

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

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1 **Abstract**

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

16 **Introduction**

17 All natural populations evolve in environments that are to some degree variable. Biologists have long
18 realized that the phenotypic expression of different genotypes may respond differently to the same
19 environmental change, and that such phenotypic plasticity may be heritable (DeWitt and Scheiner
20 2004, Pigliucci 2005). Depending on the effect this phenotypic plasticity has on selection (fitness),
21 evolution may thus bring about mechanisms that either buffer the phenotypic expression against
22 environmental variation (i.e., environmental canalization) or modify the responses to some
23 environmental influence in an adaptive manner (Nijhout 2003). Phenotypic plasticity involves
24 developmental, physiological and/or behavioral phenotypic responses to some component(s) of the
25 environment (DeWitt and Scheiner 2004, Pigliucci 2005, Pigliucci et al. 2006). These environmental

26 components, often referred to as environmental ‘cues’ (DeWitt and Scheiner 2004), are often just
27 correlated with, but not identical to, the environmental variables affecting fitness (e.g. McNamara et
28 al. 2011, Svanungsson et al. 2011, Gienapp et al. 2014). Hence, cues do not provide perfect
29 information about the optimal phenotypic expression, and it is usually adaptive to respond more
30 conservatively towards information-poor cues than more informative ones (Yoccoz et al. 1993, Ergon
31 2007, McNamara et al. 2011). The phenotypic expression of a particular genotype as a function of
32 environmental cues is called a reaction norm (Woltereck 1909, Pigliucci 2005). There has been
33 considerable interest in evolutionary processes governing reaction norms as this is crucial for our
34 understanding of how populations may respond to environmental change (e.g. Lande 2009, McNamara
35 et al. 2011, Gienapp et al. 2014).

36 Waddington (1953, 1961) originally used the term ‘genetic assimilation’ to describe
37 experimental selection results where qualitative phenotypes (such as lack of cross-veins in *Drosophila*
38 wings) that are initially only expressed in response to a particular environmental stimuli (such as heat
39 shock during a particular stage of development) becomes constitutively produced (i.e., becomes
40 expressed independently of the environmental stimuli) after continued selection. However, ‘genetic
41 assimilation’ is also used to describe similar phenomena in evolution of the mean of quantitative
42 phenotypes that may remain plastic at equilibrium in a stochastic environment after an environmental
43 change (Pigliucci and Murren 2003; Lande 2009). In such cases, the new equilibrium phenotypes will
44 not be independent of the environment unless the reaction norm slope is zero. We adopt Lande’s
45 (2009) definition of genetic assimilation in an altered environment as the reduction in the plastic
46 component of the phenotype with a concomitant genetic evolution, while maintaining the phenotype
47 initially produced by plasticity in the altered environment. By ‘plastic component’ we here mean the
48 difference between the phenotypic value and the mean phenotype in the environment where
49 phenotypic variance is minimized (i.e., where individual reaction norms tend to cross; see Fig. 1 in
50 Pigliucci et al. 2006). We consider this process of genetic assimilation as complete when the expected
51 plastic component of the phenotype is zero and the phenotypic variance is minimized in the new mean
52 environment (but both mean reaction norm slope and phenotypic variance in the mean environment
53 may remain non-zero; see Fig. 1 in Pigliucci and Murren 2003 and Fig. 2 in Pigliucci et al. 2006).

54 Several experiments, mainly on the fruit fly *Drosophila melanogaster*, have demonstrated
55 genetic assimilation in laboratory populations (Braendle and Flatt 2006), and there is substantial
56 evidence for genetic assimilation, also for quantitative traits, from field observations and experiments
57 (reviewed by Pigliucci and Murren 2003). The most commonly proposed mechanisms for genetic
58 assimilation is that extraordinary environments may expose hidden genetic variation in reaction norms
59 for subsequent selection, followed by eventual canalization of the phenotypic expression in the new
60 environment (Braendle and Flatt 2006, Pigliucci et al. 2006, Lande 2009). The latter stage of this
61 process is perhaps the least understood; it has been suggested that genetic drift or fitness costs of
62 maintaining plasticity plays a part (West-Eberhard 2003, Pigliucci et al. 2006, Lande 2009, Bateson
63 and Gluckman 2011), and changes in the genetic variances, covariances and genetic architecture of
64 reaction norm components may be involved (Wagner et al. 1997, Steppan et al. 2002, Le Rouzic et al.
65 2013).

66 One approach to quantitative genetic analysis of phenotypic plasticity (Via et al. 1995, Rice
67 2004) is to consider the intercept and slope of linear reaction norms as two quantitative traits in their
68 own right (de Jong 1990, Gavrillets and Scheiner 1993a, de Jong and Gavrillets 2000, Tufto 2000,
69 Lande 2009). More generally, reaction norms have been modeled by considering polynomial
70 coefficients as traits (Gavrillets and Scheiner 1993b, Scheiner 1993). In these models, the intercept trait
71 is the phenotypic value at a reference cue designated as zero. Lande (2009) analyzed the evolution of
72 such a linear reaction norm, assuming a stochastic environment undergoing a sudden extreme change
73 (relative to the background fluctuations) in both the mean environmental cue and the phenotypic value
74 where fitness is maximum. In his model the population responded by a rapid increase in mean reaction
75 norm slope (plasticity), followed by a slow increase in reaction norm elevation at the reference cue
76 with a concomitant decrease in plasticity. However, the genetic assimilation was not completed, as the
77 cue value at which the phenotypic variance was at its minimum could never move away from the
78 reference cue because the covariance between reaction norm slope and intercept was assumed to
79 remain constant. Lande (2009) argued that further canalization would take place (e.g., due to fitness
80 costs of maintaining plasticity), but did not include any such mechanisms in his modeling.

