

1 Modeling Adaptive and Non-adaptive Responses of Populations to
2 Environmental Change

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21 **Abstract**

22 Understanding how the natural world will be impacted by environmental change is one of the most
23 pressing challenges facing humanity. Addressing this challenge is difficult because environmental
24 change can generate both population level plastic and evolutionary responses, with plastic responses
25 being either adaptive or non-adaptive. We develop an approach that links mechanistic quantitative
26 genetic theory with data-driven structured models to allow prediction of population responses
27 to environmental change via plasticity and adaptive evolution. After introducing general new
28 theory, we construct a number of example models to demonstrate that evolutionary responses to
29 environmental change will be considerably slower than plastic responses, that adaptive plasticity
30 can accelerate population recovery to environmental change but that it slows the rate of adaptation
31 to the new environment. Parameterization of the models we develop requires information on genetic
32 and phenotypic variation and demography which will not always be available. We consequently
33 develop a method based on the statistical analysis of temporal trends in model parameter values
34 of examining whether the full machinery of the evolutionarily explicit models we develop will be
35 needed to predict responses to environmental change, or whether simpler non-evolutionary models
36 that are now widely constructed may be sufficient.

37 Introduction

38 Ecosystems from the deep ocean to the high arctic, from deserts to tropical forests are responding
39 to environmental change. Understanding and predicting these responses is one of the most pressing
40 issues currently facing humanity. For this reason, in the last quarter of a century, there has been
41 considerable interest in developing ways to understand how the natural world will be affected by
42 environmental change (Bossdorf et al., 2008; Dawson et al., 2011; Gilbert and Epel, 2009; Hoffmann
43 and Sgrò, 2011; Ives, 1995; Lavergne et al., 2010; Wiens et al., 2009). We introduce a new, general
44 approach combining insights from structured population modeling and evolutionary genetics that
45 allows us to examine how adaptive evolution and plasticity contribute to the way that populations,
46 and consequently the ecosystems in which they are embedded, respond to environmental change.

47 Environment change alters the expected demographic rates of individuals within a population
48 (Chevin et al., 2010). For example, if environmental change reduced the probability of survival
49 of all individuals within a population without impacting recruitment, then population size would
50 decline (Caswell, 2001). Predicting the way populations will respond to environmental change
51 consequently requires understanding how such change impacts demographic rates (Coulson et al.,
52 2001). Individual differences in expected demographic rates within a population are ubiquitous,
53 with some individuals having a greater propensity to survive or reproduce than others (Link et al.,
54 2002). This heterogeneity across individuals is determined by phenotypic variation (Wilson and
55 Nussey, 2010). For example, large individuals often have higher survival and recruitment rates
56 compared to their smaller counterparts (e.g. Festa-Bianchet et al., 1998; Sedinger et al., 1995). To
57 understand how environmental change influences demographic rates at the population level it is
58 consequently necessary to know (i) the distribution of phenotypes within the population and (ii)
59 their expected demographic rates in different environments (Ozgul et al., 2010).

60 Dynamic models of population responses to environmental change need to incorporate informa-
61 tion not only on the associations between phenotypic traits and expected survival and reproduction
62 in different environments, but also on the way that environmental variation influences phenotypic

63 development within individuals as they age, and the distribution of phenotypes among new born
64 individuals recruiting to the population (Rees et al., 2014). In other words we need to understand
65 the processes that determine individual phenotypic trajectories and resulting life histories. As well
66 as environmental variation, genes also influence the way that phenotypes develop within individu-
67 als (Cheverud et al., 1983), as can an individual’s current phenotypic state (Badyaev and Martin,
68 2000; Easterling et al., 2000). Parental phenotypes, parental genotypes and environmental variation
69 can all influence the distribution of offspring phenotypes as can mating patterns (Baldwin, 1896;
70 Charlesworth, 1994; Gavrillets and Scheiner, 1993; Lynch and Walsh, 1998; Monaghan, 2008). This
71 complexity makes predicting population responses to environmental change challenging.

72 Adaptive evolution in response to environmental change occurs when selection – the association
73 between phenotypes and expected survival and reproduction – results in a change in allele frequen-
74 cies. Such genetic change can lead to change in the distribution of the phenotypes that influence
75 survival and reproduction. However, phenotype distributions can respond to environmental change
76 in the absence of adaptive evolution via plasticity. The ability for phenotype distributions to change
77 in the absence of adaptive evolution is often genetically determined. Individuals can modify their
78 own phenotypes, or those of their offspring, by altering their physiology, metabolism or behavior
79 (Aubin-Horth and Renn, 2009; Richards, 2006). This is achieved by altering gene expression pat-
80 terns by up and down regulating expression of particular genes, or even turning some genes off
81 and others on (Snell-Rood et al., 2010). These effects that are not encoded in DNA are termed
82 epigenetic effects.

83 Epigenetic responses to environmental change occur at the level of the individual. For them
84 to leave a signature at the population level in the distribution of phenotypes, multiple individuals
85 need to exhibit similar epigenetic responses to environmental change (Lande, 2009). When this hap-
86 pens, populations are said to exhibit plastic responses. We distinguish between two types of plastic
87 response – phenotypic plasticity (Scheiner, 1993) and epigenetic inheritance (Richards, 2006). Phe-
88 notypic plasticity occurs when phenotype distributions change within surviving individuals due to
89 epigenetic responses to a changing environment. In contrast, epigenetic inheritance occurs when a

90 change in the environment impacts the phenotype of offspring recruiting to the population (Blake
91 and Watson, 2016). Epigenetic inheritance can be influenced by the environment the offspring find
92 themselves when they become independent, or by their parents. For example, parents may provi-
93 sion developing offspring (seeds or foetuses) with different resources or hormone levels as a function
94 of their own phenotypes (Love et al., 2005). We refer to this environment as the developmental
95 environment. Alternatively, once independent from their parents, offspring development may be
96 determined by the ecological environment they experience (Solberg et al., 2004). In germinating
97 seeds, the ecological environment could be determined by light, water and nutrient availability.

98 Any general framework that can be used to predict how environmental change will impact popu-
99 lations consequently needs to incorporate how plasticity and genetic variation generates phenotypic
100 variation, and how phenotypic variation impacts expected demography. We show how evolution-
101 arily explicit integral projection models (IPMs) (Barfield et al., 2011; Childs et al., 2016; Coulson
102 et al., 2011) provide a powerful framework within which to do this.

103 IPMs are a very flexible structured modeling tool. They project the dynamics of phenotype
104 distributions as a function of expected survival and reproduction, the way the phenotype develops
105 and the distribution of offspring phenotypes (Coulson, 2012; Easterling et al., 2000; Merow et al.,
106 2014). Because IPMs track the dynamics of the entire distribution of phenotypic traits, numerous
107 quantities of interest to ecologists and evolutionary biologists describing life history, population
108 dynamic and phenotypic traits can be calculated from them (Childs et al., 2003; Coulson et al.,
109 2011, 2010; Ellner and Rees, 2006; Rees et al., 2014; Steiner et al., 2014, 2012; Vindenes and
110 Langangen, 2015). They consequently offer great potential to study eco-evolutionary feedbacks and
111 dynamics (Coulson et al., 2011). However, most IPMs to date have been restricted to phenotypic
112 variation in that they do not include genotype-phenotype maps (Merow et al., 2014). A small
113 number of evolutionarily explicit IPMs have been developed. Coulson et al. (2011) used IPMs
114 to track the distribution of body size and coat color in wolves, where coat color was determined
115 by genotype at a single bi-allelic locus. They showed how environmental change would impact
116 genotype frequencies at this locus. Barfield et al. (2011) and Childs et al. (2016) developed IPMs

117 of quantitative characters determined by a large number of unlinked loci of small effect. However,
118 none of these models incorporates plasticity, nor different genetic influences on the phenotype at
119 different ages, and these omissions limit their utility in predicting how populations will be influenced
120 by environmental change (Chevin, 2015).

121 The aim of this paper is to introduce the general framework. We do this by (i) introducing
122 two sex IPMs of phenotypic traits (Schindler et al., 2015, 2013; Traill et al., 2014a) that are not
123 evolutionarily explicit, (ii) extending these models to include flexible genotype-phenotype maps that
124 allow the role of adaptive evolution and plastic responses to environmental change to be examined,
125 (iii) develop simple models to illustrate the framework. These models provide new results on the role
126 of plasticity on evolutionary trajectories yet also allow us to retrieve key insights from evolutionary
127 genetics.

128 **Methods and Results**

129 We start this section by introducing our general modelling approach. Our models consist of com-
130 binations of functions, so we start by focusing on the biological processes these functions capture,
131 and the way they combine to project the dynamics of phenotypic trait distributions. Our start-
132 ing point is a model of the entire phenotype that we then extend to capture the dynamics of a
133 phenotype consisting of genetic and environmentally determined components (Falconer, 1960). In
134 order to construct models within our approach it is necessary to select forms for each function so
135 we next turn our attention to this challenge. In the next sections we consider appropriate forms for
136 functions that describe the dynamics of first the genetic component of the phenotype and second
137 its environmental component. Next, we combine insights from these two sections to consider the
138 dynamics of phenotypes consisting of both a genetic and environmental component. Finally, we
139 consider how to identify circumstances when the full machinery of evolutionarily explicit IPMs are
140 required, and when purely phenotypic ones will likely suffice.

141 Modeling approach

142 We use the term mechanistic to refer to functional forms that are derived from a mechanistic un-
143 derstanding of a process. For example, Mendelian inheritance rules that are central to quantitative
144 and population genetics are mechanistic in that the distribution of offspring genotypes or breeding
145 values is known *a priori* from the parental genotypes or breeding values and the mating system
146 (Barfield et al., 2011; Charlesworth, 1994). The term phenomenological is used to refer to func-
147 tional forms that are identified from the statistical analysis of data (Crawley, 2007). We refer to
148 functions, be they mechanistic or phenomenological, as $f(\dots)$ where the dots inside parentheses
149 define the variables the function f operates on. Parameters of a function are referenced by the
150 same letter as the function, with subscripts defining the variable they influence. For example, a
151 parameter $f_{\mathcal{Z}}$ represents a parameter of function f that operates on variable \mathcal{Z} . We reserve I for
152 the intercept of functions and a for age. Age is only included in models for species with overlapping
153 generations. We use primes ($'$) to represent a possible change in trait value from one time step to
154 the next, either among surviving individuals, or between parents and their offspring. The notation
155 we use (Table 1) is the standard notation used for IPMs (Coulson, 2012; Merow et al., 2014; Rees
156 et al., 2014). We now turn to our approach.

157 Selection is the underpinning of adaptive evolution. It operates on the phenotype, and de-
158 pending upon the genotype-phenotype map, can result in some genotypes having greater fitness
159 than others. Under some circumstances such variation in genotype fitness can result in evolution
160 defined as a change in allele frequencies. However, in other circumstances, for example when phe-
161 notypes determined by heterozygote genotypes have greater fitness than phenotypes determined by
162 homozygote genotypes, variation in genotype fitness does not necessarily result in allele frequency
163 change (Charlesworth, 1994; Fisher, 1930).

164 In order to predict evolution and population dynamics it is necessary to understand: (i) the
165 genotype-phenotype map at birth, (ii) how the phenotype develops, (iii) how the phenotype influ-
166 ences survival at each developmental stage, (iv) the population's mating system and (v) patterns
167 of mate choice based on the phenotype, as well as how these mate choice patterns influence (vi)

168 reproductive success, (vii) the distribution of genotypes among offspring and (viii) how all these
 169 processes result in change in allele frequency from one generation to the next. Processes (i) to (vi)
 170 (and consequently also (viii)) can be influenced by environmental variation. Dispersal can also be
 171 an important driver of evolution. It can be added into the models we develop relatively easily, but
 172 is not considered further here.

173 Our starting point is a phenotypic modeling approach that captures all demographic processes
 174 that can contribute to the dynamics of phenotypes – survival, recruitment, development, inher-
 175 itance, and mating patterns. Two sex phenotypic IPMs (Coulson et al., 2011; Schindler et al.,
 176 2015, 2013; Traill et al., 2014a) capture processes (ii) to (vi) listed above but they do not include
 177 genotypes, or consequently a genotype-phenotype map. Instead they include a function that maps
 178 parental phenotype at time t to the phenotypes of recruiting offspring at time $t+1$ (Easterling et al.,
 179 2000). These functions are phenomenological in that no genetic mechanisms of inheritance are in-
 180 cluded (Coulson et al., 2010; Smallegange and Coulson, 2013). Having introduced these models we
 181 then extend them to include genotype-phenotype maps.

182 The model consists of two equations – one for females and one for males – with each equation
 183 consisting of two additive components (Schindler et al., 2013). The first component deals with
 184 survival and development of individuals already within the population, the second component deals
 185 with reproduction and the generation of phenotypes among newborns entering the population,

$$\begin{aligned}
 N_f(\mathcal{Z}', t+1) &= \int [D_f(\mathcal{Z}'|\mathcal{Z}, \theta, t)S_f(\mathcal{Z}, \theta, t)N_f(\mathcal{Z}, t)]d\mathcal{Z} + \\
 &+ sC_{N_f N_m} \iint [H_f(\mathcal{Z}'|\mathcal{Z}_m, \mathcal{Z}_f, \theta, t)M(\mathcal{Z}_m, \mathcal{Z}_f, t) \dots \\
 &\dots N_f(\mathcal{Z}_f, t)N_m(\mathcal{Z}_m, t)R(\mathcal{Z}_f, \mathcal{Z}_m, \theta, t)]d\mathcal{Z}_m d\mathcal{Z}_f \\
 N_m(\mathcal{Z}', t+1) &= \int [D_m(\mathcal{Z}'|\mathcal{Z}, \theta, t)S_m(\mathcal{Z}, \theta, t)N_m(\mathcal{Z}, t)]d\mathcal{Z} + \\
 &+ (1-s)C_{N_f N_m} \iint [H_m(\mathcal{Z}'|\mathcal{Z}_m, \mathcal{Z}_f, \theta, t)M(\mathcal{Z}_m, \mathcal{Z}_f, t) \dots \\
 &\dots N_f(\mathcal{Z}_f, t)N_m(\mathcal{Z}_m, t)R(\mathcal{Z}_f, \mathcal{Z}_m, \theta, t)]d\mathcal{Z}_m d\mathcal{Z}_f \tag{1}
 \end{aligned}$$

186 $N_f(\mathcal{Z}', t+1)$ and $N_m(\mathcal{Z}', t+1)$ are distributions of phenotypes \mathcal{Z}' in respectively females and males

187 at time $t+1$; $D_f(\mathcal{Z}'|\mathcal{Z}, \theta, t)$ and $D_m(\mathcal{Z}'|\mathcal{Z}, \theta, t)$ are the probability of the phenotype developing from
 188 \mathcal{Z} to \mathcal{Z}' in respectively females and males between t and $t+1$ as a function of environmental drivers
 189 θ ; $S_f(\mathcal{Z}, \theta, t)$ and $S_m(\mathcal{Z}, \theta, t)$ are survival functions for females and males from t to $t+1$ including
 190 effects of phenotype and environmental drivers θ ; s is the birth sex ratio measured as the proportion
 191 of female offspring produced; and $C_{N_f N_m}$ is a normalisation constant; $H_f(\mathcal{Z}'|\mathcal{Z}_m, \mathcal{Z}_f, \theta, t)$ and
 192 $H_m(\mathcal{Z}'|\mathcal{Z}_m, \mathcal{Z}_f, \theta, t)$ describe the probabilities of parents with phenotypes \mathcal{Z}_m and \mathcal{Z}_f respectively
 193 producing male and female offspring with phenotype \mathcal{Z}' as a function of environmental drivers θ at
 194 time t ; $M(\mathcal{Z}_m, \mathcal{Z}_f, t)$ captures the rate of mating between a male with phenotype \mathcal{Z}_m and a female
 195 with phenotype \mathcal{Z}_f ; $R(\mathcal{Z}_f, \mathcal{Z}_m, \theta, t)$ describes the expected litter size given a mating between a
 196 male and a female with phenotypes \mathcal{Z}_m and \mathcal{Z}_f in environment θ at time t . The survival, mating
 197 and litter size functions determine the strength of selection on \mathcal{Z} (Schindler et al., 2015).

