Adapting to a changing environment: modeling the interaction of directional selection and plasticity.

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Abstract

Human-induced habitat loss and fragmentation constrains the range of many species, making them unable to respond to climate change by moving. For such species to avoid extinction, they must respond with some combination of phenotypic plasticity and genetic adaptation. Haldane's "cost of natural selection" limits the rate of adaptation, but, although modeling has shown that in very large populations long-term adaptation can be maintained at rates substantially faster than Haldane's suggested limit, maintaining large populations is often an impossibility, so phenotypic plasticity may be crucial in enhancing the long-term survival of small populations. The potential importance of plasticity is in "buying time" for populations subject to directional environmental change: if genotypes can encompass a greater environmental range, then populations can maintain high fitness for a longer period of time. Alternatively, plasticity could be detrimental by lessening the effectiveness of natural selection in promoting genetic adaptation. Here I modeled a directionally changing environment in which a genotype's adaptive phenotypic plasticity is centered around the environment where its fitness is highest. Plasticity broadens environmental tolerance and, provided it is not too costly, is favored by natural selection. However, a paradoxical result of the individually advantageous spread of plasticity is that, unless the adaptive trait is determined by very few loci, the long-term extinction risk of a population increases. This effect reflects a conflict between the short-term individual benefit of plasticity and a long-term detriment to population persistence, adding to the multiple threats facing small populations under conditions of climate change.

Introduction

When climatic conditions change, species can only persist by shifting their range, by genetic adaptation, and/or through the benefits of adaptive phenotypic plasticity. Climate is never constant, but it is becoming increasingly clear that the rate of anthropogenic global warming is having a significant effect on the biosphere (Penuelas et al 2013) and is adding significantly to the background extinction risk (Thomas *et al.* 2004; Malcolm et al 2006; Williams et al 2008; Maclean and Wilson 2011; Foden et al 2013; Pacifici et al 2015).

In the geological past, many species were able to minimize the effects of climate change through a shift in their latitudinal or altitudinal range (see Dawson et al 2011; Garcia et al. 2014), and evidence is already accumulating that range shifts consistent with global warming have started to occur (Parmesan 2006; Jump et al 2009). Range shifts do not guarantee species survival (Thomas *et al.* 2004; Davis *et al.* 2005; Parmesan 2006; Garcia et al. 2014), but minimally a range shift can be expected to delay the consequences of long-term directional climate change.

Unfortunately it is increasingly the case that a range shift is precluded: many plant and animal species are confined to natural areas surrounded by urban or agricultural development that prevents dispersal from one patch of habitat to another. Under such conditions, long-term survival under climate change depends upon genetic adaptation and phenotypic plasticity. Over the last few years there has been substantial interest in the relative roles of these two factors, and their interaction, in the response to climate change (see Franks et al 2013). The empirical data support a strong involvement of plasticity in this response (Hendry et al 2008; Merila and Hendry 2014). While the benefit of adaptive plasticity to individuals in the short term is clear, it is less clear if plasticity is beneficial over the longer term since it may reduce the effectiveness of natural selection in driving adaptation to the changing conditions (Ghalambor et al 2007). In essence, plasticity can reduce the rate at which beneficial alleles highly adapted to the current environment spread in a population since imperfectly adapted genotypes retain a high fitness, reducing the selection differential. This effect allows a longer lag to build up between the current state of the environment and the optimum environment of the genotypes present. If this "lag load" gets too long, the population can no longer sustain itself (Maynard Smith 1976). Thus to be beneficial, plasticity must not inhibit adaptive change that shifts the elevation of the reaction norm to track the environment, since, given continuing directional climate change,

plasticity has limits. In a changing environment, populations must ultimately adapt or decline to extinction (Davis and Shaw 2001; Rice and Emery 2003; Davis *et al.* 2005; Jump and Peñuelas 2005; Bradshaw and Holzapfel 2006; Kinnison and Hairston 2007; Visser 2008; Moritz and Agudo 2013).