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

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

103 **Models**

104 **Optimal linear reaction norms in temporally variable environments**

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

107 exact information about the state of the environment (Yoshimura and Clark 1991, Roff 2002).
108 Whenever the phenotype yielding highest fitness is not known exactly (i.e., the individuals do not have
109 full information about the present and future environment), the long term success of a genotype
110 depends not only on the expectation of fitness, but it is also adaptive to reduce the variance in mean
111 fitness across generations (Yoshimura and Clark 1991, Starrfelt and Kokko 2012). Models that assume
112 that individuals have no information about the environment have been used to explain risk-avoidance
113 and bet-hedging strategies (den Boer 1968, Hopper et al. 2003, Starrfelt and Kokko 2012). On the
114 other side of the spectrum, models that predict optimal trait values as a function of environmental
115 variables, often assume that these variables are known to the individuals without error (e.g. Stearns
116 1992, Roff 2002).

117 The concept that phenotypic expressions are functions of more or less informative
118 environmental cues is well established in evolutionary ecology (Tollrian and Harvell 1999, DeWitt
119 and Scheiner 2004, Stephens et al. 2007, McNamara et al. 2011, Gienapp et al. 2014). For example,
120 seasonal reproduction in many organisms must take place within a rather narrow time-window which
121 often varies largely between years (Durant et al. 2007, Gienapp et al. 2014). Since such phenological
122 events must often be prepared a long time in advance (due to acquiring resources, physiological
123 developments and migration), seasonal reproduction may be influenced by rather information-poor
124 cues such as temperature and food constituents weeks before reproductive success is determined
125 (Berger et al. 1981, Korn and Taitt 1987, Lindstrom 1988, Negus and Berger 1998, Nussey et al.
126 2005). Examples of such obviously adaptive phenotypic plasticity to more or less informative
127 environmental cues are ubiquitous in nature (Pigliucci 2005, Sultan 2010, Landry and Aubin-Horth
128 2014).

129 To derive an optimal norm of reaction to an imperfect cue, we may view the cue (U) and the
130 phenotypic expression that maximize fitness (Θ) as having a joint distribution with given means, μ_U
131 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
132 (U) in a general sense as the *environmental component* that affects the phenotype, *not* how this
133 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 $\ln(W(y))$ is a quadratic function, the optimal *linear* reaction norm as a function of cue values u is

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

143 (Appendix A). Note that, due to the quadratic fitness function $\ln(W(y))$, 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 $E[\Theta|U = u]$ 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

$$y(u) = \alpha + \beta u \quad (2)$$

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

$$y(u) = z_a + z_b(u - z_c) \quad (3)$$

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

199 Note that the two-trait model (eq. (2)) is a special case of the more general three-trait model
200 (eq. (3)) where z_c is fixed to zero. Reaction norm slope is considered as a trait in both models (i.e.,
201 $\beta = z_b$), but we have used a separate notation in the two models for clarity.

202 **Analysis**

203 **Basic properties of the reaction norm models**

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

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

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

217 (Appendix B).

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

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

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

224 where P_{bc} , P_{ab} and P_{bb} are the elements of the phenotypic variance-covariance matrix indicated by the
225 subscripts (Appendix B). Thus, in this quantitative genetic model, u_0 may respond directly to
226 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
227 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} .

228 Lande (2009) defined the cue u ($\varepsilon_{t-\tau}$ in his model) to have its zero-point at u_0 as a “reference
229 environment”. Hence, one could define the two-trait model analyzed by Lande (2009) for any arbitrary
230 interval scaled cue variable as $y(u) = \alpha' + \beta(u - u_0)$ where the genetic correlation between the traits
231 α' and β is by necessity zero since u_0 is defined by $cov(y(u_0), \beta) = cov(\alpha', \beta) = 0$ (Appendix B;
232 see also last paragraph on page 1438 in Lande (2009)). This model is structurally similar to our three-
233 trait model except that the “reference environment” in our model is considered as an individual trait,
234 z_c (reflecting individual variation in cue “perception”), which is exposed to selection. Unlike in
235 Lande’s (2009) model, where the definition of trait α' depends on u_0 , there are no constraints on the
236 phenotypic or genotypic covariances in our three-trait model (other than that the covariance matrix
237 must be positive-definite). The two-trait model of Lande (2009) can only evolve in the same way as
238 the three-trait model if u_0 is treated as the mean of an individual trait with variance different from
239 zero. Hence, the three-trait quantitative genetics model and Lande’s (2009) two-trait model are not
240 alternative parameterizations of the same model. Lande’s (2009) two-trait model is a constrained
241 (nested) version of our more general three-trait model with the trait z_c fixed to u_0 , which requires that
242 $P_{cc} = P_{ac} = P_{bc} = 0$ as well as $P_{ab} = 0$ ($P_{ab} = 0$ is only required to maintain the same definition of
243 z_a and α' and to give $\bar{z}_c = u_0$).

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

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

254 **Evolution of linear reaction norms**

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

268 ***Quantitative genetics – modeling***

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

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

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

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

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

277 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
 278 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
 279 covariance matrix \mathbf{P} . When the phenotypic expression that maximizes fitness in that generation is θ_t ,
 280 this individual will have fitness

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

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

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

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

291 In the simulation results presented in Figure 2, we used the same parameter values as in
292 Lande's (2009) analysis of the two-trait model except that we, for convenience, used a somewhat less
293 extreme sudden change in the environment, with a change in μ_U and μ_Θ of 3 (instead of 5) standard
294 deviations of the background fluctuations (σ_U and σ_Θ of equation (1)). As Lande (2009) we used a
295 diagonal \mathbf{G} -matrix and set G_{cc} to half the cue variance (three-trait model) or zero (two-trait model). For
296 simplicity, in the simulations we also assumed that only trait z_a had a non-additive residual
297 component with variance σ_e^2 , such that $P_{aa} = G_{aa} + \sigma_e^2$, $P_{bb} = G_{bb}$, $P_{cc} = G_{cc}$, and $P_{ab} = P_{ac} =$
298 $P_{bc} = 0$. The two-trait model is obtained simply by setting also $P_{cc} = 0$ and $\bar{z}_c = 0$.