$C_{N_f N_m}$ can be used to capture a range of mating systems. For example, if we follow Schindler
 et al. (2013) and write,

$$C_{N_f N_m} = \frac{\int_{\mathcal{Z}_{f(\min)}}^{\infty} N_f(\mathcal{Z}_f, t) d\mathcal{Z}_f}{\int_0^{\infty} M(\mathcal{Z}_m, \mathcal{Z}_f, t) N_m(\mathcal{Z}_m, t) N_f(\mathcal{Z}_f, t) d\mathcal{Z}_m d\mathcal{Z}_f} \quad (2)$$

198 this adds a minimum size at which females can reproduce $\mathcal{Z}_{f(\min)}$. Depending on the mating be-
 199 havior of the species, $C_{N_f N_m}$ can be modified in various ways. For example, it can easily be altered
 200 such that the number of birth events is determined by the number of the rarer sex, as in monog-
 201 amous species. Mate choice can be influenced by specifying different functions for $M(\mathcal{Z}_m, \mathcal{Z}_f, t)$.
 202 Schindler et al. (2013) demonstrate how it can be specified for random mating, assortative mating,
 203 disassortative mating and size-selective mating.

In phenotypic IPMs, the phenotypic development functions are usually Gaussian probability
 functions (Easterling et al., 2000), e.g.:

$$D(\mathcal{Z}'|\mathcal{Z}, \theta, t) = \frac{1}{V^D(\mathcal{Z}, \theta, t) \sqrt{2\pi}} e^{-\frac{(\mathcal{Z}' - \mu^D(\mathcal{Z}, \theta, t))^2}{2V^D(\mathcal{Z}, \theta, t)^2}}. \quad (3)$$

204 The functions $\mu^D(\mathcal{Z}, \theta, t)$ and $V^D(\mathcal{Z}, \theta, t)$ respectively describe the expected value of \mathcal{Z}' given \mathcal{Z}
 205 and θ at time t and the variance around $\mu^D(\mathcal{Z}, \theta, t)$. The Gaussian form can also be used for
 206 development functions $H(\mathcal{Z}'|\mathcal{Z}, \theta, t)$ with functions $\mu^H(\dots)$ and $V^H(\dots)$.

207 We extend the two sex phenotypic IPM in equation (1) to include genotypes by writing the
208 phenotype as a function $\mathcal{Z} = z(\mathcal{G}, \mathcal{E})$. We assume that \mathcal{Z} is a quantitative phenotype (i.e. measured
209 in integer or real values). The genotypic value \mathcal{G} and environmental value \mathcal{E} describe the numerical
210 contributions of the genetic and environmental components of the phenotype to an individual's
211 phenotypic trait value. A simple map can consequently be written $\mathcal{Z} = \mathcal{G} + \mathcal{E}$ (Falconer, 1960).

212 \mathcal{G} is determined by genotype, g . When the map between g and \mathcal{G} is additive, the dynamics of
213 g and \mathcal{G} are identical. In contrast, when alleles interact, either at a locus (dominance) or across
214 loci (epistasis) the map between g and \mathcal{G} is not additive, and the dynamics of \mathcal{G} are not identical
215 to the dynamics of g (Fisher, 1930). In classical quantitative genetics it is assumed that the map
216 between g and \mathcal{G} is additive (Falconer, 1960). Under these assumptions, it is not necessary to track
217 the dynamics of g but evolution can be investigated by modeling the dynamics of just \mathcal{G} . When
218 the map is additive we refer to the genetic component of the phenotype as a breeding value and
219 denote it \mathcal{A} .

220 In classical population genetics, when the contribution of dominance and epistasis to evolution
221 are often a key focus, it is necessary to track the dynamics of g and calculate \mathcal{G} from each g . The
222 map between \mathcal{G} and the phenotype \mathcal{Z} is often assumed to be one-to-one (Hartl et al., 1997). In other
223 words, the dynamics of \mathcal{G} and \mathcal{Z} are identical. In contrast, in quantitative genetics, the environment
224 can influence the map between \mathcal{A} and \mathcal{Z} by influencing the value of the environmental component
225 of the phenotype, \mathcal{E} (Falconer, 1960). \mathcal{E} can take different values in different individuals and can
226 vary within individuals throughout life. The dynamics of the phenotype may not consequently
227 represent the dynamics of the genotypic value \mathcal{A} . Statistical quantitative genetics is concerned
228 with estimating moments of \mathcal{A} from \mathcal{Z} by correcting for environmental and individual variables
229 that determine \mathcal{E} (Kruuk et al., 2008).

230 The genotype-phenotype map for phenotypic traits measured by biologists in free living pop-
231 ulations is rarely known, and quantitative genetic assumptions are widely adopted (Kruuk et al.,
232 2008). In particular, the infinitesimal model is assumed in which \mathcal{A} is determined by a large number
233 of unlinked loci of small, additive, effect (Fisher, 1930). Until we have a better understanding of the

234 genetic architecture of complex traits, this approach is the most powerful available to investigate
235 evolution in the wild (Kruuk et al., 2008). We consequently adopt it here.

236 We track the joint distribution of the two components $N(\mathcal{A}, \mathcal{E}, t)$. The utility of this is we
237 can write expressions to describe the dynamics of each of the components separately, if necessary,
238 before easily combining them to retrieve the dynamics of the phenotype. For $\mathcal{Z} = \mathcal{A} + \mathcal{E}$ we can
239 use a convolution (represented by the mathematical operator $*$) between the two components of
240 the phenotype to construct the phenotype (Barfield et al., 2011).

241 Phenotypic plasticity and epigenetic inheritance are captured in the dynamics of \mathcal{E} . In previous
242 quantitative genetic IPMs \mathcal{E} is a randomly distributed variable that captures developmental noise
243 (Barfield et al., 2011; Childs et al., 2016). A key contribution of this paper is to show how \mathcal{E} can be
244 extended to also capture the biotic or abiotic environment as well as signatures of parental \mathcal{A} s and
245 \mathcal{E} s. \mathcal{E} is consequently defined as function of these drivers. There are various notations we could use
246 to capture this. To be consistent with previous IPMs formulations (Coulson, 2012; Merow et al.,
247 2014; Rees et al., 2014) we write $\mathcal{E}'|\mathcal{E}, \mathcal{A}, \theta, t$ to capture the effects of \mathcal{E} , \mathcal{A} and the environment θ
248 at time t on \mathcal{E}' .

249 We now expand terms in our two-sex phenotypic IPM to include the genotype-phenotype map
250 $\mathcal{Z} = z(\mathcal{A}, \mathcal{E})$. We start with the bivariate distribution of \mathcal{A} and \mathcal{E} at time t among females that
251 are already within the population at time t : $N_f(\mathcal{A}, \mathcal{E}, t)$. Viability selection now operates on this
252 distribution. Viability selection is a simple multiplicative process describing the expected survival
253 from t to $t + 1$ as a function of the phenotype. We can consequently write,

$$N_f^s(\mathcal{A}, \mathcal{E}, t) = S_f(z(\mathcal{A}, \mathcal{E}), \theta, t)N_f(\mathcal{A}, \mathcal{E}, t). \quad (4)$$

When it comes to development, the genotype does not develop but remains fixed for life. However,
 \mathcal{A} can vary with age if different genes contribute to the phenotype at different ages (Wilson et al.,
2005). In the section §**Adaptive Evolution** we consider the dynamics of age-structured breeding
values. We focus here on the case where \mathcal{A} remains fixed for life but the environmental component

may vary,

$$N_f^s(\mathcal{A}, \mathcal{E}', t + 1) = \int D_f(\mathcal{E}' | (\mathcal{E}, \mathcal{A}, \theta), t) N_f^s(\mathcal{A}, \mathcal{E}, t) d\mathcal{E}. \quad (5)$$

254 Recruitment is dealt with in a similar way to survival in that it is a multiplicative process,

$$\begin{aligned} N^r((\mathcal{A}_m, \mathcal{E}_m), (\mathcal{A}_f, \mathcal{E}_f), t) &= M((\mathcal{A}_m, \mathcal{E}_m), (\mathcal{A}_f, \mathcal{E}_f), t) N(\mathcal{A}_m, \mathcal{E}_m, t) \dots \\ &\dots N(\mathcal{A}_f, \mathcal{E}_f, t) R(z(\mathcal{A}_m, \mathcal{E}_m), (z\mathcal{A}_f, \mathcal{E}_f), \theta, t). \end{aligned}$$

255 Note this is a recruitment related term of both male and female offspring that is not yet scaled by
256 the normalization factor $C_{N_f N_m}$.

257 As with development, inheritance of the genetic and environmental components of the phenotype
258 operates in different ways. For example, once mating pairs have formed and the number of offspring
259 from each mating has been determined, the distribution of offspring genotypes is predictable. We
260 can write the inheritance function for the genetic and environmental components of the phenotype
261 as,

$$\begin{aligned} N_f^r(\mathcal{A}', \mathcal{E}', t + 1) &= sC_{N_f N_m} \iiint H_f(\mathcal{A}' | (\mathcal{A}_m, \mathcal{A}_f), \mathcal{E}' | (\mathcal{E}_m, \mathcal{E}_f, \theta, t)) \dots \\ &\dots N^r((\mathcal{A}_m, \mathcal{E}_m), (\mathcal{A}_f, \mathcal{E}_f), t) d\mathcal{A}_m d\mathcal{E}_m d\mathcal{A}_f d\mathcal{E}_f \end{aligned} \quad (6)$$

then,

$$N_f(\mathcal{A}', \mathcal{E}', t + 1) = N_f^r(\mathcal{A}', \mathcal{E}', t + 1) + N_f^s(\mathcal{A}, \mathcal{E}', t + 1). \quad (7)$$

262 The same logic applies to the production of male offspring.

We can construct the phenotype from the two components \mathcal{A}' and \mathcal{E}' , e.g.

$$N_f(\mathcal{Z}', t + 1) = \int_{\Omega_{\mathcal{Z}'}} N_f^r(\mathcal{A}', \mathcal{E}', t + 1) d\mathcal{E}' d\mathcal{A}' + \int_{\Omega_{\mathcal{Z}'}} N_f^s(\mathcal{A}, \mathcal{E}', t + 1) d\mathcal{E}' \quad (8)$$

263 where $\Omega_{\mathcal{Z}'}$ is the set of $(\mathcal{A}', \mathcal{E}')$ values satisfying $z(\mathcal{A}', \mathcal{E}') = \mathcal{Z}'$. For the second integral in equation
264 (8) we have $z(\mathcal{A}, \mathcal{E}') = \mathcal{Z}'$ as the \mathcal{A} does not change within individuals and consequently has no
265 prime.

266 The additivity assumption means that models of clonal inheritance can generate very similar
267 predictions to models of two sexes, particularly if both males and females have similar demography.
268 However, clonal models are simpler than two sex models (Lande, 1982). We utilize this consequence
269 of the additivity assumption and initially work with clonal reproduction to examine how the dy-
270 namics of \mathcal{A} and \mathcal{E} influence population and phenotypic trait dynamics and adaptive evolution. We
271 can write a clonal model,

$$\begin{aligned} N(\mathcal{A}, \mathcal{E}', t + 1) &= \int [D(\mathcal{E}'|\mathcal{E}, \mathcal{A}, \theta, t)S(z(\mathcal{A}, \mathcal{E}), \theta, t) + H(\mathcal{E}'|\mathcal{E}, \mathcal{A}, \theta, t) \dots \\ &\dots R(z(\mathcal{A}, \mathcal{E}), \theta, t)]N(\mathcal{A}, \mathcal{E}, t)d\mathcal{E} \end{aligned} \quad (9)$$

and

$$N(\mathcal{Z}', t + 1) = \int_{\omega'_{\mathcal{Z}}} N(\mathcal{A}, \mathcal{E}', t + 1)d\mathcal{E}'. \quad (10)$$

272 **Functional Forms**

273 In order to construct models it is necessary to identify forms for each of the functions described in
274 the section above. These forms can differ for development and inheritance of \mathcal{A} and \mathcal{E} . To illustrate
275 this we construct models for two limits. At one limit, all phenotypic variation is attributable to
276 individual differences in \mathcal{A} . At the other limit, all individuals are genetically identical: they have
277 the same \mathcal{A} and all individual variation is attributable to \mathcal{E} . This captures plasticity defined as
278 the same genotype expressing different phenotypes in different environments. Having considered
279 functional forms for these two limits we combine insights to construct models for phenotypes that
280 are determined by \mathcal{A} and \mathcal{E} .

281 We primarily focus on linear functions for three reasons. First, they are easier to interpret and
282 analyze than non-linear or non-additive forms. Second, when the environment changes impacting
283 populations, responses, at least in the short term, can be well described with linear or linearized
284 additive models (Cooch et al., 2001). Third, selection, the underpinning of evolution, is often
285 directional and well described with linear or linearized associations between phenotypic traits and

286 components of fitness (Kingsolver et al., 2001). Parameters used for all models are provided in the
287 Supplementary Information (SI §1.1), as are expressions to calculate key statistics used to show
288 ecological and evolutionary change from model outputs (SI §1.2). Code to produce each figure is
289 available on GitHub – <https://github.com/tncoulson/QG-meets-IPM-figure-code/tree/master>.

290 The environmental drivers θ, t can be both abiotic and biotic. We focus primarily on a biotic
291 driver, population density.

292 **Adaptive Evolution**

293 In this section we start with a simple clonal model of a univariate distribution of \mathcal{A} . We go on to
294 show how genetic constraints can be imposed to slow, or stop, evolution. We then extend this clonal
295 model in two ways: first, to include a multivariate, age-structured, distribution of \mathcal{A} , and second
296 we relax the clonality assumption and compare the dynamics of clonal and sexual models. Finally,
297 we introduce a new approximation to describe sexual reproduction and compare its performance
298 with our initial approach.

299 Genotypes (and hence \mathcal{A}) are determined at birth and remain fixed throughout life; neither
300 are influenced by the environment. A consequence of this is the development function simplifies
301 to a one-to-one map and can be removed from equation (5). We also start by considering clonal
302 reproduction, which means that the inheritance function can also be removed as offspring genotype
303 is identical to parental genotype. The dynamics of \mathcal{A} are consequently determined by the survival
304 and reproduction functions – selection. In these models, as long as there is genetic variation within
305 a population, and fitness is a monotonic function of genotype, evolution, defined as $\mathbb{E}(N(\mathcal{A}, t+1)) =$
306 $\mathbb{E}(N^r(\mathcal{A}, t)) \neq \mathbb{E}(N(\mathcal{A}, t))$ (where \mathbb{E} represents expectations) will occur.

In our first models we assume non-overlapping generations,

$$N(\mathcal{A}, t+1) = N^r(\mathcal{A}, t) = R(\mathcal{A}, t)N(\mathcal{A}, t).$$

307 and a linear reproduction function $R(\mathcal{A}, t) = R_I + R_A\mathcal{A}$ with expected fitness increasing with the
308 value of \mathcal{A} . Over the course of a simulation of 30 generations (SI §1.1 Model A), the population

309 never achieves an equilibrium structure or growth rate; it grows hyper-exponentially (Figure 1(a),
310 black line) and the shape of the breeding value distribution continually changes location (Figure
311 3(b), black line) and shape (Figure 1(b,d, black lines)). Linear selection only slowly erodes the
312 genetic variance and skew (Figure 1(c,d)) and these changes lead to a slight slowing of the rate of
313 change in the mean breeding value (Figure 1(b)) and the population growth rate (Figure 1(a)) each
314 generation (the black lines are not linear).