Haldane (1957) was the first to analyze the important problem of how rapidly populations can genetically adapt to a changing environment. He argued that gene frequency change due to natural selection could be viewed in terms of genetic deaths and that this "cost of natural selection" was an important limiting factor. The cost (C), expressed in units of population number, depends primarily on the initial frequency of a beneficial mutation (p_0), e.g. given additive fitness, $C \approx - 2 \ln(p_0)$. Haldane concluded that this cost, when combined with extrinsic mortality, would limit the rate of adaptation to an average sustainable over long evolutionary periods of about 1 substitution per 300 generations. This estimate was an important factor in Kimura's (1968) argument for the prevalence of neutral substitutions in molecular evolution, but more recently it has taken on a new level of importance in relation to long-term climate change and the future of biodiversity (Nunney 2003). In particular, the results derived from Haldane's model suggest revisiting estimates of the population size consistent with long-term viability. Early theoretical analyses of the effects of reduced population size on extinction risk and the loss of genetic variation suggested species conservation guidelines of at least several thousand individuals (Nunney and Campbell 1993, Lande 1995); however, under conditions of environmental change this guideline may prove to be a serious underestimate (Nunney 2003).

The limits on the rate of adaptation, especially in small populations, serve to emphasize the question of whether adaptive phenotypic plasticity has an important role in promoting persistence. Plasticity broadens the range of conditions under which an individual genotype can maintain a high fitness, and, as a result, enable individuals to successfully survive and reproduce despite local environmental change. However, this leaves unanswered the important question of whether phenotypic plasticity can weaken the effect of natural selection and so impede long-term adaptation.

This possibility has prompted the development of theory and simulations that model the interaction of plasticity and adaptation, most notably Chevin et al (2010). They showed that (a) low- or no-cost plasticity promotes long-term adaptation, and (b) that as the costs of plasticity increase there is an intermediate level of plasticity that makes the population most resilient given

environmental change. The goal of the present work was to build on this foundation by incorporating adaptive plasticity into simulations based around Haldane's original model., focusing on whether or not plasticity is likely to be beneficial over the long term. Following the approach of Chevin et al (2010), it was assumed that environmental change was linear with time and that plasticity was defined by a linear reaction norm. However, unlike in the earlier model, the reaction norm of each genotype was defined relative to the environment in which it was best adapted, rather than relative to a standard reference environment. This seemingly minor change alters the effect of plasticity on new mutations and was found to have important consequences.

A demographic evolutionary model

To simulate the relationship between the rate of environmental change, genetic adaptation, plasticity and the risk of extinction it is necessary to (a) interpret environmental change in genetic terms, (b) define a fitness model, (c) define a demographic model, and (d) relate phenotypic plasticity to fitness. Modeling the first three of these features employed the methods introduced by Nunney (2003), and are summarized below, while modeling phenotypic plasticity is considered in the next section.

The discrete-generation model was individual based with a lottery polygyny mating system with females mating once (Nunney 1993) and density-dependent female fecundity. The sex of offspring was assigned randomly (with a 1:1 sex ratio) and the n-locus genotype of each offspring was determined from its parents assuming free recombination.

An offspring's fitness depended on the match between its genotype and the current state of the environment, mediated through Gaussian stabilizing selection acting on a single phenotypic trait, z'. The optimum value of the trait (i.e. the phenotype with the highest fitness) was arbitrarily set to zero for all times $t \leq 0$ (with t in generations); however for $t > 0$, it was assumed that the optimum trait value increased linearly with time, due to the effect of a changing environment. Specifically, it was assumed that some environmental variable (E) was increasing linearly with time, driving a linear increase in the optimum trait value (see Figure 1), i.e. $z'_{opt} = bE_{(t)} = abt$. Thus the fitness function of a genotype maps directly from a scale of trait values to a scale of the environmental variable and to a scale of time. As a result, the mean of the fitness function for a given genotype i ($z'_{i,opt}$) defines both the environmental condition and the time at which that genotype would have maximum fitness, with the standard deviation reflecting the tolerance of that genotype to environmental conditions around its specific genotypic optimum.

The trait value of each genotype was made up of the additive effect of one or more (= n) loci plus a random environmental effect (e_i). Following Lynch and Gabriel (1987), the fitness of individual i can be defined as:

$$w_i = \exp\left\{-\frac{r}{s_i^2}\left[\frac{1}{n}\sum_j z_{ij} - \left(\frac{t}{T}\right) + e_i\right]^2\right\} \qquad (1)$$

where the trait value (z') was transformed to a genetic scale z, so that each allele adds or subtracts one unit across the n loci determining the trait, as outlined below, with the current optimum $z_{(t)} = cz'_{(t)} = bcE_{(t)} = abct = t/T$ (where $1/T = abc$). Thus z_{ij} is the average allele score (across the 2 copies) at locus j. The breadth of environmental tolerance was measured by s_i, which is proportional to the standard deviation of the fitness function ω_i ($= s_i/(2r)^{1/2}$), while each individual's environmental component (e_i) was normally distributed with zero mean and specified variance. The population's intrinsic rate of increase r was included in equation (1) specifically (and only) for the purpose of enabling comparisons if r is varied (see below for more details).