299 ***Quantitative genetics – results***

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

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

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

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

326 Interestingly, as seen in Figure 2B, the mean reaction norm slope \bar{z}_b in the three-trait model,
327 stabilizes at a lower level than the optimal slope yielding the highest expected fitness of an individual,
328 $\sigma_{U\Theta}/\sigma_U^2$ (see eq. (1)), which is also the equilibrium mean slope in the two-trait model (Gavrilets and
329 Scheiner 1993a, Lande 2009). Intuitively, this is because the optimal value of trait z_b of an individual
330 depends on the value of trait z_c that this individual possesses, which is stochastic. As it is not
331 straightforward to calculate equilibrium mean traits in the three-trait model (see Supplementary
332 Information S2), we first investigated this by calculating the mean trait values that yield maximum
333 expected logarithm of fitness, $E[\ln(W)]$, for a *random* individual in the population (Appendix C).

334 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
 335 $\bar{z}_c^{(*)} = \mu_U$, which is close to the stationary means in the simulations (Figure 2). For comparison, the
 336 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
 337 and Scheiner 1993a, Lande 2009). Note that the denominator in the expression for $\bar{z}_b^{(*)}$ is the variance
 338 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;
 339 i.e., genetic variance in the perception trait z_c inflates the variance of the perceived cue $(U - z_c)$.
 340 Hence, if $P_{ac} = 0$, $\bar{z}_b^{(*)}$ is always lower than the optimal slope in equation (1) unless $P_{cc} = 0$ (which
 341 gives the two-trait reaction norm model). This is indicated by a stippled reaction norm in Figure 1.
 342 Note again, however, that selection will maximize $E[\ln(\bar{W})]$ in the population and not $E[\ln(W)]$ for a
 343 random individual. Nevertheless, as shown in Figure 2 and Supporting Information S2, maximizing
 344 $E[\ln(\bar{W})]$ 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
 345 maximize $E[\ln(W)]$), but the equilibrium mean slope \bar{z}_b^* equals $\bar{z}_b^{(*)}$ above only when $\sigma_U^2 = \sigma_\Theta^2 =$
 346 $\sigma_{U\Theta} = 0$ (i.e. only in a constant environment). Note that \bar{z}_a^* and \bar{z}_c^* are independent of the variances
 347 and covariance of U and Θ when $P_{ab} = P_{bc} = 0$. In Supporting Information S2 we conjecture that the
 348 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
 349 $P_{ab} = P_{bc} = 0$, \bar{z}_a^* and \bar{z}_c^* are independent of \bar{z}_b^* , and hence also of σ_U^2 , σ_Θ^2 , and $\sigma_{U\Theta}$).

350 As seen in Figure 2B the asymptotic mean \bar{z}_b in the simulations (where $P_{ac} = 0$) is close to
 351 but somewhat larger than $\bar{z}_b^{(*)} = \sigma_{U\Theta}/(\sigma_U^2 + P_{cc})$. This discrepancy is to be expected for two reasons.
 352 First, as pointed out above, the mean trait $\bar{z}_b^{(*)}$ that maximizes $E[\ln(W)]$ for a random individual is not
 353 identical to the equilibrium mean trait where the selection gradient (7) is zero, i.e. where $E[\ln(\bar{W})]$ is
 354 maximized (Supporting Information S2). As shown in Supporting Information S2, the equilibrium
 355 mean reaction norm slope \bar{z}_b^* can be approximated analytically if we assume that the plastic phenotype
 356 $y(u)$ has a normal distribution, which is very nearly the case with the parameter values in our
 357 simulations in Figure 2. The integral (9) then has an analytical solution, and as a result an approximate
 358 equilibrium slope \bar{z}_b^* can be found numerically from the equation (assuming $P_{ab} = P_{bc} = 0$)

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

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

363 The second reason for the discrepancy between the asymptotic mean \bar{z}_b in the simulations and
 364 $\bar{z}_b^{(*)}$ is that when the population under directional selection based on equation (6) evolves towards a
 365 stationary state, the mean traits will fluctuate around the equilibrium because of the influence from the
 366 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.
 367 (where $E[\bar{z}_a] = \lim_{N \rightarrow \infty} \frac{1}{N} \sum_{t=1}^N \bar{z}_{a,t}$ and $E[v_a] = 0$ etc.), and, as shown in Supporting Information S2,
 368 the variances and covariances of v_a , v_b and v_c then enter into equation (10). Note that we assume that
 369 u_t and θ_t have zero autocorrelation, such that the covariances between the mean reaction norm
 370 parameters and the environment caused by adaptive tracking (Tufto 2015) can be neglected.

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

382 Discussion

383 Quantitative genetics models are theoretical models for the joint evolution of population means of
384 quantitative individual phenotypic traits, where the researchers define traits that they find most
385 meaningful in the context they are studied. In quantitative genetics models of reaction norms where
386 plastic phenotypes are modeled as a linear function of an interval scaled environmental cue, the
387 reaction norm intercept and slope are often considered as individual traits subjected to selection
388 (Gavrilets and Scheiner 1993b, Scheiner 1993, de Jong and Gavrilets 2000, Tufto 2000, Lande 2009,
389 Scheiner 2013, Tufto 2015). The intercept of such a reaction norm (i.e., the reaction norm value at cue
390 value zero) is often not very biologically meaningful since this trait, as well as its variance and
391 covariance with other traits, depend on the defined zero-point, or “reference cue”, of the (arbitrary)
392 interval scaled cue variable. One may, however, as in Lande (2009), define the zero-point of the cue to
393 be the mean cue value for which the population is adapted to. This ensures that the variance of the
394 plastic phenotype is minimized in the mean environment, which is theoretically plausible (Bürger
395 2000, Lande 2009, Le Rouzic et al. 2013), but it is not clear how this “reference cue” may evolve (in
396 Lande’s (2009) analysis it is assumed to remain constant; see however de Jong and Gavrilets 2000).

397 We have here suggested that the “reference cue” can be considered as an individual trait that
398 reflects genetic variation in cue “perception” in a general sense, and hence considered a linear reaction
399 norm on the form $y(u) = z_a + z_b(u - z_c)$. In this model, the biological meaning of all the traits, and
400 their variances and covariances, is not modified when redefining the zero-point of the cue variable u
401 (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
402 model reflect three fundamentally different genetic effects on linear reaction norms. While z_b
403 represents genetic effects on cue sensitivity, z_c reflects genetic effects on cue “perception” (in the
404 general sense discussed below) and has the same scale as the environmental cue, and z_a represents
405 genetic effects that are both independent of the cue value and are invariant to its defined zero-point
406 (the latter is not the case for the intercept). These structural differences in the reaction norm models
407 matter for the equilibrium mean reaction norms (and distributions) because the traits do not have
408 independent effects on the plastic phenotype ($y(u)$) (note the product $z_b z_c$ in the three-trait model).