315 In this model there are two ways to prevent the fitness function from generating change in the
316 location of the distribution. First, the fitness function can take unimodal non-linear forms such as
317 $R(\mathcal{A}, t) = R_I + R_{\mathcal{A}}\mathcal{A} + R_{\mathcal{A}^2}\mathcal{A}^2$ with $R_{\mathcal{A}^2} < 0$ and $R(\mathcal{A}, t)$ constrained to non-negative values. This
318 generates stabilizing selection, with the mean breeding value being maintained at the value that
319 maximizes fitness. Eventually, in this model, the breeding value distribution will achieve a trivial
320 equilibrium – a Dirac delta function at this value. Second, continual change in the location of the
321 distribution can be prevented by defining a maximum possible value for \mathcal{A} that cannot be exceeded.
322 This captures a genetic constraint in the maximum possible character value – i.e. evolution has
323 not evolved a genetic solution to creating a larger breeding value. In our models, this process can
324 be captured by setting the abundance of $N(\mathcal{A} > x, 1) = 0$ where x is the maximum possible trait
325 value that evolution can achieve. Selection now pushes the breeding value distribution up to x ,
326 again eventually achieving a trivial equilibrium captured by a Dirac delta function where all mass
327 of the distribution is at $\mathcal{A} = x$.

328 Genetic constraints can also impact the transient dynamics of the breeding value distribution
329 (Figure 1(a-d, red lines)). When we impose a genetic constraint (SI §1.1 model A with $x = 11.5$),
330 the genetic variance and skew evolve faster than when no genetic constraint is in place (Figure 1(c)
331 and (d)). These more rapid changes result in a slowing in the evolution of the mean breeding value
332 (Figure 1(b)), and of the population growth rate (Figure 1(a)).

333 Genetic covariances between traits can also capture genetic constraints and can also influence the
334 outcome of evolution. We demonstrate this by developing an age-structured model. \mathcal{A} now becomes
335 age-structured but is still inherited at birth. We construct a multivariate character \mathcal{A} describing the

336 breeding values that influence a character at each age (e.g. $\mathcal{A}1, \mathcal{A}2, \dots, \mathcal{A}n$ for breeding values at
 337 ages $a = 1, 2, \dots, n$). If some of the same loci contribute to the genetic components of the character
 338 at different ages there is a genetic covariation across ages. The genetic variances within each age,
 339 and the covariances between ages, can be used to construct a \mathbf{G} matrix (Lande, 1979). Such age-
 340 structured \mathbf{G} matrices underpin the character-state approach of quantitative genetics (Lynch and
 341 Walsh, 1998). In the age-structured model that follows, we define a bivariate normal distribution
 342 with a known variance-covariance structure as our starting point and iterate this forwards (SI §1.1
 343 models B-D). We consider a simple case: a monocarpic biennial life cycle where individuals in their
 344 first year of life do not reproduce and all age 2 individuals die after reproduction. As with our
 345 model for a species with non-overlapping generations we assume clonal inheritance,

$$\begin{aligned} N(\mathcal{A}1, 1, t + 1) &= R(\mathcal{A}2, 2, t)N(\mathcal{A}2, 2, t) \\ N(\mathcal{A}2, 2, t + 1) &= S(\mathcal{A}1, 1, t)N(\mathcal{A}1, 1, t), \end{aligned} \quad (11)$$

where survival from age 1 to age 2 is specified as

$$S(\mathcal{A}1, 1, t) = \frac{1}{1 + e^{-(S_{I,1} + S_{\mathcal{A}1,1}\mathcal{A}1)}} \quad (12)$$

346 with expected survival to age 2 being highest for larger values of $\mathcal{A}1$. Although $\mathcal{A}2$ is not under
 347 direct selection, its distribution is modified by its covariance with $\mathcal{A}1$.

$\mathcal{A}2$, the genotype at age 2, determines expected reproduction,

$$R(\mathcal{A}2, 2, t) = e^{(R_{I,2} + R_{\mathcal{A}2}\mathcal{A}2)}. \quad (13)$$

348 Although $\mathcal{A}1$ does not directly influence reproduction, there is an association between it and repro-
 349 duction via its covariance with $\mathcal{A}2$. All age 2 individuals die following reproduction in this model,
 350 although it is possible to extend our approach to any arbitrary number of ages.

351 The evolutionary dynamics that particular parameterizations of the fitness functions $S(\mathcal{A}1, 1, t)$
 352 and $R(\mathcal{A}2, 2, t)$ generate are dependent upon (i) the initial covariance between the characters and
 353 (ii) the fitness functions (SI §1.1 models B-D). Many parameterizations and initial covariances are

354 likely to generate evolutionary dynamics that may be biologically unrealistic. We demonstrate this
355 with three contrasting parameterizations, considering size as our trait (Figure 1(e)-(g)). In the first
356 example, (Figure 1(e) SI §1.1 model B), the two characters positively covary and experience selection
357 in the same direction. Over the course of the simulation the average developmental trajectory has
358 evolved with $\mathcal{A}1$ evolving to be 1.76 times larger and $\mathcal{A}2$ evolving to be 1.52 times larger. For a
359 trait like body size, such a proportional change at different ages may be appropriate. In examples
360 (Figure 1(f and g), SI §1.1 models C and D) the bivariate character evolves in contrasting ways. In
361 (F), $\mathcal{A}2$ evolves much faster than $\mathcal{A}1$ while in (G) $\mathcal{A}1$ evolves to be larger, while $\mathcal{A}2$ evolves to be
362 smaller. These simulations demonstrate that only a constrained set of fitness functions and genetic
363 covariances will give biologically realistic evolutionary trajectories for the size-related traits that
364 biologists often study.

365 We now return to a univariate model and examine the clonality assumption. How can the
366 clonality assumption be relaxed, and what are the consequences? In sexually reproducing species,
367 offspring inherit a mix of their parent's genomes. However, genetic segregation means that full
368 siblings do not have the same genotype. When additivity is assumed, the breeding value of offspring
369 is expected to be midway between parental breeding values. However, to obtain the distribution
370 of offspring genotypes, the contribution of genetic segregation to variation among offspring needs
371 to be taken into account. In two sex models, three steps are required to generate the distribution
372 of offspring genotypes or breeding values given parental values. First, a distribution of mating
373 pairs needs to be constructed. Second, the distribution of midpoint parental genotypes or breeding
374 values given the distribution of mating pairs needs to be constructed. Third, segregation variance
375 needs to be added to the distribution (Feldman and Cavalli-Sforza, 1979; Felsenstein, 1981; Turelli
376 and Barton, 1994). The mating system and the segregation variance are related: when mating is
377 assortative with respect to genotype, the segregation variance is small and siblings closely resemble
378 one another and their parents. In contrast, when mating is disassortative with respect to genotype,
379 siblings can differ markedly from one another, and the segregation variance is large.

Expressions have been derived for the segregation variance for the infinitesimal model where

it is assumed that traits are determined by a very large number of unlinked loci of small additive effects and mating is random (Fisher, 1930). The infinitesimal model is assumed in most empirical quantitative genetic analyses (Kruuk et al., 2008) and in our initial model. For random mating where both sexes have identical demographies, the distribution of offspring breeding values given parental breeding values is (Barfield et al., 2011):

$$N(\mathcal{A}, t + 1) = \left(\frac{N^r(\cdot, t)}{2} * \frac{N^r(\cdot, t)}{2} * \phi \left(\cdot, \frac{\sigma_r^2(\mathcal{A}, t)}{2} \right) \right) (\mathcal{A}), \quad (14)$$

380 where $*$ represents convolution and $\phi(\mathcal{A}, \sigma^2) = \frac{1}{\sqrt{2\pi\sigma^2}} \exp \left[-\frac{\mathcal{A}^2}{\sigma^2} \right]$ is a Gaussian function with mean
381 zero and variance σ^2 representing the segregation variance.

If males and females have different demographies then they will have different distributions of genetic values after selection; we represent these as $N_M^r(\mathcal{A}, t)$ and $N_F^r(\mathcal{A}, t)$, respectively. In this case, eq. (14) is replaced by

$$N(\mathcal{A}, t + 1) = \left(\frac{N_M^r(\cdot, t)}{2} * \frac{N_F^r(\cdot, t)}{2} * \phi \left(\cdot, \frac{\sigma_{r(M)}^2(\mathcal{A}, t) + \sigma_{r(F)}^2(\mathcal{A}, t)}{2} \right) \right) (\mathcal{A}), \quad (15)$$

382 where $\sigma_{r(M)}^2(\mathcal{A}, t)$ and $\sigma_{r(F)}^2(\mathcal{A}, t)$ are variances of the post-recruitment-selection genetic value of
383 males and females, respectively. We do not superscript the r s with σ^2 to avoid a notation making
384 it appear σ is raised to some quantity $2r$.

385 The first two terms on the right hand side of equation (15) generates the distribution of ex-
386 pected parental midpoint values; it ensures that the mean breeding value among offspring is midway
387 between the two parental breeding values. However, because the parental distributions are halved,
388 the variance of this distribution is half that of the parental distributions. The third term on the
389 right hand side of equation (15) adds the segregation variance. For random mating, the variance
390 is assumed to be normally distributed with a mean of 0 and a variance of half the additive genetic
391 variance among the entire population when the population is at linkage equilibrium (Felsenstein,
392 1981). We approximate this variance as half the additive genetic variance in the parental distribu-
393 tion (Feldman and Cavalli-Sforza, 1979). This approach has already been incorporated into IPMs
394 (Barfield et al., 2011; Childs et al., 2016).

395 We now run two simulations (Figure 2(a)-(d)) to examine differences in the predictions of clonal
396 and sexual models. The first model assumes clonal inheritance and the second the convolution in
397 Equation (15), with both models assuming a linear function $R(\mathcal{Z}, t)$ (SI §1.1 model E). The two
398 models predict slightly divergent dynamics. The reason for this is that equation (15) results in the
399 skew and kurtosis in $N_R(\mathcal{A}, t)$ is reduced at each time step in the sexual model compared to in the
400 clonal model. If selection is exponential (and the starting distribution proportional to a Gaussian
401 distribution) then there will be no difference between the two approaches. This is because a normal
402 distribution multiplied by an exponential fitness function results in a normal distribution with an
403 unchanged variance (Diaconis et al., 1979). These results suggest that insights from clonal models
404 will approximate those from sexual models reasonably well, at least when males and females have
405 similar demography.

406 Some authors have queried the use of Equation (3) as an approximation in IPMs to the inheri-
407 tance convolution in Equation (15) used in models of sexually reproducing species (Chevin et al.,
408 2010; Janeiro et al., in press). However, being able to construct inheritance functions for \mathcal{A} that
409 are of the form of equation (3) would be useful as it would permit methods developed for two sex
410 phenotypic IPMs to be applied to evolutionarily explicit IPMs (e.g. Schindler et al., 2015). Given
411 Gaussian approximations frequently perform well in models of evolution (Turelli and Barton, 1994)
412 we hypothesize that Gaussian inheritance functions may perform well in evolutionarily explicit
413 IPMs. We consequently constructed a Gaussian inheritance function and compared results with
414 those obtained from the convolution.

415 Equation (15) results in the mean and variance of the parental and offspring breeding value
416 being the same. We can approximate this by ensuring that the function $\mu^H(\mathcal{A}, t)$ passes through
417 the coordinate $x = \mathbb{E}(N_R(\mathcal{A}, t)), y = \mathbb{E}(N_R(\mathcal{A}, t))$ and that the variance $V^H(\mathcal{A}, t) = \sigma^2(N_R(\mathcal{A}, t))$.
418 When both sexes have the same demography, we can write,

$$\begin{aligned}\mu^H(\mathcal{A}, t) &= (1 - \eta)\mathbb{E}_R(N_R(\mathcal{A}, t)) + \eta\mathcal{A} \\ V^H(\mathcal{A}, t) &= (1 - \eta)^2\sigma^2(N_R(\mathcal{A}, t))\end{aligned}\tag{16}$$

419 where \mathbb{E} and σ^2 represent expectations and variances respectively and η represents the degree of
420 assortative mating. When $\eta = 1$ mating is entirely assortative, when $\theta = 0.5$ mating is random
421 and when $\eta = 0$ mating is completely disassortative. An equation for the case when males and
422 females have different demographics is provided in the SI §1.3. The approximation in Equation
423 (16) will increase in accuracy as the distribution of mid-point parental breeding values becomes
424 more Gaussian.

425 When we compared predictions from equations (15) and (16) with $\eta = 0.5$ using the same model
426 used to compare clonal and sexual life histories, results were indistinguishable (Figure 2(a)-(d)). This
427 reveals that, for linear selection, Gaussian inheritance functions for \mathcal{A} perform remarkably well.

428 Our approximation can be used to examine the dynamical contributions of non-additive genetic
429 processes to population responses to environmental change in a phenomenological manner. Fisher
430 (1930) demonstrated that dominance variance can be treated as an offset, and in our models this
431 would lower the intercept of the function $\mu^H(\mathcal{G}, t)$ in equation (16). A consequence of this is that
432 the mean of the offspring genotype is no longer equal to the mean of parental genotype and the
433 dynamics of genotypes no longer exactly match the dynamics of alleles. We demonstrate this
434 with a single locus-two allele model. When the effects of alleles are additive, the dynamics of the
435 genotype captures the dynamics of alleles (Figure 2(e)). In contrast, when the heterozygote has
436 higher fitness, allele frequencies do not change once the equilibrium is achieved. However, selection
437 and inheritance alter genotype frequencies (Figure 2(f)). This effect of dominance variance can be
438 phenomenologically capturing within an IPM by setting the intercept of the inheritance function
439 for the genetic component of the phenotype to be less than $\frac{\mathbb{E}_R(N_{R,\mathcal{A}}, t)}{2}$ – this imposes an offset that
440 can reverse gains made by selection (Figure 2(g)). Because this offset is negative when dominance
441 variance is operating, dominance variance will slow, or prevent, rates of evolutionary change. We
442 could easily phenomenologically explore how a particular value of this offset impacts predicted
443 dynamics, however, further work is required to relate different levels of dominance variance to
444 specific values of the offset in our models.

445 Having shown how IPMs can be formulated to project forwards the dynamics of the genetic

446 component of the phenotype under a wide range of circumstances, we now turn our attention to
447 the dynamics of the environmental component of the phenotype.

448 **Plasticity**

449 Plasticity is determined by the dynamics of \mathcal{E} and in particular in how \mathcal{E} is influenced by the
450 ecological environment θ . For this, we require a probability density function. We show in this
451 section how different forms of plasticity can be incorporated into evolutionarily explicit IPMs, and
452 explore the dynamics of some simple cases.

453 To capture plasticity in IPMs we need to model the probability of transition from \mathcal{E} at time
454 t to \mathcal{E}' at time $t + 1$ as a function of the environment θ . For most plastic traits we have a poor
455 mechanistic understanding of development and inheritance patterns, and for that reason we use
456 the Gaussian probability density function in Equation (3).

457 In quantitative genetics it is often assumed that the mean of $\mathbb{E}(\mathcal{E}, t) = 0$ and any individual
458 departures are purely random (Falconer, 1960). In equation 3 this requires the intercepts and slopes
459 of the functions $\mu^D(\dots)$ and $\mu^H(\dots)$ to take the following values: $\mu_I^H = 0$, $\mu_I^D = 0$, $\mu_{\mathcal{E}}^H = 1$ and
460 $\mu_{\mathcal{E}}^D = 1$. We relax this assumption and allow the mean (and variance) of \mathcal{E} to vary with time as θ
461 varies by specifying particular forms for development and inheritance functions of \mathcal{E} .