The parameter T creates the link between environmental change and allelic substitutions by defining the rate of environmental change in terms of an "allelic cycle". An allelic cycle (T) is the average interval (in generations) between allelic substitutions at each of the n identical loci that is necessary to maintain adaptation. As the rate of environmental change increases, a faster average rate of substitution is necessary, and T decreases. Note that this is an average interval across loci since, in the additive model used, extra substitutions at one locus can substitute for fewer substitutions at another.

An important feature of the model is the assumption that the genetic basis of the adaptive trait can be defined along a continuum from a single gene of major effect, through a few genes of moderate effect, to many genes of small effect. The intent is to span the range from a genetically simple traits (e.g. the classic case of industrial melanism) to a typical quantitative genetic trait. For this reason, the additive effect of a locus declines with n, the number of loci.

Since adaptation to environmental change involves n loci, the average number of generations between consecutive adaptive substitutions consistent with tracking the environment is $S = T/n$. This interval S between adaptive substitutions is the timescale used by Haldane (1957), when he concluded that, given the genetic deaths associated with adaptation and the probable level of extrinsic background mortality, the average rate of substitution interval likely

to be sustainable over evolutionary time was about one per 300 generations (i.e. $S = 300$). In the simulations presented, Haldane's "cost of natural selection" was quantified using this same metric as the minimum interval between substitutions that the population could withstand without going extinct (S_{min}), so that $1/ S_{min}$ is the maximum rate of adaptive genetic change per generation consistent with population persistence.

Adaptation requires the continuous substitution of new beneficial alleles at a rate proportional to the rate of environmental change. Populations that fail to adapt fast enough eventually decline to extinction. To facilitate long-term adaptation, the model incorporated an individual's beneficial mutation rate as u/locus/generation. The alleles at each adaptive locus were arranged in an increasing integer sequence (0, 1, 2,..), each with an effect matching their label. Thus in the simulations the allele "0" was favored (at all loci) during the initial burn-in period from $t = -T$ to $t = 0$ (to initiate mutation-selection balance), but as the environment begins to change (at $t = 0$) the "0" alleles become less advantageous and the "1" allele increasingly favored; however after $t = T$ generations (one allelic cycle, when $t/T = 1$) the advantage of having an average allelic score of "1" begins to decline and allelic combinations with an average score of "2" increasingly favored, and so on. Beneficial mutation always gave rise to a new allele that was a single step further along the sequence than the parent allele, i.e. allele z to allele $z+1$. An equal and opposite production of deleterious alleles was also included.

The evolutionary response (or lack of it) to environmental change was linked to extinction risk by a logistic-like demographic model. Population regulation acted via female fecundity (f) according to:

$$f = 2\exp\left\{r\left[1-\left(\frac{N}{K}\right)^{1/r}\right]\right\} \qquad (2)$$

where K is the carrying capacity, and r is defined by $R = 2e^r$, the maximum reproductive rate of females. In all simulations discussed, $R = 10$.

The density dependence used in equation (2) is a special case of the function advocated by Gilpin and Ayala (1973). It was chosen to avoid oscillatory or chaotic dynamics, i.e. to have an eigenvalue (and hence local stability) independent of the intrinsic growth rate (r). This feature was incorporated so that the model's behavior population dynamics close to the carrying capacity would be independent of r allowing a comparison of simulations varying r based on population genetic rather than population dynamic effects. Similarly, r was included in the fitness function

(equation 1) so that the parameter s would define the allelic lag that placed a genotype on the threshold of extinction (i.e. with an absolute fitness of $f.w = 2$ under ideal conditions of small N) regardless of the value of r. If all genotypes in the population crossed this threshold due to the population's failure to adapt to the continuing environmental change, then extinction would follow since their absolute fitness would be too low to sustain population growth.

The simulations were used to estimate the minimum allelic cycle (T_{min}) in terms of the product $2Ku$ (= M), which defines the expected input of beneficial mutations per locus per generation in a population at its carrying capacity. M captures most of the effect of varying K and u independently (Nunney 2003). T_{min} was defined as the smallest value of T for which all replicated panmictic populations (out of 20) persisted for $16T$ generations for all $T > T_{min}$. To avoid local effects of T, once a possible T_{min} was identified, persistence was confirmed (requiring 10/10 persistent simulations) at 5% and 10% above this value. Given T_{min}, S_{min} (= T_{min} / n) defines the shortest interval between adaptive allelic substitutions consistent with long-term population persistence.