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

432 In the three-trait model, phenotypic variance in a given environment increases with both \bar{z}_b
433 and the distance between \bar{z}_c and the environmental cue (u), at least when the traits are independent
434 (see equation S2-3 in Supporting Information S2), whereas in the two-trait model, phenotypic variance
435 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

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

471 While there is ample evidence for widespread genetic variation for reaction norms in natural
472 populations (Falconer and Mackay 1996, Sultan and Stearns 2005, Sengupta et al. 2015), there are not
473 many examples where the full pathway of phenotypic plasticity from cue perception to phenotype
474 expression is known in great detail (Sultan 2010, Morris and Rogers 2014), and even less is known
475 about the genetic variation of the different elements of these pathways. It seems, however, obvious
476 that there may be substantial genotypic variation in perception of (and not just responses to)
477 environmental cues (i.e., variation in trait z_c in our model). Examples indicating genetic variation in
478 environmental perception include, among other examples, substantial among-population variation in
479 the signal transduction pathway of induced plant defense in *Arabidopsis thaliana* (Kliebenstein et al.
480 2002), and individual variation in systemic stress responses has likely components of individual
481 variation in what is perceived as stressful (Hoffmann and Parsons 1991, Badyaev 2005, Dingemanse et
482 al. 2010). There is also considerable variation and “fine tuning” in light (and shading) perception
483 systems involving phytochromes that are sensitive to different wave lengths in plants (Smith 1990,
484 1995, Schlichting and Smith 2002).

485 **Predictions and empirical evaluations**

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

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

500 The cue value that gives minimum phenotypic variation in the population (u_0), may be
501 estimated by fitting data on genotype specific phenotypic measurements to mixed-effects linear
502 models with random individual slopes and intercepts (Martin et al. 2011, Bates et al. 2014), or from a
503 random regression “animal model” building on a known relatedness among individuals (Nussey et al.
504 2007). Our three-trait quantitative genetics model gives certain predictions about the evolution of u_0
505 under environmental change. Our analysis shows that the mean cue reference trait (\bar{z}_c), and hence u_0
506 (eq. (5)), will respond rapidly to changes in the mean environment (provided sufficient additive
507 genetic variation). Whenever there is selection for increased plasticity (i.e., selection for higher $|\bar{z}_b|$),
508 it also becomes adaptive to exaggerate the perception of the environmental change, and u_0 will swing
509 away in the *opposite* direction of the change in the mean cue during a “phase of alarm” (see Figure 2).
510 Later, u_0 will move towards, and eventually fluctuate around, the new cue value. In contrast, under the
511 two-trait model u_0 will not change in response to changes in the mean cue values.

512 **Future directions**

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

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

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

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

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544 Oslo.

545 **Appendix A: Optimal reaction norms**

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

549

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

550

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

554 A linear reaction norm, may be described as $y(U) = \alpha + \beta U$. To find the intercept (α) and

555 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

556 $E[y(U)^2] = var(y(U)) + E[y(U)]^2$, etc., we get

557

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

558

559 Substituting $var(y(U)) = \beta^2 \sigma_U^2$ and $cov(y(U), \Theta) = \beta \sigma_{U\Theta}$, and further solving $\frac{\partial J}{\partial \alpha} = 0$ and

560 $\frac{\partial J}{\partial \beta} = 0$ 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

561 correspond to equation (1) in the main text.

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

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

567

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

568

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

570

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

571

572 We find the same from

573

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

574

575 In the three-trait genetic model (3), on the other hand, the reaction norm intercept and slope
576 are $\alpha = z_a - z_b z_c$ and $\beta = z_b$, respectively. Since $E[z_a - z_b z_c] = \bar{z}_a - E[(z_b - \bar{z}_b + \bar{z}_b)(z_c - \bar{z}_c +$
577 $\bar{z}_c)] = \bar{z}_a - P_{bc} - \bar{z}_b \bar{z}_c$, and since normal distributions give $E[(z_b - \bar{z}_b)^2 (z_c - \bar{z}_c)] = 0$ (Isserlis'
578 (1918) theorem), we then have

579

$$\begin{aligned}
 P_{\alpha\beta} &= E[(z_a - z_b z_c - \bar{z}_a + P_{bc} + \bar{z}_b \bar{z}_c)(z_b - \bar{z}_b)] \\
 &= P_{ab} - E[(z_b - \bar{z}_b + \bar{z}_b)(z_c - \bar{z}_c + \bar{z}_c)(z_b - \bar{z}_b)] = P_{ab} - \bar{z}_c P_{bb} - \bar{z}_b P_{bc}
 \end{aligned}$$

580

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

582

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

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

584 We here apply the same procedure as in Appendix A to find the mean trait values in the population
 585 that maximize $E[\ln(W)]$ for a random individual for which the traits are stochastic quantities. Starting
 586 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
 587 distributed with phenotypic covariance matrix \mathbf{P} , we develop the criterion function J by using
 588 $z_a = z_a - \bar{z}_a + \bar{z}_a$ etc., and $E[(z_a - \bar{z}_a)(z_a - \bar{z}_a)] = P_{aa}$ etc. According to Isserlis' (1918) theorem
 589 (given normal traits) we have $E[(z_a - \bar{z}_a)(z_b - \bar{z}_b)(z_c - \bar{z}_c)] = 0$, $E[(z_b - \bar{z}_b)^2(z_c - \bar{z}_c)] = 0$ and
 590 $E[(z_b - \bar{z}_b)(z_c - \bar{z}_c)^2] = 0$. Further assuming that the traits are uncorrelated with both Θ and U we
 591 find