462 Gaussian transition functions (equation 3) can be formulated to predictably modify moments
463 of the distribution of \mathcal{E} from time t to time $t + 1$. For example, careful choice of intercepts and
464 slopes of $\mu^D \mathcal{E}, t$, $\mu^H \mathcal{E}, t$, $V^D \mathcal{E}, t$ and $V^H \mathcal{E}, t$ can be used to predictably grow, or shrink, the variance
465 of \mathcal{E} via either development or inheritance (SI §1.4). In addition, specific biological processes can
466 be easily incorporated into the dynamics of \mathcal{E} : if the slopes $\mu_{\mathcal{E}}^D \neq 0$ or $\mu_{\mathcal{E}}^H \neq 0$ then there will
467 be temporal autocorrelation in the value of \mathcal{E} among individuals, and between parents and their
468 offspring. For example, if $\mu_{\mathcal{E}}^D > 0$ then individuals with a relatively large value of \mathcal{E} at time t
469 will be expected to have a relatively large value of \mathcal{E}' at time $t + 1$. This property of development
470 functions is useful as it allows some memory of \mathcal{E} across ages: if an individual has benefited from a
471 particularly good set of circumstances at one age, any phenotypic consequences can persist to older

472 ages. In a similar vein, if $\mu_{\mathcal{E}}^H > 0$ then a parent with a relatively large \mathcal{E} at time t will produce
473 offspring with relatively large \mathcal{E} 's at time $t + 1$, a form of parental environmental effect (Nussey
474 et al., 2007).

475 Deterministic IPMs incorporate probabilistic transitions when $V^H(\mathcal{E}'|\mathcal{E}, \mathcal{A}, t) = 0$ and $V^D(\mathcal{E}'|\mathcal{E}, \mathcal{A}, t) =$
476 0. These probabilities do not vary from one time step to the next. In stochastic models these func-
477 tions can include terms for an environmental driver θ , such that the variation in trajectories changes
478 with the environment. In evolutionarily explicit models, the variance in transition rates among dif-
479 ferent values of \mathcal{E} can be made to depend upon θ , \mathcal{A} and their interaction (if desired). This means
480 that individuals with specific values of \mathcal{A} can produce offspring with more variable values of \mathcal{E} (and
481 consequently \mathcal{Z}) in particular environments than individuals with other values of \mathcal{A} . This is an
482 example of bet-hedging (Childs et al., 2010). We do not provide examples of bet-hedging in this
483 paper, but instead focus on the incorporation of θ into $\mu^H(\mathcal{E}'|\mathcal{E}, \mathcal{A}, \theta, t)$ and $\mu^D(\mathcal{E}'|\mathcal{E}, \mathcal{A}, \theta, t)$.

484 Different formulations of $\mu^H(\dots)$ and $\mu^D(\dots)$ can be used to capture a variety of different
485 forms of plasticity (Table 2). When θ is incorporated as an additive effect, it acts to shift the
486 intercept of these functions as t changes. This means that the environment influences all values
487 of \mathcal{A} in the same manner. If $\mathcal{Z} = \mathcal{A} + \mathcal{E}$ then \mathcal{Z} changes as a function of how θ influences \mathcal{E} if \mathcal{A}
488 remains constant. \mathcal{A} remains constant when it does not vary within individuals as they age, or if
489 \mathcal{A}' in offspring is the same as \mathcal{A} in parents.

490 Interactions between \mathcal{E} , \mathcal{A} and θ are listed in Table 2. Each form describes a different type of
491 reaction norm (Gavrilets and Scheiner, 1993). These forms allow \mathcal{E} to develop among individuals
492 (phenotypic plasticity) or be inherited (epigenetic inheritance) as a function of an individual's
493 breeding value \mathcal{A} and the environment θ as well as the value of \mathcal{E} at time t .

494 Plasticity can be either adaptive or non-adaptive (Ghalambor et al., 2015), and both forms
495 can be captured into our models. Adaptive plasticity enables populations to rapidly respond to an
496 environmental change. For example, if environmental change reduces population size, then adaptive
497 plasticity would result in a change to the mean of the phenotype via either phenotypic plasticity
498 (the development function) or epigenetic inheritance (the inheritance function) that leads to an

499 increase in survival or recruitment rates. In contrast, non-adaptive plasticity does the opposite,
 500 potentially exacerbating the detrimental effects of environmental change.

501 We demonstrate this with an example of a simple IPM of a species with non-overlapping gen-
 502 erations: $N(\mathcal{E}', t + 1) = \int H(\mathcal{E}'|\mathcal{E}, \theta, t)R(\mathcal{E}, t)N(\mathcal{E}, t)d\mathcal{E}$. Because plasticity is defined as different
 503 breeding values \mathcal{A} or genotypes expressing a different phenotype \mathcal{Z} in different environments, our
 504 models assume all individuals have the same \mathcal{A} but that \mathcal{E} , and consequently \mathcal{Z} , is a function of the
 505 environment θ . This means we can remove \mathcal{A} from the model. We assume a linear fitness function
 506 and a Gaussian inheritance function,

$$\begin{aligned} R(\mathcal{E}, t) &= R_I + R_{\mathcal{E}}\mathcal{E} + R_{\theta}\theta \\ \mu^H(\mathcal{E}, t) &= \mu_I^H + \mu_{\mathcal{E}}^H\mathcal{E} + \mu_{\theta}^H\theta \\ V^H(\mathcal{E}, t) &= V_I^H \end{aligned}$$

507 Next, we assume that the phenotypic trait is positively associated with expected recruitment such
 508 that $R_{\mathcal{E}} > 0$. We also assume that the environmental driver is positively associated with expected
 509 recruitment such that as θ increases in value, fitness increases ($R_{\theta} > 0$). This means that the
 510 population growth rate (in a density-independent model) or population size (in a density-dependent
 511 model) also increases with θ . Now assume that a negative environmental perturbation decreases
 512 θ such that fitness decreases. For adaptive plasticity to counter this, the effect of the decrease in
 513 θ on epigenetic inheritance must increase the expected value of \mathcal{E} . In our simple model, this can
 514 only occur if $\mu_{\theta}^H < 0$. Then, as θ declines, $\mu_{\theta}^H\theta$ becomes less, and the value of $\mu_I^H + \mu_{\theta}^H\theta$ becomes
 515 larger, increasing the mean of \mathcal{E} and fitness. In general, in additive linear models like this, if $R_{\mathcal{E}}$
 516 and μ_{θ}^H take opposing signs then plasticity will be adaptive.

517 We develop three density-dependent models of a phenotype in a species with non-overlapping
 518 generations. In all models we define the fitness function to be $R(\mathcal{E}, t) = R_I + R_{\mathcal{E}}\mathcal{E} + R_{n(t)}n(t)$ where
 519 $n(t) = \int N(\mathcal{E}, t)d\mathcal{E}$ and where $R_{n(t)} < 0$. In each model we define $\mu^H(\mathcal{E}, t) = \mu_I^H + \mu_{\mathcal{E}}^H\mathcal{E} + \mu_{n(t)}^Hn(t)$.
 520 We set in model (F) $\mu_{n(t)}^H = 0$; in model (G) $\mu_{n(t)}^H < 0$; and in model (H) $\mu_{n(t)}^H > 0$ (SI §1.1).

521 The first model (F) does not include plasticity ($\mu_{n(t)}^H = 0$), the second (G) captures adaptive

522 plasticity ($\mu_{n(t)}^H < 0$ and $R_{\mathcal{E}} > 0$), and the third (H) captures non-adaptive plasticity ($\mu_{n(t)}^H >$
523 0 and $R_{\mathcal{E}} > 0$). Because the models are not age-structured and do not include development,
524 plasticity operates via epigenetic inheritance (e.g. maternal environmental effects). The same
525 logic can be extended to the development function in age-structured populations. In our examples,
526 parameterizations are chosen so all models converge to the same value of carrying capacity, K . Once
527 all three models have converged, we initially impose a one off perturbation. Model (G) regains the
528 equilibrium first, followed by model (F), and then model (H) (Figure 3(a)) showing that adaptive
529 plasticity allows the population to recover from a one off environmental perturbation much faster
530 than when there is no plasticity, or plasticity is non-adaptive. Non-adaptivity plasticity significantly
531 slows the rate at which the population can recover from a perturbation, with the initial population
532 size pre-perturbation only re-attained after 80 generations.

533 Adaptive and non-adaptive plasticity also impact the way populations respond to permanent
534 environmental change. We demonstrate this by running the same models (F), (G) and (H), except
535 now we impose a constant change in fitness by permanently changing the intercept of the fitness
536 function R_I . When we do this, the three models attain different equilibria population sizes (Figure
537 3(b)) and different mean phenotypes (Figure 3(c)). Model (G) achieves a larger population size
538 than the two other models. This buffering of the population against environmental change happens
539 because adaptive phenotypic plasticity results in a change in the mean phenotype (Figure 2(c)) that
540 increases the expected recruitment rate and asymptotic population size (Figure 2(b)). In contrast,
541 non-adaptive plasticity exacerbates the consequences via a change in the mean phenotype that
542 decreases fitness.

In contrast to our example models in the §**Adaptive Evolution**, the IPMs we have developed in this section, and indeed all non-genetic IPMs so far published, achieve an asymptotic population growth rate or equilibrium population size and a stable population structure. These IPMs have monotonically increasing or decreasing fitness functions: an increase in the character results in an increase in expected fitness. A consequence of this is that in these models the recruitment function acts to alter the location of the character distribution, and often also alter its shape (Wallace et al.,

2013). In other words, $N_R(\mathcal{E}, t) - N(\mathcal{E}, t) \neq 0$. In models of species with non-overlapping generations at equilibrium like those above, the inheritance function for \mathcal{E} must exactly reverse the changes to the character distribution generated by the fitness function. This means, for deterministic models, that

$$N_R(\mathcal{E}, t) - N(\mathcal{E}, t) = N(\mathcal{E}', t + 1) - N_R(\mathcal{E}, t). \quad (17)$$

543 This equality requires moments of parental and offspring characters to differ from one another if
544 $N_R(\mathcal{E}, t) - N(\mathcal{E}, t) \neq 0$. When there is a correlation between parental and offspring traits in the
545 inheritance function for \mathcal{E} as in our models, the intercept of the inheritance function must take a
546 value such that offspring characters are smaller than their parent's were at the same age (Coulson
547 and Tuljapurkar, 2008).

548 IPMs for species with overlapping generations include development functions $D(\mathcal{E}'|\mathcal{E}, a, t)$.
549 These functions can alter the size and distribution of the character distribution as individuals age.
550 When generations are overlapping, and at equilibrium, changes to the location of the character
551 distribution via survival, recruitment and development are all exactly countered by the inheritance
552 functions $H(\mathcal{X}'|\mathcal{X}, a, t)$.

553 Coulson and Tuljapurkar (2008) showed that in red deer age-specific effects meant that young
554 and old parents were incapable of producing offspring that had the same body weight as they did
555 at birth. This mechanism reversed the effects of viability selection removing small individuals from
556 the population early in life. The same process was observed in marmots (Ozgul et al., 2010) and
557 Soay sheep (Ozgul et al., 2009) and may be general for body size in mammals.

558 We have now developed IPMs for (i) \mathcal{A} where we assumed all individuals had the same, constant,
559 \mathcal{E} and (ii) \mathcal{E} where we assumed all individuals had the same, constant, \mathcal{A} . We have shown how IPMs
560 can capture a wide range of biological processes including adaptive and non-adaptive plasticity and
561 correlated characters, and the circumstances when equilibria are achieved. We now link together
562 these advances into models of the joint dynamics of the bivariate distribution $N(\mathcal{A}, \mathcal{E}, t)$.

563 **Models for the phenotype consisting of genetic and environmental components**

564 In the section we construct models where the character can be determined by a mixture of the
565 genetic and environmental components. These models allow us to explore how adaptive evolution
566 is influenced by plasticity.

567 We first develop a dynamic univariate version of the Breeders equation (Falconer, 1960) for a
568 species with non-overlapping generations in a constant environment. In this case, the environmental
569 component of the phenotype is assumed to be a consequence of developmental noise: individuals
570 achieve their genetic potential, plus or minus a departure. At each generation within each breeding
571 value, the distribution of the environmental component of the phenotype is assumed to be Gaussian
572 with a mean of 0 and a constant variance (SI §1.1 Model I).

573 Our initial conditions are a bivariate Gaussian distribution of \mathcal{A} and \mathcal{E} which we iterate forwards
574 for 300 time steps. Over time, the mean of the genetic component of the phenotype increases. In
575 contrast, the mean of the environmental component is constant. The population grows hyper-
576 exponentially (Figure 4(a)), the mean of the phenotype increases in value due to evolution (Figure
577 4(a,d)) and the additive genetic variance is slowly eroded (Figure S2). Because the additive genetic
578 variance is eroded, while the phenotypic variance remains constant, the heritability declines over
579 time (Figure S2).

580 Our second model (SI §1.1 model J) has a negative density-dependent term in the fitness
581 function. The phenotype evolves faster in this model than in our density-independent model (Figure
582 4(b)). Population size grows nearly linearly in this model (Figure 4(d)), although the rate of increase
583 does slow slightly each generation as genetic variation is eroded. The difference between the hyper-
584 exponential (density-independent model) and nearly linear increases (density-dependent model)
585 in population size explain the difference in the rates of evolution. This is because the selection
586 differential that determines the rate of evolution (an emergent property from our model (Wallace
587 et al., 2013)) has the population growth rate in its denominator. The population growth rate is
588 smaller in the density-dependent model (just above unity) than in our density-independent one
589 (it increases with time), and this leads to an increase in the strength of selection and the rate of

590 evolution (see also Pelletier and Coulson, 2012). A consequence of this is that the additive genetic
591 variation and heritability tend towards zero faster than in the density-dependent model than in the
592 density-independent one (Figure S2).

593 In our third model (SI §1.1 model K), negative density-dependence is included in the inheritance
594 function for the environmental component of the phenotype as well as in the fitness function. This
595 captures adaptive phenotypic plasticity. This results in a negative change in the mean of the
596 environmental component of the phenotype with time (Figure 4(c)). This decrease is reflected in
597 a change in the mean of the phenotype itself. Adaptive phenotypic plasticity leads to a decline in
598 the population growth rate which results in a slight increase in the rate of evolution compared to
599 the density-dependent model with no plasticity. However, the effect is not large and is only just
600 distinguishable when comparing Figures 4(b) and (c).

601 In our final models (SI §1.1 models L to N) we examine how a one off perturbation influences
602 the mean of the phenotype, its components and the population growth rate (Figure 4(g)-(l)) when
603 there is no plasticity, adaptive plasticity and non-adaptive plasticity. We set the variance in the
604 genetic and environmental component of the phenotype to be equal, giving an initial heritability of
605 $h^2 = 0.5$. In each model we allow the population to achieve the same equilibrium population size in
606 the absence of selection ($R_Z = 0$). We then impose a one off mortality event when 99% of individuals
607 above the mean of the phenotype are killed off. At this point we also impose selection ($R_Z = 0.1$). In
608 all three models the mortality event results in a small change in the mean value of the phenotype
609 (SI §1.5 for an explanation) (Figure 4(g)-(i), red lines) but a halving of population size (Figure
610 4(j)-(l)). Adaptive plasticity results in the environmental component of the phenotype returning
611 to its pre-perturbation value very quickly (Figure 4(g)-(i) blue lines). In contrast, although the
612 perturbation causes a modest change in the mean of the genetic component of the phenotype,
613 it takes > 10 generations for evolution to reverse the change (Figure 4(g)-(i), black lines). This
614 demonstrates that a strong selective effect can leave a large population dynamic impact, but leave
615 only a small initial signature in the phenotype even when the trait is highly heritable.

