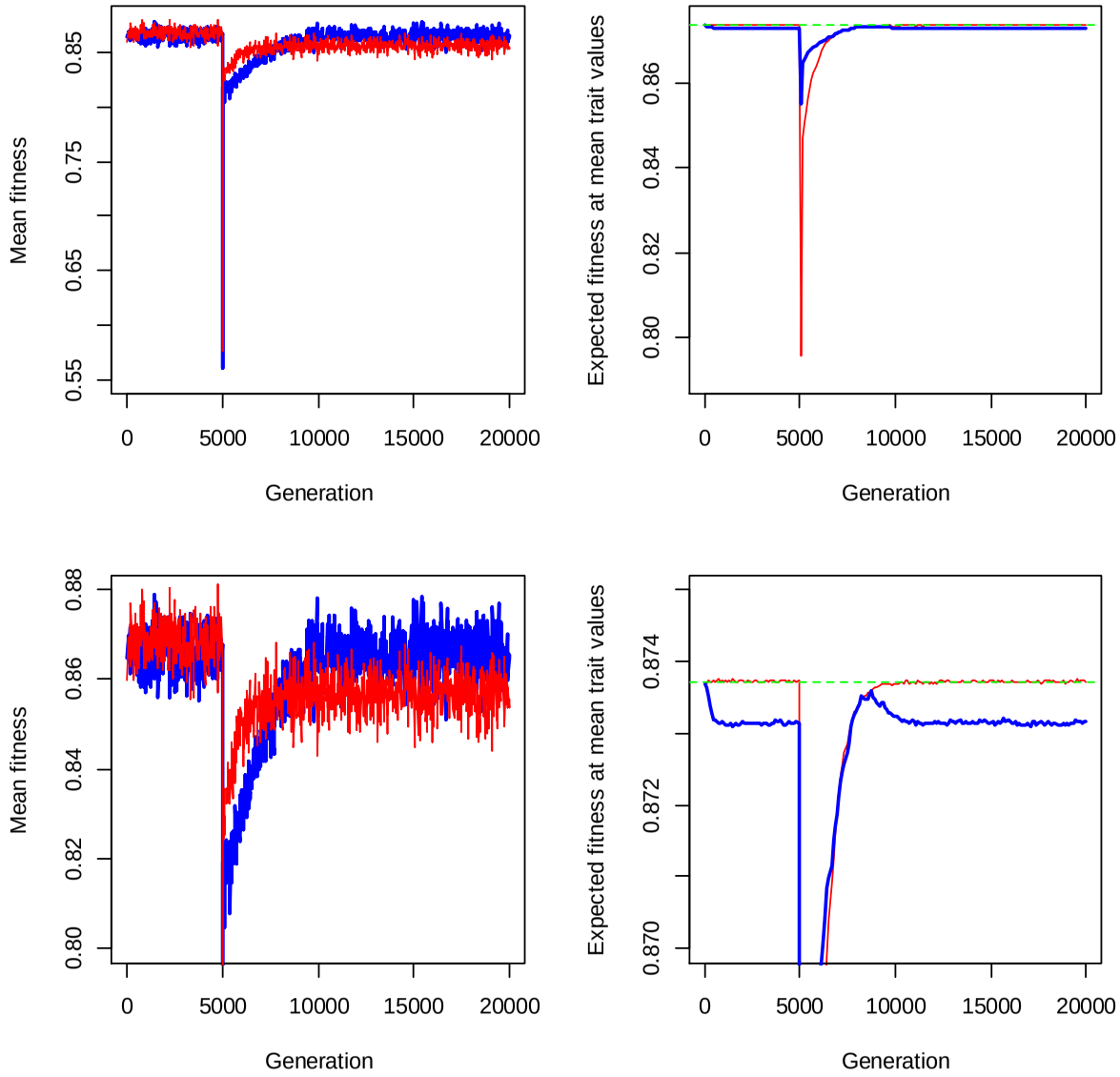


Figure 5