$$\begin{aligned}
 J &= E[(z_a + z_b(U - z_c) - \Theta)^2] \\
 &= E\left[\left(\begin{aligned} &z_a - \bar{z}_a + \bar{z}_a + (z_b - \bar{z}_b)(U - \mu_U) + (z_b - \bar{z}_b)(\mu_U - \bar{z}_c) - (z_b - \bar{z}_b)(z_c - \bar{z}_c) \\ &+ \bar{z}_b(U - \mu_U) + \bar{z}_b(\mu_U - \bar{z}_c) - \bar{z}_b(z_c - \bar{z}_c) - (\Theta - \mu_\Theta) - \mu_\Theta \end{aligned}\right)^2\right] \\
 &= P_{aa} + 2P_{ab}(\mu_U - \bar{z}_c) - 2\bar{z}_b P_{ac} + \bar{z}_a^2 - 2\bar{z}_a P_{bc} + 2\bar{z}_a \bar{z}_b(\mu_U - \bar{z}_c) - 2\bar{z}_a \mu_\Theta + P_{bb} \sigma_U^2 \\
 &\quad + P_{bb}(\mu_U - \bar{z}_c)^2 - 2P_{bc} \bar{z}_b(\mu_U - \bar{z}_c) + P_{bb} P_{cc} - 2P_{bc} \bar{z}_b(\mu_U - \bar{z}_c) + 2P_{bc} \mu_\Theta \\
 &\quad + \bar{z}_b^2 \sigma_U^2 - 2\bar{z}_b \sigma_{U\Theta} + \bar{z}_b^2(\mu_U - \bar{z}_c)^2 - 2\bar{z}_b(\mu_U - \bar{z}_c)\mu_\Theta + \bar{z}_b^2 P_{cc} + \sigma_\Theta^2 + \mu_\Theta^2
 \end{aligned}$$

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

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

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

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

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

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

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

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

595 With $P_{bb} = P_{ab} = P_{bc} = 0$ and hence constant z_b (i.e., “Baldwin effect” sensu Lande (2009))

596 we can only show that $\bar{z}_a^{(*)} + \bar{z}_b^{(*)}(\mu_U - \bar{z}_c^{(*)}) = \mu_\Theta$, i.e., that the point $[\bar{z}_c^{(*)}, \bar{z}_a^{(*)}]$ is located on

597 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

598 point $[\bar{z}_c^{(*)}, \bar{z}_a^{(*)}]$ is thus forced towards the solution above only when $P_{bb} > 0$.

599 The traditional two-trait model is a special case of the three-trait model where $\bar{z}_c = 0$, $\bar{z}_a = \alpha$,

600 $\bar{z}_b = \beta$, $P_{cc} = 0$, $P_{ac} = 0$ and $P_{bc} = 0$. Solving $\frac{\partial J}{\partial \alpha} = 0$ and $\frac{\partial J}{\partial \beta} = 0$ gives the solution $\alpha^{(*)} = \mu_\Theta -$

601 $\frac{\sigma_{U\Theta}}{\sigma_U^2} \mu_U$ and $\beta^{(*)} = \frac{\sigma_{U\Theta}}{\sigma_U^2}$, which is identical to the optimal intercept and slope derived in Appendix A, as

602 well as the asymptotic trait means found by Lande (2009).

603

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- 785

786 **Figure legends**

787 Figure 1. Conceptual overview of optimal linear reaction norms in stochastic environments. The
788 environmental component U (cue) that determines the mean phenotype and the environmental
789 component E determining the phenotypic expression that maximize fitness (Θ) have a bivariate
790 distribution with correlation ρ (central 95% of a bi-normal distribution with $\rho = 0.5$ is indicated by
791 the ellipses in the lower right panel). This leads to a bivariate distribution of U and Θ with means μ_U
792 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
793 show the conditional probability distributions of E and Θ given a cue value u (with $\rho = 0.5$). If
794 fitness, W , is a Gaussian function of the plastic phenotype value $y(u)$, the optimal reaction norm as a
795 function of cue values u is the same as the least squares prediction of Θ given u , $y_{opt}(u) = \mu_\Theta +$
796 $\rho \frac{\sigma_\Theta}{\sigma_U}(u - \mu_U)$, Appendix A. Some authors refer to U in this context as a “proxy cue” of environmental
797 component E . However, it is sufficient to only consider U and Θ as two correlated components of a
798 temporally varying environment. Blue line represents the optimal reaction norm under perfect
799 information ($\rho = 1$) (when the ellipses collapse to a line), and green line represents the optimal
800 reaction norm when U and Θ are uncorrelated ($\rho = 0$). Solid red line represents the optimal reaction
801 norm when $\rho = 0.5$ (corresponding to the drawn ellipses). Thick stippled red line is referred to in the
802 Analysis section. Note that in Lande’s (2009) notation, ε_t corresponds to a random value of E in
803 generation t , and $\varepsilon_{t-\tau}$ corresponds to a random U in the same generation.

804

805 Figure 2. Evolution of linear reaction norms after a sudden environmental change. Panels A-C:
806 Trajectories of the population mean trait values with a sudden environmental change at generation
807 5000 (see text). Panel D: Phase plane diagram showing \bar{z}_a plotted against \bar{z}_c through all generations
808 (this is the point in the cue-phenotype plane where reaction norms “tend to cross” (see Fig. 3), since
809 the phenotypic variance-covariance matrix here is diagonal (see eq. (5)). Solid blue lines represent the
810 three-trait model (3) and the stippled red lines represent the two-trait model (2). The trajectories were
811 calculated as the mean of 1000 independent simulations. Grey lines show the realization of a single
812 simulation. Solid green lines show μ_Θ (panel A), the optimal slope when reaction norm slope and

813 intercept can be tuned independently, $\sigma_{U\Theta}/\sigma_U^2$ (eq. (1)) (panel B), and μ_U (panel C). In panel A, the
814 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
815 the intercept trait in the two-trait model (stippled red line). In panel B, stippled green line shows the
816 mean slope that gives maximum expected logarithm of fitness of a random individual (Appendix C).
817 Parameter values in the initial environment were $\mu_U = 0$, $\mu_\Theta = 0$, $\sigma_U = 2$, $\sigma_\Theta = 4$, and $\rho = \frac{\sigma_{U\Theta}}{\sigma_U\sigma_\Theta} =$
818 0.25. At generation 5000, μ_U jumps to 6 and μ_Θ jumps to 12 while the other parameters remain
819 unchanged. Diagonal \mathbf{G} and \mathbf{P} matrices were used with $G_{aa} = 0.5$, $P_{aa} = G_{aa} + 0.5$, $P_{bb} = G_{bb} =$
820 0.045, and $P_{cc} = G_{cc} = 2$ (three-trait model) or $P_{cc} = G_{cc} = 0$ (two-trait model). Initial mean trait
821 values were $\bar{z}_a = 0$, $\bar{z}_b = \rho \frac{\sigma_\Theta}{\sigma_U} = 0.5$, and $\bar{z}_c = 0$.