616 Over the longer term, the dynamics of the all components of the phenotype, the phenotype

617 itself and the population dynamics all depend upon whether plasticity is adaptive or non-adaptive.
618 Adaptive plasticity allows the population size to initially recover from the perturbation more quickly
619 than when plasticity is absent or non-adaptive (Figure 4(j)-(l)). However, over a longer time
620 period, non-adaptive plasticity results in the population achieving a larger size than when plasticity
621 is absent or adaptive. These differences in population growth rate impact rates of evolution:
622 immediately following the perturbation, the rate of evolution is greatest when plasticity is non-
623 adaptive. However, the rate of evolution then increases when plasticity is adaptive (Figures S2 and
624 S3). As with our previous models, the effects of adaptive and non-adaptive plasticity on rates of
625 evolution are relatively small, but our results demonstrate how the two processes can interact.

626 **Signatures of evolution in phenomenological descriptions of mechanistic pro-** 627 **cesses**

628 The models in the previous section are quite complex. Do we always need to construct such
629 evolutionarily explicit IPMs to predict population responses to environmental change, or can we
630 rely on simpler, phenotypic IPMs? There are two reasons why it may be preferable to not construct
631 evolutionarily explicit models. First, evolutionarily explicit IPMs are more complicated to construct
632 than those that do not include genotypes or breeding values. Second, when data are unavailable
633 to explicitly include breeding values into models (Traill et al., 2014*b*), the effects of evolution on
634 predicted dynamics can still be explored by examining the consequences of perturbing parameter
635 values (Traill et al., 2014*a*).

636 When evolution occurs within a system we would expect parameters in phenomenological in-
637 heritance and development functions that are fitted to data to change with time. We can see this
638 in Figure 1(e)-(g)). In these age-structured evolutionarily explicit models, the bivariate breeding
639 value distribution (black contours) changes location as evolution occurs. We have fitted Gaussian
640 development functions to these bivariate distributions at the beginning of each simulation and
641 at the end (coloured image plots). The parameters that determine these development functions
642 have clearly changed as the location of the functions have changed. A similar process occurs for

643 inheritance functions (not shown).

644 Numerous authors have previously noted this phenomenon in models of evolution. For exam-
645 ple, in population genetic (Charlesworth, 1994) and eco-evolutionary models (Coulson et al., 2011;
646 Yoshida et al., 2003) when genotype frequencies change with time, macroscopic, population level
647 quantities like mean survival and recruitment also change; in adaptive dynamic models, as one
648 strategy invades another, population level parameters inevitably change with strategy frequency
649 over time (Metz et al., 1996); in quantitative genetic predator-prey models population level param-
650 eters of both predators and prey vary over time leading to persistence of the interaction (Doebeli,
651 1997); and in evolutionarily explicit IPMs parameters in inheritance functions have been shown
652 to change with time as evolution progresses (Rees and Ellner, 2016). These insights are useful
653 because if evolution is occurring within a system, then temporal trends in statistical estimates of
654 model parameters would be expected – in other words, the effect of time, either additively or in
655 an interaction with other parameters, would be expected in $\mu^H(\mathcal{Z}, t)$, $\mu^H(\mathcal{Z}, a, t)$ or $\mu^D(\mathcal{Z}, t)$. If
656 marked temporal trends are observed in parameters in development and inheritance functions that
657 cannot be attributed to a changing environmental driver, then evolutionarily explicit IPMs may be
658 required.

659 What about parameters in fitness functions $S(\mathcal{Z}, t)$ and $R(\mathcal{Z}, t)$? Can any inferences from
660 temporal trends in these parameters be made? In our approach, evolution of a focal trait would
661 not be expected to alter statistical estimates of the fitness functions. In our models, evolution
662 simply moves the location and shape of the phenotype distribution, but not its association with
663 survival or recruitment.

664 We have identified one circumstance where evolution will leave a signature in the dynamics of
665 fitness function parameters. Parameters in these functions can evolve in the presence of a genetically
666 unmeasured correlated character that is also evolving. To demonstrate this we construct a model
667 of a bivariate character, examine the dynamics it predicts, before exploring the consequences of
668 failing to measure one of the characters.

We assume clonal inheritance such that dynamics of the characters are solely determined by a

bivariate fitness function,

$$R(\mathcal{A}, t) = R_I - R_{\mathcal{A}1}\mathcal{A}1 + R_{\mathcal{A}2}\mathcal{A}2 \quad (18)$$

669 The dynamics this model predicts depend upon the initial covariance between the two characters
670 in a similar way to our age-structured model (equation 11). In our first example the two characters
671 negatively covary, while in the second they positively covary (SI §1.1 for model parameterizations).
672 The initial negative covariation allows rapid evolution, with population growth (Figure 5(a)), the
673 mean of the characters (Figure 5(b)), their variances (Figure 5(c)) and the covariance between
674 them (Figure 5(d)) evolving relatively quickly. In contrast, when the two characters positively
675 covary, evolution is much slower, with the character means, variances and covariance changing
676 much more slowly, even though the fitness functions are identical in each model (Figure 5(e)-(h)).

We now construct a fitness function for $\mathcal{A}1$ when $\mathcal{A}2$ is not measured. We start by defining mean fitness, an observable, as $\mathbb{E}(R, t) = \mathbb{E}(R(\mathcal{A}, t))$. The slope $\hat{R}_{\mathcal{A}1, t}$ is given by,

$$\hat{R}_{\mathcal{A}1, t} = R_{\mathcal{A}1} + \frac{\sigma(\mathcal{A}1, \mathcal{A}2, t)}{\sigma^2(\mathcal{A}1, t)} R_{\mathcal{A}2}. \quad (19)$$

The intercept can be calculated in the usual manner by estimating the means of fitness and $\mathcal{A}1$

$$\hat{R}_{I, t} = \mathbb{E}(R, t) - \hat{R}_{\mathcal{A}1, t} \mathbb{E}(\mathcal{A}1, t), \quad (20)$$

giving,

$$R(\mathcal{A}, t) = \hat{R}_{I, t} + \hat{R}_{\mathcal{A}1, t} \mathcal{A}1. \quad (21)$$

677 Equation (21) is what would be estimated from data if $\mathcal{A}2$ were not measured and included in
678 analyses (Kendall, 2015; Söderström and Stoica, 2002). It will correctly describe the consequences
679 of selection on $\mathcal{A}1$ even though $\mathcal{A}2$ could be correlated with it. This is because the unmeasured
680 correlated character impacts fitness whether it is measured or not, and consequently impacts the
681 association between the focal character and fitness in its absence (Lande and Arnold, 1983). How-
682 ever, the fitness function cannot provide accurate predictions over multiple generations when it is
683 assumed that the fitness function is constant.

684 Over multiple generations the existence of unmeasured correlated characters will alter parame-
685 ters in the fitness function in Equation (21) if selection alters genetic variances and covariances of
686 measured and unmeasured correlated characters (Figure 5(i)-(j)). This is because $\hat{R}_{I,t}$ and $\hat{R}_{A1,t}$
687 are both functions of the covariance between the two characters (equations 19-21). If selection
688 alters this covariance, parameters $\hat{R}_{I,t}$ and $\hat{R}_{A1,t}$ will evolve with time. It is also why we use the
689 subscript t for $\hat{R}_{I,t}$ and $\hat{R}_{A1,t}$. Evidence of correlated characters under selection can consequently
690 be inferred if parameters in fitness functions are observed to change with time in a system in the
691 absence of a changing environmental driver. Note that a non-stationary unmeasured environmen-
692 tal driver could also generate trends in parameter values in fitness functions in phenomenological
693 IPMs.

694 Discussion

695 In this paper we develop an approach that allows prediction of how populations respond to envi-
696 ronmental change via adaptive evolution and plasticity. We do this by incorporating mechanistic
697 insights from evolutionary genetics into data-driven structured population models. Our approach is
698 to split the phenotype into its genetic and environmental components and to model the dynamics
699 of the genetic component with functions based on mechanistic understanding. In contrast, the
700 dynamics of the environmental component of the phenotype, where mechanistic insight is lacking,
701 are modeled with phenomenological functions that can be identified from the analysis of data.
702 Our approach is appropriate for sexually reproducing or clonal species with either overlapping or
703 non-overlapping generations.

704 Evolutionarily explicit structured models

705 IPMs are now a widely used tool in ecology and evolution because of their versatility and the ease
706 with which they can be parameterized (Merow et al., 2014). All key statistics routinely estimated
707 in population ecology, quantitative genetics, population genetics and life history describe some

708 aspect of a character distribution or its dynamics (Coulson et al., 2010). IPMs are so versatile
709 because they describe the dynamics of these distributions. Characterization of the determinants
710 of these statistics gained via sensitivity or elasticity analysis of models have provided insight into
711 how ecological and evolutionary quantities that interest biologists are linked (Coulson et al., 2011).
712 Although this logic was developed several years ago, there has recently been criticism that IPMs
713 cannot be used to track the dynamics of multivariate breeding values expressed at different ages
714 (Chevin, 2015; Janeiro et al., in press). Our paper addresses this criticism head-on—we show how
715 IPMs can be formulated to capture such mechanistic complexity. In demonstrating this we develop
716 a general modeling approach to capture population responses to environmental change. Having
717 done this, we are now in a position to construct IPMs of quantitative characters and examine how
718 perturbing the environment will influence not only the dynamics of the phenotype and its genetic
719 and environmental components, but also the life history (Steiner et al., 2014, 2012) and population
720 dynamics (Easterling et al., 2000).

721 The work we present here adds to a growing literature that explicitly incorporates evolution into
722 structured models, and IPMs in particular. Within the population genetics paradigm, Charlesworth
723 (1994) developed structured models with a one-to-one map between genotype and phenotype in
724 age-structured populations. Building on this work, Coulson et al. (2011) showed how simple genetic
725 architectures can be incorporated into IPMs, developing a model to explore how evolution at a single
726 locus would occur simultaneously with phenotypic change of discrete and continuous characters,
727 life history and population dynamics.

728 Working in the quantitative genetic paradigm, Lande (1982) derived age-structured models
729 that tracked the dynamics of the mean of the additive genetic component of the phenotype ($\mathbb{E}(\mathcal{A})$
730 in our notation) and the mean of the phenotype itself ($\mathbb{E}(\mathcal{Z})$). He assumed a constant genetic-
731 variance covariance matrix and consequently weak selection and normally distributed character
732 values—assumptions we relax. Barfield et al. (2011) extended Lande (1982)’s approach to track
733 the dynamics of the entire character distribution and to stage-structured populations. In doing so,
734 they developed a general, flexible approach to track the entire distributions of \mathcal{A} and \mathcal{Z} . Childs

735 et al. (2016) extended this approach to two sexes. Because \mathcal{A} is inherited with mechanistic rules
736 that are not impacted by the environment, while inheritance and development of \mathcal{E} are plastic and
737 can be impacted by the ecological environment (Falconer, 1960), it is difficult to incorporate the
738 effects of the environment on the dynamics of the phenotype by focusing on \mathcal{A} and \mathcal{Z} as Lande
739 (1982), Barfield et al. (2011) and Childs et al. (2016) have done. In contrast, our approach (which
740 otherwise has a similar logic to Barfield et al. (2011) and Childs et al. (2016)) tracks the dynamics of
741 \mathcal{E} and \mathcal{A} (or \mathcal{G} —the full genotypic value, including non-additive components—if desired), making
742 incorporation of environmental drivers that influence inheritance and development of $[\mathcal{E}]$ more
743 straightforward. We show that it is possible to have selection operating on the phenotype while
744 incorporating mechanistic insights into the dynamics of the genetic component of the phenotype
745 and phenomenological insight into the role of the ecological environment on the dynamics of the
746 environmental component of the phenotype. By doing this, we show how population responses to
747 environmental change via adaptive evolution, phenotypic plasticity and epigenetic inheritance can
748 be simultaneously explored. This opens up the way to provide novel insights into the circumstances
749 when each process is expected to contribute to population responses to environmental change.

750 **Population responses to environmental change**

751 Unlike previous evolutionarily explicit IPMs (Barfield et al., 2011; Childs et al., 2016; Rees and
752 Ellner, 2016), our approach requires explicit consideration of the inheritance and development of
753 \mathcal{E} , the environmental component of the phenotype. This allows our models to capture a range of
754 plastic responses to environmental change along with adaptive ones. What do our findings say
755 about the contributions of plasticity, evolution, and their interaction to population responses to
756 environmental change?

757 Detrimental environmental change often causes a decline in population size. When there is an
758 association between a phenotypic trait and survival and recruitment rates, phenotypic change can
759 lead to increased survival and recruitment rates (Ozgul et al., 2010) and consequently an increase
760 in population growth rate and size. Two processes can lead to phenotypic change – plasticity

761 and adaptive evolution. There has been considerable discussion about the relative roles of each in
762 allowing populations to respond to change (e.g. Bonduriansky et al., 2012; Chevin et al., 2010).

763 Genotypes and breeding values remain fixed within individuals throughout life which means
764 that differential survival and recruitment rates are the processes that alter these distributions and
765 underpin evolution. The strength of differential survival and recruitment can be impacted by envi-
766 ronmental variation generating fluctuating selection (Lande, 2007). Environmental variation does
767 not influence genetic inheritance: once mating pairs are formed, inheritance of breeding values, \mathcal{A} ,
768 does not alter the mean or variance of breeding value distributions (Fisher, 1930). In contrast,
769 distributions of the environmental component of the phenotype can be altered via survival, re-
770 cruitment, development and inheritance with each process potentially impacted by environmental
771 variation (Reed et al., 2010). Given these differences between the dynamics of \mathcal{A} and \mathcal{E} plasticity
772 can lead to more rapid change than evolution in our models (e.g. Figure 4). This is because more
773 biological processes can directly alter the distribution of plastic characters than can impact dis-
774 tributions of breeding values. These results are consistent with those of other authors, including
775 Lande (2009) and Chevin et al. (2010), who also concluded that plastic change should be faster
776 than evolutionary change. But how quickly will evolution alter phenotypic trait distributions?

777 Our results on the speed of evolution suggest that claims of detectable rapid evolution in
778 quantitative phenotypes is likely to take a few tens of generations. For example, environmental
779 change increases mortality leading to a decline in population size, but for mortality selection to lead
780 to evolutionary change over the course of a generation, a large proportion of the population needs
781 to be selectively removed and the phenotype needs to be highly heritable. This is seen in our model
782 results (Figure 4(g)-(i)) and with a simple numerical example: when all individuals above the mean
783 of a normally distributed character are removed from the population and the trait has a heritable
784 of $h^2 = 0.5$, population size halves in a single time step but the mean of the character will only shift
785 from the 50th percentile to the 37.5th percentile. For a standard normal distribution with a mean
786 of 0 and a standard deviation of unity, this means the mean would only shift by 0.319 – i.e. less
787 than $\frac{1}{3}$ rd of a standard deviation – i.e. a long way from statistical significance. In reality, mortality

788 selection resulting from environmental change will likely result in a change to the mean of the
789 distribution that is only a fraction of a standard deviation compared to our example. Given this,
790 reports of rapid evolution due to environmental change increasing mortality selection over a small
791 number of generations (e.g. Coltman et al., 2003) should be treated with extreme caution. It is
792 much more likely that change is a consequence of phenotypic plasticity. Over multiple generations,
793 recruitment selection can also contribute to evolutionary change and our approach allows the role
794 of this to be investigated. However, unless reproduction is restricted to individuals with extreme
795 phenotypic trait values in both sexes, it seems unlikely that evolution can generate statistically
796 demonstrable evolutionary change over a small number of generations (Coulson et al., in revision).
797 This is not to say that evolution is not important over longer time scales. Over tens of generations
798 evolution can shift phenotypic trait means to a greater extent than phenotypic plasticity (Figure
799 4(g)-(i) blue versus black lines).

800 In order for plasticity to allow populations to rapidly respond to environmental change, a large
801 proportion of individuals within the population must exhibit the same plastic response. A good
802 example of such a dynamic is for size-related traits that are determined by resource availability,
803 particularly when scramble competition is operating. When resources becoming limiting, all indi-
804 viduals will be unable to develop as rapidly as when resources are more common. A consequence
805 of this is that individuals that developed in cohorts when resource were sparse will exhibit smaller
806 body sizes compared to individuals in those cohorts that developed when resources were more
807 abundant. We can capture this form of plasticity in our framework with an additive effect of den-
808 sity in the inheritance or development function for \mathcal{E} (e.g. Figure 3). In contrast, when contest
809 competition operates, larger individuals would acquire more resources than those that are smaller,
810 and would develop faster. We can capture this in our models with interactions between density, \mathcal{E}
811 and \mathcal{A} in either the inheritance or development functions for \mathcal{E} .

812 The above discussion demonstrates how our approach can be used to capture different forms of
813 plasticity. However, for plasticity to help populations respond to environmental change it must be
814 adaptive: plasticity must change the mean trait value in a way that increases fitness (Ghalambor

815 et al., 2007). We demonstrate that for additive, linear models, adaptive and non-adaptive plasticity
816 can be specified by altering the sign for the effect of the environment in the function specifying
817 the mean dynamics of the inheritance or development functions (Figure 3). When interactions are
818 included in these functions specifying general rules for whether plasticity is adaptive or non-adaptive
819 will likely be more challenging. However, our approach provides a way in which to investigate when
820 plasticity is adaptive or non-adaptive, and how different types of plasticity will influence population
821 responses to environmental change.

822 Our results also show how plasticity can influence evolutionary rates. Plasticity, operating via
823 development and inheritance functions for the environmental component of the phenotype, alters
824 the distribution of the phenotype, and this can alter the strength of selection, which can then
825 influence the dynamics of the genetic component of the phenotype (evolution). The effects of plas-
826 ticity on selection and evolution can be surprisingly complex. We only examined the evolutionary
827 consequences of plasticity following an environmental shock that influenced all individuals in the
828 same way, but even in this simple case we found that adaptive plasticity initially slowed the rate
829 of evolution compared to non-adaptive plasticity, before increasing it (Figure 5 and SI). In general
830 in order to understand how plasticity will influence selection, it is necessary to understand how it
831 influences both the numerator and denominator of the selection differential that underpins evolu-
832 tion (Pelletier and Coulson, 2012). The numerator is the covariance between the phenotype and
833 absolute fitness (Falconer, 1960) and the denominator is mean fitness. In our models of species with
834 non-overlapping generations this is mean recruitment – the population growth rate (Fisher, 1930).
835 Selection is linear in our models where plasticity influences all individuals in the same way via an
836 additive effect of density on inheritance of the environmental component of the phenotype (figure
837 5), and this means that plasticity influences the population growth rate rather than the numerator
838 of the selection differential. A consequence of this is that it is differences in the population growth
839 rate that generates the differences in evolutionary rates between models when plasticity is adaptive
840 and non-adaptive. In more complex cases when plasticity influences the covariance between the
841 phenotype and fitness via genotype-phenotype interactions within a generation, to understand how

842 selection influences evolution it is necessary to understand how plasticity not only influences mean
843 fitness, but also how it generates differences between the covariance between the genetic component
844 of the phenotype and fitness and the covariance between the phenotype itself and fitness. Because
845 the components of the selection differential can be calculated from IPMs (Coulson et al., 2010;
846 Wallace et al., 2013) the approach we develop here provides a flexible way to examine how different
847 types of plasticity can influence evolution following environmental change. But in order to explore
848 such dynamics in real systems it will be necessary to parameterize our models for real systems.

849 **Parameterizing and analyzing evolutionarily explicit IPMs**

850 A large literature exists on how to statistically parameterize IPMs (Easterling et al., 2000; Merow
851 et al., 2014; Rees et al., 2014). The vast majority of IPMs have been constructed phenomenologi-
852 cally, using statistical descriptions of observational data. Several authors have shown how fixed and
853 random effects incorporated into these statistical functions can be formulated within IPMs (Childs
854 et al., 2003; Coulson, 2012; Rees and Ellner, 2009), but additional statistical estimation is required
855 to parameterize the evolutionarily explicit IPMs we have developed.

856 Fitness functions in evolutionarily explicit IPMs can be parameterized using standard general,
857 generalized and additive regression methods that are routinely used to parameterize phenomeno-
858 logical IPMs (Rees and Ellner, 2009). If relatedness information is available and the infinitesimal
859 model is assumed, genetic and phenotypic variances and covariances can be estimated using the
860 animal model (Lynch and Walsh, 1998). These quantities can be used to construct the initial dis-
861 tributions of the genetic and environmental components of the phenotype. Parameter estimates of
862 ecological drivers fitted as fixed or random effects in the animal model can be used to parameterize
863 inheritance and development functions for the environmental component of the phenotype. It is
864 consequently possible to parameterize models using our approach with existing methods.

865 There is also a large literature on how to analyze IPMs (Ellner and Rees, 2006; Steiner et al.,
866 2014, 2012). The majority of these tools, including sensitivity and elasticity analysis of model
867 predictions to transition rates and function parameters (Coulson et al., 2011, 2010; Ellner and Rees,

2006; Steiner et al., 2014, 2012), are likely sufficiently general to be applicable to evolutionarily explicit IPMs. In future work we plan to parameterize models for bird, mammal and fish species with overlapping generations and to analyze them with existing methods. Once evolutionarily explicit IPMs have been parameterized and analyzed we will be able to explore how populations, phenotypic characters and life histories are predicted to respond to a range of environmental changes via plasticity and adaptation.

When should evolutionarily explicit IPMs be used to predict population responses to environmental change?

Chevin (2015) and Janeiro et al. (in press) speculated that published IPMs that did not include explicit evolutionary processes could provide spurious insight. Three strands of evidence suggest this speculation may often be unwarranted.

First, the signature of evolutionary change in model predictions is a function of the heritability of the trait: when the phenotypic variance is dominated by the environmental component of the phenotype then the dynamics of that component will dominate model predictions. Most IPMs to date have been constructed for body weight (Merow et al., 2014), a trait that often has a heritability of less than 0.2 in vertebrates (e.g., blue tits; Garnett, 1981) and often around 0.1 (e.g., bighorn sheep; Wilson et al., 2005). This means that model predictions will be dominated by the dynamics of the environmental component of the phenotype and that a phenomenological statistical approach to parameterising these models has the potential to capture observed dynamics well.

Second, even when phenotypic traits are heritable, they rarely evolve in the wild as predicted: evolutionary stasis of heritable phenotypic traits in the presence of directional selection is frequently observed in nature (Merilä et al., 2001). When fitness functions are monotonic in the phenotypic value and selection is directional (which is typical for body size (Kingsolver et al., 2001)), then in order to maintain an equilibrium trait distribution the inheritance function must reverse the phenotypic changes caused by selection. Coulson and Tuljapurkar (2008) showed this for the mean phenotypic trait; equation (17) demonstrates that this must apply to all moments of the phenotype

894 distribution. However, when the genotype-phenotype map is additive and there is additive genetic
895 variance for the trait, directional selection is expected to result in evolutionary change and the
896 inheritance function for the genetic component of the phenotype can not reverse genetic changes
897 attributable to selection. Unmeasured genetically correlated characters can prevent evolutionary
898 change in these circumstances, although the cases when this is likely to prevent evolution are restric-
899 tive, and evidence for such characters playing a major role in limiting evolution in the wild is lacking
900 (Agrawal and Stinchcombe, 2009). Assuming selection on the phenotype has been measured ap-
901 propriately and is directional, this suggests that the assumption of an additive genotype-phenotype
902 map may be violated, and the mean of the parental and offspring breeding value distributions may
903 not be equal. A mechanism such as over-dominance can achieve this (Fisher, 1930). Our approach
904 allows the effects of relaxing assumptions of quantitative genetics on evolutionary change to be ap-
905 proximated through the use of phenomenological inheritance functions for the genetic component
906 of the phenotype.

907 Third, because evolutionary change is rarely observed in the wild when it is predicted, observed
908 phenotype change in natural populations is usually attributable to plasticity (e.g. Ozgul et al.,
909 2010, 2009). In these cases, standard, non-evolutionarily explicit, IPMs have accurately captured
910 observed dynamics (Childs et al., 2003; Merow et al., 2014; Ozgul et al., 2010).

911 These three strands of evidence suggest that evolutionarily explicit IPMs may frequently not
912 be required to gain useful insight into population responses to environmental change. If there is no
913 statistical evidence of temporal trends in inheritance, development or fitness function parameters
914 once variation in the ecological environment has been corrected for, then the use of evolutionarily
915 explicit IPMs may result in the construction of unnecessarily complex models. There is often a
916 temptation to include ever more complexity into models, but this comes at the cost of analyt-
917 ical tractability: as more mechanisms or processes are incorporated into models, understanding
918 why a model produces the predictions it does becomes increasingly challenging. However, when
919 evolutionary change is convincingly documented (e.g. Reznick et al., 1997) or is proposed to be a
920 possible mechanism generating rapid phenotypic change (Coltman et al., 2003), the construction of

921 evolutionarily explicit IPMs is advised as the models allow separation of the roles of adaptive and
922 plastic responses to environmental change.

923 We have shown how evolutionarily explicit IPMs can be constructed, invalidating the criticisms
924 of Chevin (2015) and Janeiro et al. (in press) that IPMs have not been developed to incorporate the
925 character-state approach of quantitative genetics. IPMs that are not evolutionarily explicit have
926 been used to address many questions in ecology and their application has proven insightful (Merow
927 et al., 2014). They are likely to remain widely used and we expect this use to result in important
928 new insights. However, we have extended their utility to cases where evolutionary processes are
929 known, or proposed, to be drivers of phenotypic change.

930 **Conclusions**

931 In this paper we have developed a theoretical modeling approach that links demography and quan-
932 titative genetics to explore how populations will respond to environmental change. The approach
933 is general, providing formal links between ecology and evolution. Our work builds upon a growing
934 literature of developing evolutionarily explicit structured population models. This body of litera-
935 ture shows how flexible IPMs are. They provide a powerful tool with the potential to unify ecology
936 and evolution.

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Table 1: Notation used in the paper.

Notation	Definition
\mathcal{Z}	An individual's phenotypic trait value. \mathcal{Z} can be anything that can be measured on an organism when it is captured or observed. \mathcal{Z} cannot be a life history quantity (like life expectancy) which are emergent properties of the dynamics of \mathcal{Z} .
\mathcal{G}	The genetic component of the phenotype defined as the total genotypic contribution of an individual's genotype to \mathcal{Z} . \mathcal{G} can be calculated across multiple loci and can be decomposed into contributions from epistasis, dominance, and additive genetic effects.
\mathcal{A}	The additive genetic component (breeding value) of \mathcal{G} . Change in the distribution of \mathcal{A} reflects change in allele frequencies and consequently evolution.
\mathcal{E}	The environmental component of the phenotype defined as phenotypic variation not attributable to genetic contributions. Determined by gene expression patterns or developmental noise. Nutrient or energy availability may influence gene expression meaning \mathcal{E} may be correlated with environmental drivers θ .
θ	An environmental driver
\mathcal{X}	$\mathcal{X} \in \{\mathcal{Z}, \mathcal{G}, \mathcal{A}, \mathcal{E}\}$
$N(\mathcal{X}, t)$	The distribution of \mathcal{X} at time t
$S(\mathcal{X}, t)$	Survival function: describes the expected association between \mathcal{X} and survival between t and $t+1$. Only used in age-structured models.
$R(\mathcal{X}, t)$	Recruitment function: describes the expected association between \mathcal{X} and the number of offspring produced between t and $t+1$ that survive to recruit into the population at time $t+1$.
$H(\mathcal{X}' \mathcal{X}, t)$	Inheritance function: describes the expected probability of a reproducing individual with character value \mathcal{X} at t producing an offspring with character value \mathcal{X}' at $t+1$ when it recruits to the population.

$D(\mathcal{X}' \mathcal{X}, t)$	Development function: describes expected probability of a surviving individual with character value \mathcal{X} at t expressing character value \mathcal{X}' at $t + 1$. Only used in age-structured models.
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Table 2: Different forms of plasticity and their incorporation into IPMs. Each term in the table below can be included in the functions $\mu^H(\mathcal{E}, t)$, $\mu^H(\mathcal{E}, a, t)$ or $\mu^D(\mathcal{E}, a, t)$. Similar terms could be included in $V^H(\mathcal{E}, t)$, $V^H(\mathcal{E}, a, t)$ or $V^D(\mathcal{E}, a, t)$ if the variance in inheritance or development varied for specific values of \mathcal{E} in predictable ways. This would capture different forms of bet-hedging.

Term	Biological interpretation	Type of plasticity
μ_I^H		No plasticity.
$+\mu_{\mathcal{E}, \mathcal{E}'}^H$	Temporal autocorrelation in \mathcal{E}	No plasticity.
$+\mu_{\theta}^H$	Ecological environment influences all values of \mathcal{E} in the same way.	Additive plasticity generated by temporal variation in the ecological environment.
$+\mu_{\theta, \mathcal{E}}^H \theta \mathcal{E}$	Temporal autocorrelation in \mathcal{E} depends upon the ecological environment.	Non-additive plasticity generated by temporal and spatial variation in the ecological environment.
$+\mu_{\mathcal{A}}^H \mathcal{A}$	Value of \mathcal{E} depends upon \mathcal{E} .	No plasticity unless \mathcal{E} also depends upon θ .
$+\mu_{\theta, \mathcal{A}}^H \theta \mathcal{A}$	Value of the \mathcal{E} depends upon an interaction between \mathcal{A} and the ecological environment.	Genotype by environment interaction.
$+\mu_{\mathcal{A}, \mathcal{E}'}^H \mathcal{A} \mathcal{E}'$	Temporal autocorrelation in \mathcal{E} depends upon the \mathcal{A} .	Genotype by environment interaction.

1145

1146 Figure legends

1147 **Figure 1.** The role of selection on the dynamics of \mathcal{A} . Dynamics of univariate \mathcal{A} subject to
 1148 linear selection and clonal inheritance (a)-(d) (SI §1.1 Model A). The population does not reach

1149 an equilibrium, with (a) population growth, and the (b) mean, (c) variance and (d) skew of the
1150 character continually evolving. Imposing a maximum possible character value constrains change
1151 (red lines versus black lines (a)-(d)). In the age-structured case we track the dynamics of a bivariate
1152 character distribution (e)-(g) (SI §1.1 models B, C and D). The models in (e) and (f) (SI Models
1153 B and C) are identical except the starting distribution at time $t = 1$ has a covariance of -0.2 in (f)
1154 compared to 0.7 in (e). The parameterisation in (g) is chosen to demonstrate a case where the two
1155 traits evolve in different directions. The coloured image plots in figures (e)-(g) represent Gaussian
1156 development functions $D(\mathcal{Z}'|\mathcal{Z}, t)$ fitted to the bivariate distributions of \mathcal{A} at the beginning and end
1157 of the simulation. Evolution of the bivariate character has resulted in different parameterisations
1158 of these phenomenological functions. The lighter the shading, the greater the probability of a
1159 transition from character value \mathcal{Z} at age 1 and to \mathcal{Z}' age 2.

1160 **Figure 2.** The dynamics of inheritance (SI Model E). The dynamics of (a) population growth rate
1161 (R_0), the (b) mean and (c) variance of \mathcal{A} vary between models with clonal inheritance (black line),
1162 the convolution in equation (15) (red line) and the Gaussian inheritance function in equation (16)
1163 (blue line). Dynamics predicted from the convolution and the Gaussian inheritance function are
1164 indistinguishable in this model. (d) the temporal dynamics of the clonal model versus the other
1165 models. The initial distribution at $t = 1$ is Gaussian. After 100 generations the character distribu-
1166 tions predicted by the clonal and sexual models have only diverged slightly. The infinitesimal model
1167 of quantitative genetics assumes that the dynamics of alleles can be inferred from the dynamics of
1168 genotypes. Under this assumption (e) selection alters genotype and allele frequencies, while inheri-
1169 tance does not. In contrast, (f) when dominance variance operates, both selection and inheritance
1170 alter genotype frequency while neither alter allele frequencies. For a Gaussian distributed char-
1171 acter, (g) dominance variance acts as an offset, reducing the intercept of a Gaussian inheritance
1172 function.