822

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

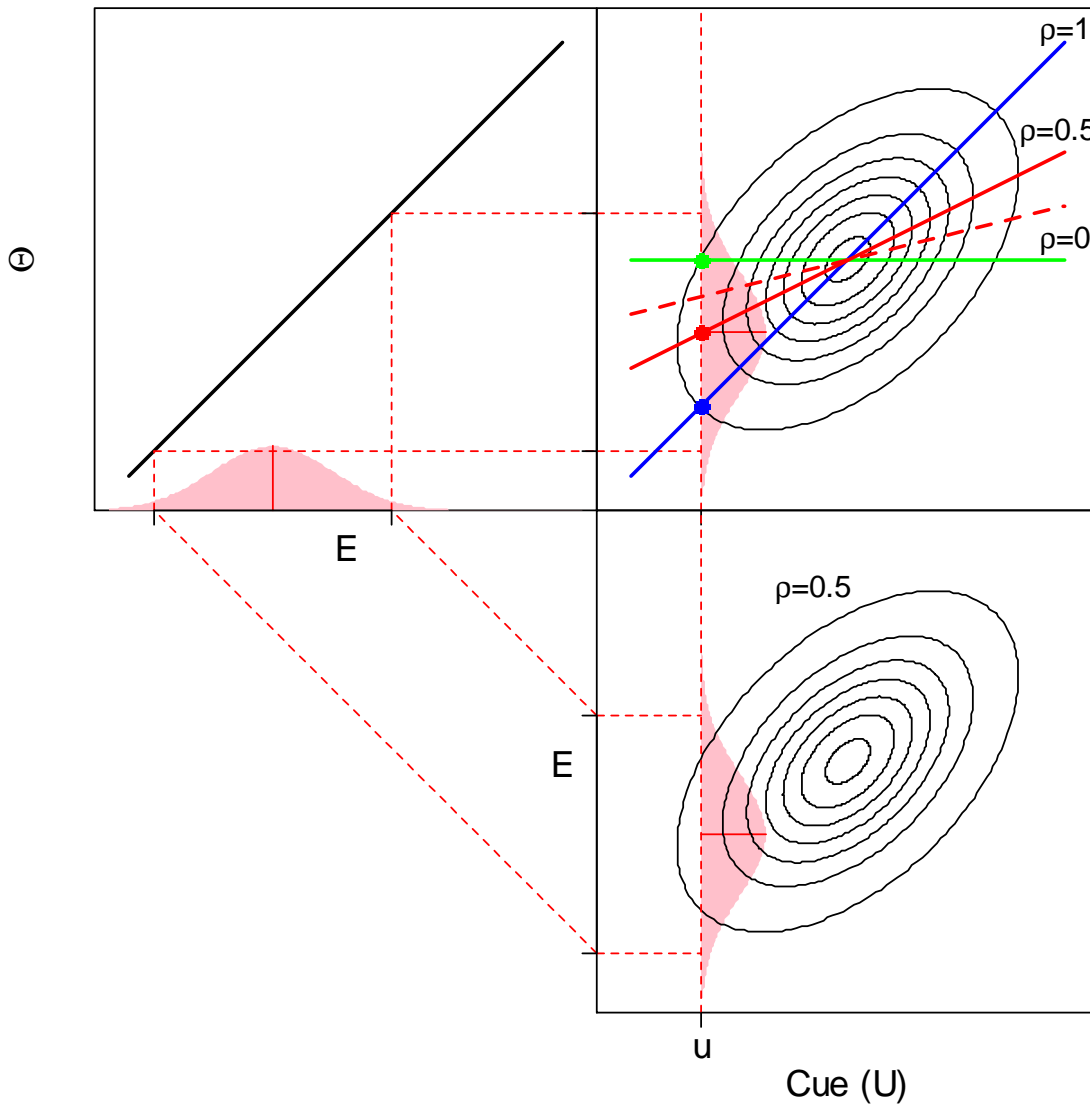
832

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

839

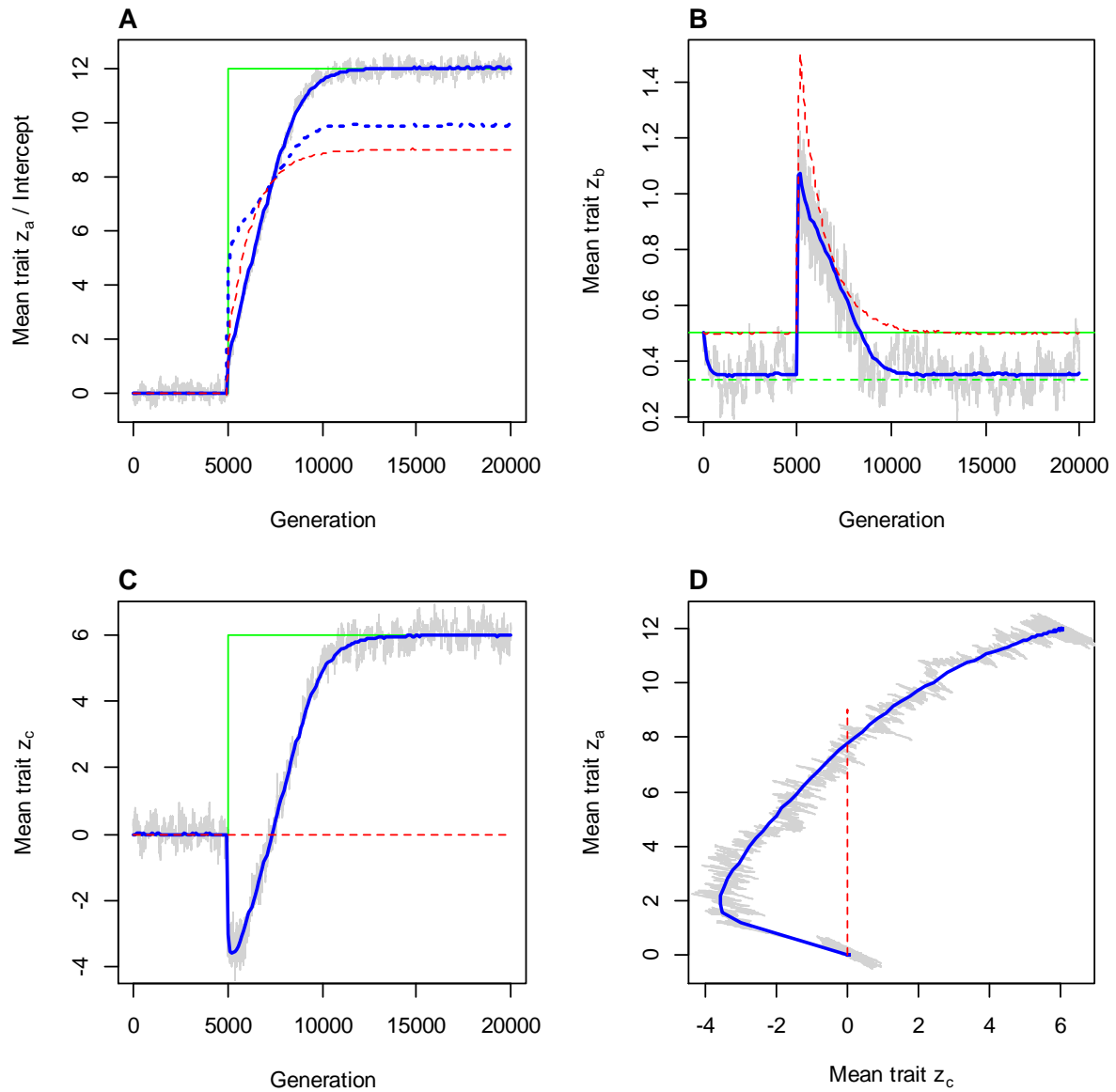
840 Figure 4. Fitness trajectories in the simulation example (Figure 2). Left panels: Population mean
841 fitness relative to maximum fitness (W_{max}). Right panels: Expected fitness at the mean trait values.
842 The lower panels show the same values plotted with a narrower range on the y-axis. Thick 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 20th generation is plotted in
845 the left panels and every 100th generation is plotted in the right panels. Plotted values are the mean of
846 the same 1000 independent simulations used for Figure 2.
847