1173 **Figure 3.** Dynamics of \mathcal{E} and plasticity. (a) Return times to equilibrium for three inheritance
1174 functions (SI §1.1 models F-H) following a one off perturbation (see main text). There is no
1175 plasticity incorporated into model F (black line). Model G (red line) and model H (blue line)

1176 respectively incorporate adaptive and non-adaptive phenotypic plasticity. In (b) and (c) we imposed
1177 a permanent environmental change by reducing the intercept of the fitness function. (c) Represents
1178 the mean phenotype.

1179 **Figure 4.** A dynamic version of the Breeders Equation. The dynamics of the phenotype (red lines)
1180 and its genetic (black lines) and environmental (blue lines) components (a)-(c) and (g)-(i), and the
1181 dynamics of the population (d)-(f) and (j)-(l). In the first model (a) and (d), both fitness and
1182 inheritance of the environmental component of the phenotype are independent of density (SI §1.1
1183 model I). In the second model (b) and (e) fitness is negatively density-dependent and inheritance
1184 of the environmental component of the phenotype is density-independent (SI §1.1 model J). In the
1185 third model, both fitness and inheritance of the environmental component of the phenotype are
1186 negative density-dependent (SI §1.1 Model K). The treatment of plasticity can dramatically influ-
1187 ence the dynamics of the phenotype and population size (SI §1.1 models L-N). Adaptive phenotypic
1188 plasticity (h) and (k) leads to the population size and phenotype recovering from a perturbation
1189 much faster than non-adaptive plasticity (i)-(l). The absence of a plastic response (g) and (j) re-
1190 sults in the population recovering from a perturbation at an intermediate rate between cases where
1191 adaptive and non-adaptive plasticity are operating.

1192 **Figure 5.** Dynamics of bivariate characters and evolution of fitness functions in the presence of
1193 an unmeasured, genetically correlated character (SI §1.1 model P and Q). We construct a simple
1194 model with clonal inheritance of two correlated characters that both influence fitness. We explore
1195 two initial starting conditions that only differ in their genetic covariance (SI §1.1 models P and Q).
1196 In (a)-(d) the covariance accelerates the rate of evolution compared to (e)-(h). The dynamics of the
1197 fitness function for each character when the other character is not measured (i) and (j). Regardless
1198 of the covariance between characters, the fitness functions evolve during the course of 120 time step
1199 simulation.

Figure 1

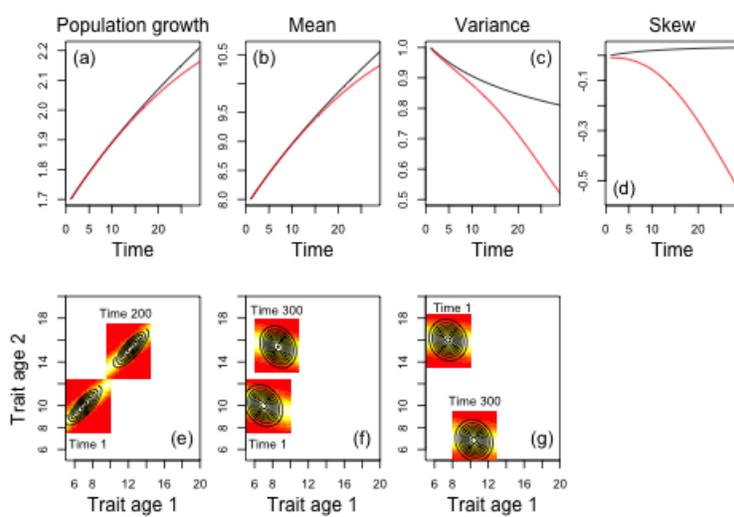


Figure 2

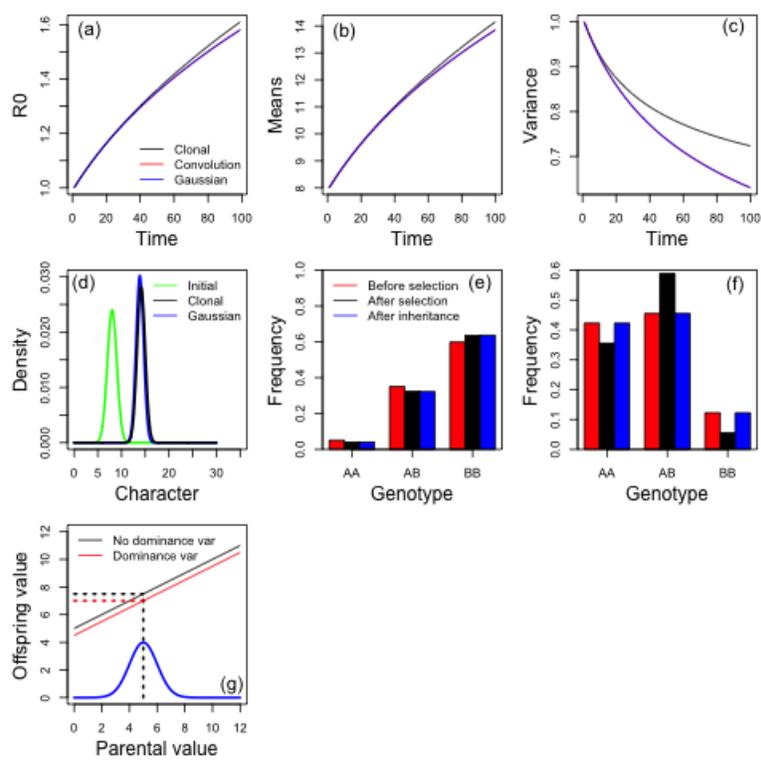


Figure 3

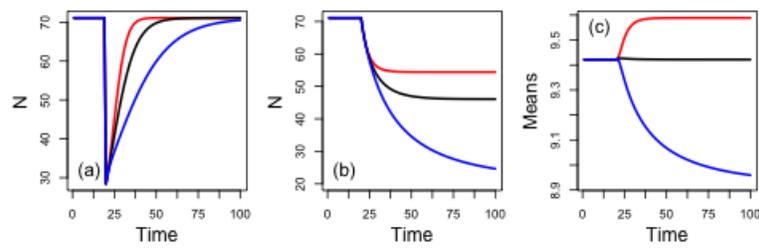


Figure 4

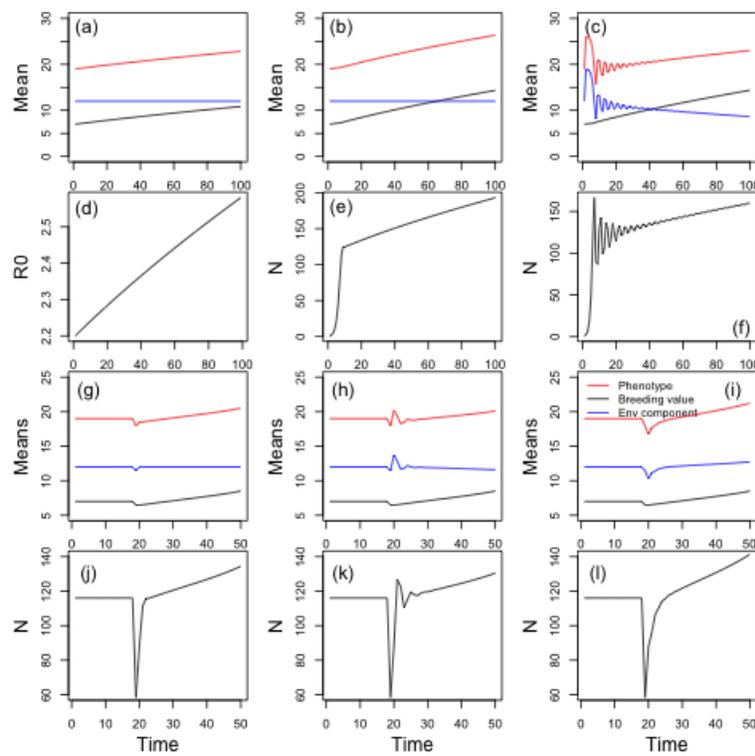
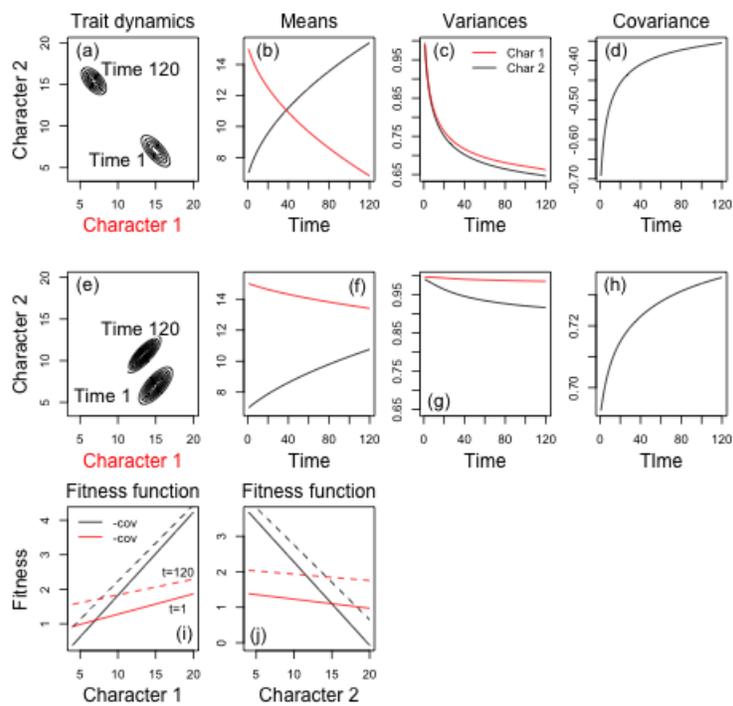


Figure 5



1200 Supplementary information

1201 1.1 Model Parameterization

1202 Model A:

$$N(\mathcal{A}, t = 1) = \phi(8, 1)$$

$$R(\mathcal{A}, t) = 0.1 + 0.2\mathcal{A}$$

$$\mu_H(\mathcal{A}, t) = \mathcal{A}$$

$$V(\mathcal{A}, t) = 0$$

$$x = \infty \text{ or } x = 11.5$$

1203 Models B and C:

$$S(\mathcal{A}1, 1, t) = \frac{1}{1 + e^{-(0.1+0.03\mathcal{A})}}$$

$$S(\mathcal{A}2, 2, t) = 0$$

$$R(\mathcal{A}1, 1, t) = 0$$

$$R(\mathcal{A}2, 2, t) = e^{0.01-0.075\mathcal{A}}.$$

1204 Starting conditions at time $t = 1$ are multivariate normal with the following parameters, **Model**

1205 B:

$$\mathbb{E}(\mathcal{A}1) = 7$$

$$\mathbb{E}(\mathcal{A}2) = 10$$

$$\sigma^2(\mathcal{A}1) = 1$$

$$\sigma^2(\mathcal{A}2) = 0.8$$

$$\sigma(\mathcal{A}1, \mathcal{A}2) = -0.2$$

1206 **Model C:**

$$\begin{aligned}\mathbb{E}(\mathcal{A}1) &= 7 \\ \mathbb{E}(\mathcal{A}2) &= 10 \\ \sigma^2(\mathcal{A}1) &= 1 \\ \sigma^2(\mathcal{A}2) &= 0.8 \\ \sigma(\mathcal{A}1, \mathcal{A}2) &= 0.2\end{aligned}$$

1207 **Model D:**

$$\begin{aligned}S(\mathcal{A}, 1, t) &= \frac{1}{1 + e^{-(0.1+0.06\mathcal{A})}} \\ S(\mathcal{A}, 2, t) &= 0 \\ R(\mathcal{A}, 1, t) &= 0 \\ R(\mathcal{A}, 2, t) &= e^{0.01+0.05\mathcal{A}}.\end{aligned}$$

1208 Starting conditions at time $t = 1$ for **model D:**

$$\begin{aligned}\mathbb{E}(\mathcal{A}1) &= 7.5 \\ \mathbb{E}(\mathcal{A}2) &= 16 \\ \sigma^2(\mathcal{A}1) &= 1 \\ \sigma^2(\mathcal{A}2) &= 0.8 \\ \sigma(\mathcal{A}1, \mathcal{A}2) &= -0.1\end{aligned}$$

Model E:

$$R(\mathcal{A}, t) = 0.2 + 0.1\mathcal{A}. \quad (22)$$

1209 **Model F:** no plasticity:

$$\begin{aligned}R(\mathcal{E}, t) &= 0.2 + 0.1\mathcal{E} - 0.002n(t) \\ \mu_H(\mathcal{E}, t) &= 4.64 + 0.5\mathcal{E} \\ V_H(\mathcal{E}, t) &= 1\end{aligned}$$

1210 **Model G:** Adaptive phenotypic plasticity:

$$\begin{aligned}R(\mathcal{E}, t) &= 0.2 + 0.1\mathcal{E} - 0.002n(t) \\ \mu_H(\mathcal{E}, t) &= 5 + 0.5\mathcal{E} - 0.005n(t) \\ V_H(\mathcal{E}, t) &= 1\end{aligned}$$

1211 **Model H:** Non-adaptive plasticity:

$$\begin{aligned}R(\mathcal{E}, t) &= 0.2 + 0.1\mathcal{E} - 0.002n(t) \\ \mu_H(\mathcal{E}, t) &= 4.29 + 0.5\mathcal{E} + 0.005n(t) \\ V_H(\mathcal{E}, t) &= 1\end{aligned}$$

1212 **Model I**

$$\begin{aligned}w(\mathcal{Z}, t) &= 0.3 + 0.1\mathcal{Z} \\ \mu^H(\mathcal{E}, t) &= 0 \\ v^H(\mathcal{E}, t) &= 1\end{aligned}$$

1213 **Model J**

$$\begin{aligned}w(\mathcal{Z}, t) &= 0.3 + 0.1\mathcal{Z} - 0.01n(t) \\ \mu^H(\mathcal{E}, t) &= 0 \\ v^H(\mathcal{E}, t) &= 1\end{aligned}$$

1214 **Model K**

$$\begin{aligned}w(\mathcal{Z}, t) &= 0.3 + 0.1\mathcal{Z} - 0.01n(t) \\ \mu^H(\mathcal{E}, t) &= 19 - 0.065n(t) \\ v^H(\mathcal{E}, t) &= 1\end{aligned}$$

1215 Initial starting conditions for $\mathcal{Z} = \mathcal{A} + \mathcal{E}$ for **models I to K**:

$$\begin{aligned}\mathbb{E}(\mathcal{A}) &= 7 \\ \mathbb{E}(\mathcal{E}) &= 12 \\ \sigma^2(\mathcal{A}) &= 1 \\ \sigma^2(\mathcal{E}) &= 1 \\ \sigma(\mathcal{A}, \mathcal{E}) &= 0\end{aligned}$$

1216 **Model L**

$$\begin{aligned}w(\mathcal{Z}, t) &= 0.3 + 0.1\mathcal{Z} - 0.01n(t) \\ \mu^H(\mathcal{E}, t) &= 12 \\ v^H(\mathcal{E}, t) &= 1\end{aligned}$$

1217 **Model M**

$$\begin{aligned}w(\mathcal{Z}, t) &= 0.3 + 0.1\mathcal{Z} - 0.01n(t) \\ \mu^H(\mathcal{E}, t) &= 15.48 - 0.03n(t) \\ v^H(\mathcal{E}, t) &= 1\end{aligned}$$

1218 **Model N**

$$\begin{aligned}w(\mathcal{Z}, t) &= 0.3 + 0.1\mathcal{Z} - 0.01n(t) \\ \mu^H(\mathcal{E}, t) &= 8.52 + 0.03n(t) \\ v^H(\mathcal{E}, t) &= 1\end{aligned}$$

1219 Initial starting conditions for $\mathcal{Z} = \mathcal{A} + \mathcal{E}$ for **models L to N**:

$$\begin{aligned}\mathbb{E}(\mathcal{A}) &= 7 \\ \mathbb{E}(\mathcal{E}) &= 12 \\ \sigma^2(\mathcal{A}) &= 1 \\ \sigma^2(\mathcal{E}) &= 1 \\ \sigma(\mathcal{A}, \mathcal{E}) &= 0\end{aligned}$$

1220 **Models P and Q:**

$$\begin{aligned}w(\mathcal{A}, t) &= 2 - 0.13\mathcal{A}_1 + 0.15\mathcal{A}_2 \\ N(\mathcal{A}', t + 1) &= w(\mathcal{A}, t)N(\mathcal{A}, t)\end{aligned}$$

1221 Starting conditions at time $t + 1$ for **model P**:

$$\begin{aligned}\mathbb{E}(\mathcal{A}_1) &= 7 \\ \mathbb{E}(\mathcal{A}_2) &= 15 \\ \sigma^2(\mathcal{A}_1) &= 1 \\ \sigma^2(\mathcal{A}_2) &= 1 \\ \sigma(\mathcal{A}_1, \mathcal{A}_2) &= -0.7\end{aligned}$$

1222 Starting conditions at time $t + 1$ for **model Q**:

$$\begin{aligned}\mathbb{E}(\mathcal{A}_1) &= 7 \\ \mathbb{E}(\mathcal{A}_2) &= 15 \\ \sigma^2(\mathcal{A}_1) &= 1 \\ \sigma^2(\mathcal{A}_2) &= 1 \\ \sigma(\mathcal{A}_1, \mathcal{A}_2) &= 0.7\end{aligned}$$

1223 1.2 Calculating quantities from model outputs

The expectation of a distribution of $\mathcal{X} = (\mathcal{G}, \mathcal{A}, \mathcal{E}, \mathcal{Z})$ can be calculated as

$$\mathbb{E}(\mathcal{X}, t) = \frac{\int \mathcal{X} N(\mathcal{X}, t) d\mathcal{X}}{\int N(\mathcal{X}, t) d\mathcal{X}}, \quad (23)$$

The variance of a distribution can be calculated as

$$\sigma^2(\mathcal{X}, t) = \frac{\int \mathcal{X} \mathcal{X} N(\mathcal{X}, t) d\mathcal{X}}{\int N(\mathcal{X}, t) d\mathcal{X}} - \mathbb{E}(\mathcal{X}, t)^2. \quad (24)$$

For a bivariate distribution \mathcal{X} consisting of traits $\mathcal{X}1$ and $\mathcal{X}2$ then the covariance between these two traits will be,

$$\sigma(\mathcal{X}1, \mathcal{X}2, t) = \frac{\int \mathcal{X}1 \mathcal{X}2 N(\mathcal{X}, t) d\mathcal{X}}{\int N(\mathcal{X}, t) d\mathcal{X}} - \mathbb{E}(\mathcal{X}1, t) \mathbb{E}(\mathcal{X}2, t). \quad (25)$$

The skew can be calculated as,

$$s^3(\mathcal{X}) = \frac{\int \mathcal{X}^3 N(\mathcal{X}, t) d\mathcal{X}}{\int N(\mathcal{X}, t) d\mathcal{X}} - 3\mathbb{E}(\mathcal{X}, t)\sigma^2(\mathcal{X}, t) - \frac{\mathbb{E}(\mathcal{X}, t)^3}{\sqrt{\sigma^2(\mathcal{X}, t)^3}}$$

1224 The kurtosis can be calculated in the following way. First, we define the n^{th} non-central moment
1225 of a distribution at time t as $m^n(\mathcal{X}, t) = \frac{\int \mathcal{X}^n N(\mathcal{X}, t) d\mathcal{X}}{\int N(\mathcal{X}, t) d\mathcal{X}}$, then,

$$k^4(\mathcal{X}) = \frac{\frac{\int \mathcal{X}^4 N(\mathcal{X}, t) d\mathcal{X}}{\int N(\mathcal{X}, t) d\mathcal{X}} - 4\mathbb{E}(\mathcal{X}, t)m^3(\mathcal{X}, t) + 6\mathbb{E}(\mathcal{X}, t)^2 m^2(\mathcal{X}) - 3\mathbb{E}(\mathcal{X}, t)^4}{\sigma^2(\mathcal{X}, t)} - 3$$

1226 1.3 Gaussian inheritance function when demography differs between males and 1227 females

1228 The distribution of mothers and fathers at time t is respectively defined as $N_R^f(\mathcal{A}, t)$ and $N_R^m(\mathcal{A}, t)$.

1229 These distributions are the same size.

We can write

$$N(\mathcal{A}, t + 1) = \int H(\mathcal{A}' | \mathcal{A}_m, \mathcal{A}_f, t) N_R^m(\mathcal{A}, t) d\mathcal{A} \quad (26)$$

1230 where the component functions of $H(\mathcal{A}' | \mathcal{A}_m, \mathcal{A}_f, t)$ are

$$\begin{aligned} \mu^H(\mathcal{A}, t) &= (1 - \eta) \mathbb{E}(N_R^f(\mathcal{A}, t)) + \eta \mathcal{A} \\ V^H(\mathcal{A}, t) &= (1 - \eta)^2 \sigma^2(N_R(\mathcal{A}, t)) \end{aligned} \quad (27)$$

1231 and $\sigma^2(N_R(\mathcal{A}, t))$ is the variance in \mathcal{A} across all parents.

Alternatively,

$$N(\mathcal{A}, t + 1) = \int H(\mathcal{A}'|\mathcal{A}_m, \mathcal{A}_f, t) N_R^f(\mathcal{A}, t) d\mathcal{A} \quad (28)$$

1232 where the component functions of $H(\mathcal{A}'|\mathcal{A}_m, \mathcal{A}_f, t)$ are

$$\begin{aligned} \mu^H(\mathcal{A}, t) &= (1 - \eta)\mathbb{E}(N_R^m(\mathcal{A}, t)) + \eta\mathcal{A} \\ V^H(\mathcal{A}, t) &= (1 - \eta)^2\sigma^2(N_R(\mathcal{A}, t)). \end{aligned} \quad (29)$$

1233 As the distributions $N_R^f(\mathcal{A}, t)$ and $N_R^m(\mathcal{A}, t)$ depart from normality, the approximations will
1234 predict dynamics that diverge from those predicted by the convolution.

1235 1.4 How do different functions alter character distributions?

1236 Assume $N(\mathcal{X}, t)$ is proportional to a Gaussian distribution. The following parameterizations of a
1237 transition functions $H(\mathcal{X}'|\mathcal{X}, t)$ in a model $N(\mathcal{X}', t + 1) = \int H(\mathcal{X}'|\mathcal{X}, t)N(\mathcal{X}, t)$ will have no effect
1238 on the location or shape of the distribution such that $N(\mathcal{X}, t) = N(\mathcal{X}', t + 1)$,

$$\begin{aligned} \mu^H(\mathcal{X}, t) &= (1 - \beta)\mathbb{E}(\mathcal{X}, t) + \beta\mathcal{X} \\ V^H(\mathcal{X}, t) &= (1 - \beta^2)\sigma^2(\mathcal{X}, t). \end{aligned} \quad (30)$$

1239 Note that in this model there is no fitness function and no selection.

1240 When the intercept of $\mu^H(\mathcal{X}, t)$ is less than $(1 - \beta)\mathbb{E}(\mathcal{X}, t)$ then $\mathbb{E}(\mathcal{X}', t + 1) < \mathbb{E}(\mathcal{X}', t)$ and vice
1241 versa. A function $\mu^H(\mathcal{X}, t)$ can consequently be parameterized to reduce the mean of a distribution
1242 across generations or time steps if desired.

1243 The slope β will reduce $\sigma^2(\mathcal{X}', t + 1)$ by β^2 compared to $\sigma^2(\mathcal{X}, t)$. The intercept of $V^H(\mathcal{X}, t)$
1244 injects additional variation. If the intercept is larger than $(1 - \beta^2)\sigma^2(\mathcal{X}, t)$ then $\sigma^2(\mathcal{X}', t + 1) >$
1245 $\sigma^2(\mathcal{X}, t)$. Functions $\mu^H(\mathcal{X}, t)$ and $V^H(\mathcal{X}, t)$ can consequently be selected to alter the variance from
1246 one time step or age to the next.

1247 The further the distribution $N(\mathcal{X}, t)$ departs from normality, the more approximate these equal-
1248 ities will become. However, large departures from these equalities can be used to increase the mean
1249 or variance of any distribution in a desired direction.

1250 In Figure S1 we show how $\mu^H(\mathcal{X}, t)$ and $V^H(\mathcal{X}, t)$ can be parameterized to modify the mean
1251 and variance of $N(\mathcal{X}, t)$ when it is proportional to a normal distribution.

1252 1.5 mortality selection and changes in the mean phenotype

1253 When a trait is normally distribution, selection needs to be strong in order to substantially shift the
1254 mean of a phenotype distribution. Such strong selection inevitably leads to a decrease in population
1255 size. In Figure S3 we show how killing 25% of the heaviest individuals has only a small effect on
1256 the mean for a distribution with a mean of 0 and a standard deviation of unity. The evolutionary
1257 response is even less if \mathcal{E} and \mathcal{G} are uncorrelated. For example, in the example in Figure S3, the
1258 evolutionary response would be half the phenotypic response for $h=0.5$. In order to substantially
1259 shift the mean of the a normal distribution via mortality selection it is necessary for the majority
1260 of the population to die.

1261 Supplementary Information Figure Legends

1262 **Figure S1.** How parameterizations of transition functions for the environmental component of the
1263 phenotype $H(\mathcal{E}|\mathcal{E}', t)$ can be used to grow, maintain or shrink the mean and variance of $N(\mathcal{E}, t+1)$.
1264 We start with a normal distribution. The initial distribution is represented with a black line in
1265 the main figures. The inset figures in (a) to (c) shows the transition functions, with the black line
1266 representing the function that has no effect on the location or shape of $N(\mathcal{E}, t)$. (a) increasing or
1267 decreasing the intercept of $\mu^H(\mathcal{E}, t)$ influences the location, but not the shape of $N(\mathcal{E}, t)$. (b) How
1268 altering the slope of $\mu^H(\mathcal{E}, t)$ influences the shape of $N(\mathcal{E}, t)$. In this example the mean is unaffected
1269 as the function passes through the x, y co-ordinate $(\mathbb{E}(\mathcal{E}, t), \mathbb{E}(\mathcal{E}, t))$. (c) how altering the intercept
1270 of $V^H(\mathcal{E}, t)$ influences the variance. The transition functions in the insets of (b) and (c) generate
1271 distributions with the same means and variances (compare blue, red and black distributions in (b)
1272 and (c)). A change in variance between $N(\mathcal{E}, t)$ and $N(\mathcal{E}', t+1)$ achieved by altering the slope
1273 of $\mu^H(\mathcal{E}, t)$ or the intercept of $V^H(\mathcal{E}, t)$ generates different amounts of mixing. In (d) upper and
1274 lower $H(\mathcal{E}'|\mathcal{E}, t)$ functions impact the variance to the same extend (left hand figures) except the red

1275 function simply spreads out the distribution without altering the relative rank of each individual.

1276 In contrast, the blue function changes relative ranks (right hand figures).

1277 **Figure S2.** Dynamics of the additive genetic variance (a)-(c) and the heritability (d)-(f) in models

1278 I to K. Models of the additive genetic (back line) and environmental (red line) variance (g)-(i)

1279 and the heritability (j)-(l) in models L to N. See Figure 5 main paper for dynamics of means and

1280 population growth.

1281 **Figure S3.** A normal distribution with mean 0 and standard deviation 1 prior to mortality

1282 selection (black line). Mortality occurs, killing off the top 25% of individuals (red distribution).

1283 The mean changes from 0 (vertical dashed line) to -0.0324. In other words, even a large highly

1284 selective mortality event has a relatively small effect on the mean of a normal distribution.

Figure S1

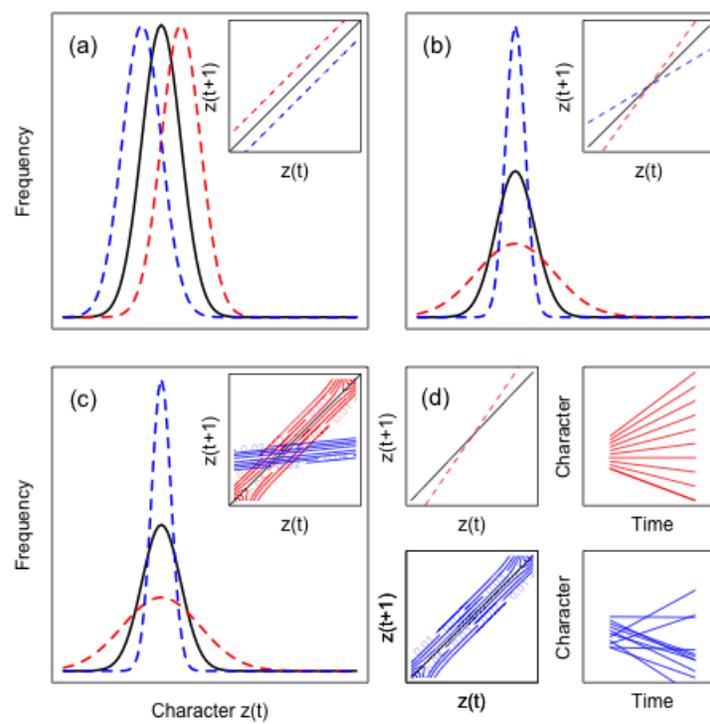


Figure S2

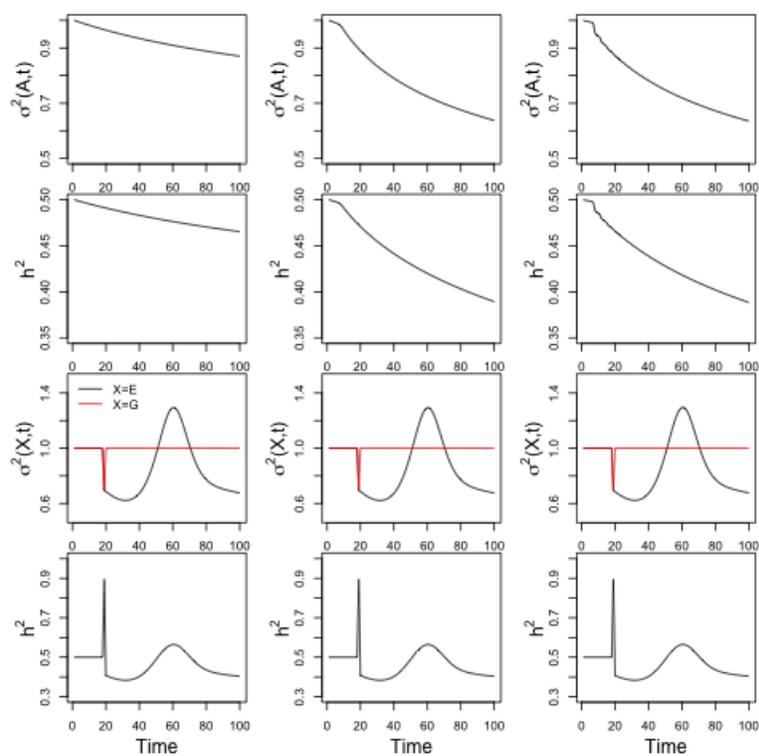


Figure S3