848



849

850 **Figure 1**



851

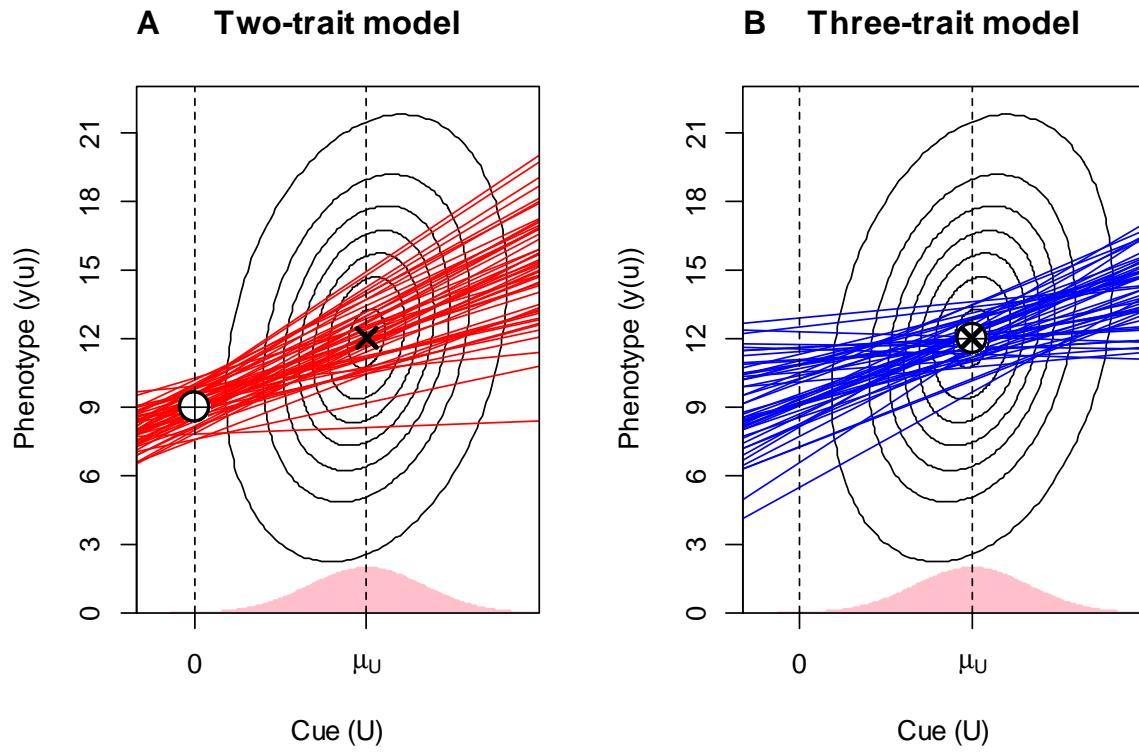
852 **Figure 2**

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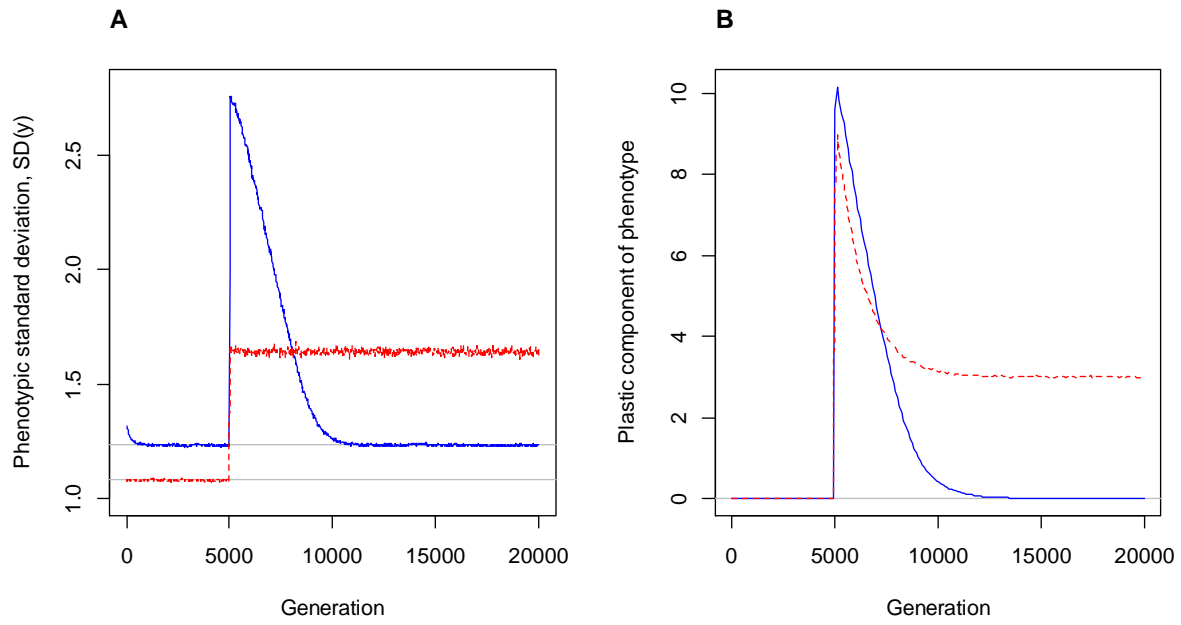
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859 **Figure 3**

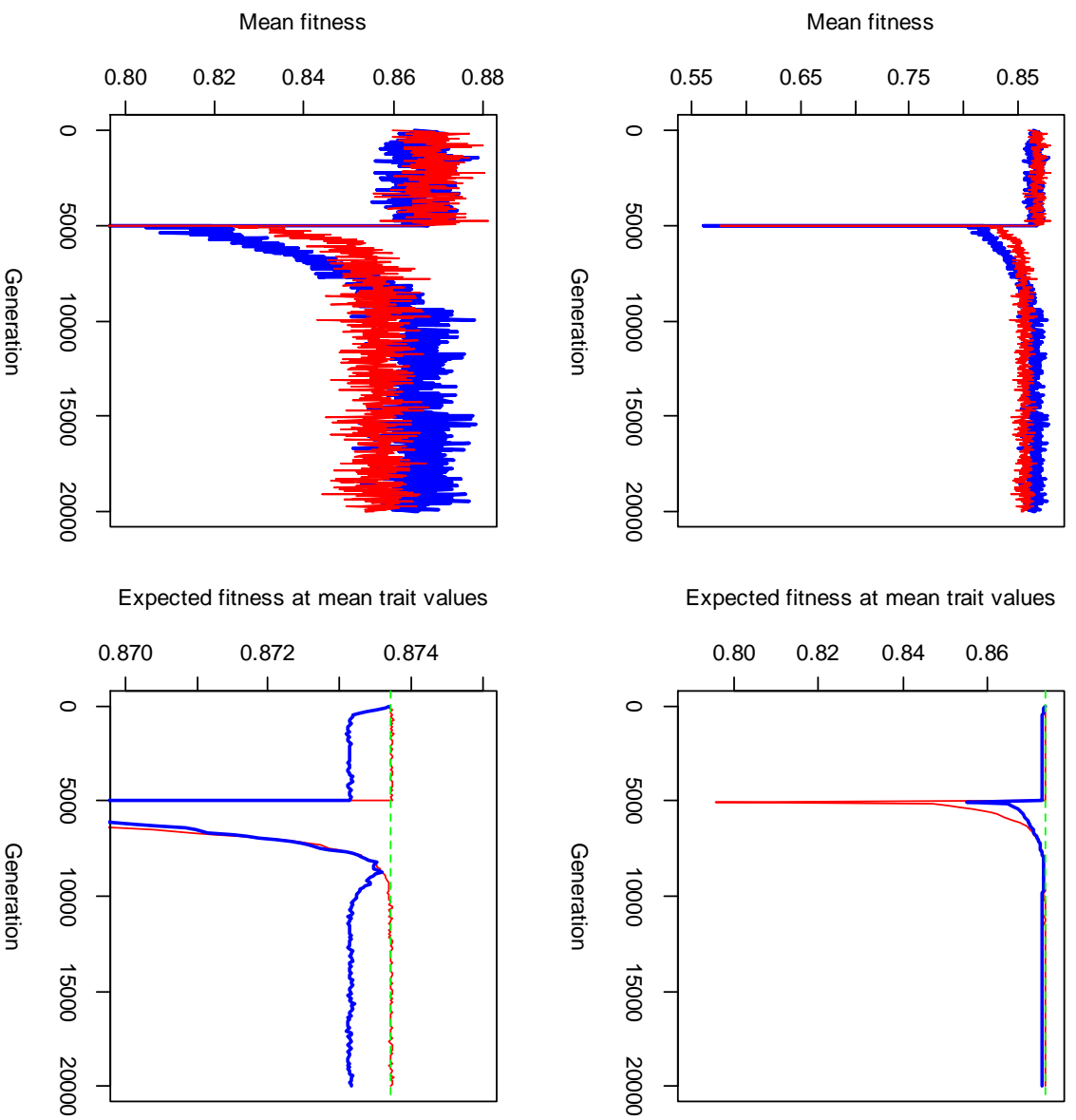
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861



862

863 **Figure 4**



864
865 **Figure 5**

866
867