Modeling Phenotypic Plasticity

The fitness variation of any given genotype along an environmental gradient defines its tolerance curve (Lynch and Gabriel 1987; e.g., warming tolerance; Deutsch et al. 2008). Chevin et al (2010) used this approach to link tolerance curves to the reaction norms defining phenotypic plasticity, and to develop a model of adaptation in a changing environment that included developmental plasticity. This plastic response was determined by environmental conditions a short period before adulthood, and was characterized by two important features. First, the breeding value (A) of each genotype was defined by its performance in the reference environment prevailing at time $t = 0$. Second, the plastic response was proportional to the prevailing state of the environment ($E_{(t)}$), so that the plastic response became progressively larger relative to the trait value at $t = 0$ as environmental change progressed. Thus the reaction norm can be defined as:

$$z'_i = A_i + bmE_{(t)} \quad (3)$$

where the product bm is the slope of the reaction norm (see below), A_i is the zero intercept and the small developmental time (and hence environment) difference noted above are ignored.

I adopted a similar approach following the tolerance-curve/reaction-norm framework used by Chevin et al. (2010) and further developed by Lande (2014). I also assumed a linear reaction

norm; however, it was based on a slightly different assumption that has important implications for the evolutionary interpretation of the model when different levels of plasticity are compared.

The basis of the adaptive plasticity adopted in the model is illustrated in Figure 1. The genetically scaled trait value (or breeding value) of genotype i is z_i, and the environmental value that results in its maximum fitness is $E_{i,opt}$ (= z_i / (bc)), which prevails at time t_i (= Tz_i). In the absence of plasticity, the trait value of genotype i ($z'_{i,opt} = z_i/c$) is independent of the environment. Given adaptive plasticity, the trait value exhibited by genotype i shifts if $E_{(t)} \neq E_{i,opt}$ from $z'_{i,opt}$ towards a more adaptive trait value. As in the model of Chevin et al (2010), that shift is determined by a linear reaction norm which in the new model is defined as:

$$z'_i = b[E_{i,opt} + (E_{(t)} - E_{i,opt})] = b[E_{i,opt}(1-m) + mE_{(t)}] \quad (4)$$

where the slope of the reaction norm is bm, given that environmental change shifts the optimum trait at a rate b (see Figure 1).

We can now examine whether the differences between the current model (equation 4) and that of Chevin et al (2010) (equation 3) in how plasticity is modeled are likely to influence the current model's behavior:

1. For simplicity the model does not include a developmental critical period when an individual's plastic response was determined. Chevin et al. (2010) included such a delay, which is a necessary feature of plasticity; however the rate of environmental change being modeled was on a much longer time scale than a single generation. It was therefore assumed that the shift in the environment between the time of the developmental response and adulthood was negligible. This difference is unlikely to materially affect the behavior of the model.

2. The breeding value of a genotype was defined in its optimum environment rather than in a standard environment. Although it can be argued that in practice it is usual to measure genotypes under standard conditions, this can become impossible under long-term environmental change when it is likely that the tolerances of some genotypes become non-overlapping. Even when this is not the case, the elevation (and indeed the shape) of the reaction norm of genotypes measured around the limits of their tolerances may be atypical. However, this issue is a practical one that does not directly affect the behavior of the models.

3. By defining each genotype in its optimum environment, plasticity becomes proportional to environmental tolerance. The difference (Δ_i) between the trait value (equation 4) and the current optimum is:

$$\Delta_i = b[E_{i,opt}(1-m) + mE_t] - bE_t = (1-m)[z'_i - \frac{ct}{T}] \quad (5)$$

Defining plasticity by $\alpha_i = 1 / (1 - m_i)$ where $\alpha_i \geq 1$, we can substitute equation (5) in equation (1):

$$w_i = \exp\left\{-\frac{r}{s_i^2}\left[\left(\frac{1}{n}\sum_j z_{ij} - \left(\frac{t}{T}\right) + e_i\right)(1-m_i)\right]^2\right\} = \exp\left\{-\frac{r}{(\alpha_i s_i)^2}\left[\left(\frac{1}{n}\sum_j z_{ij} - \left(\frac{t}{T}\right) + e_i\right)\right]^2\right\} \quad (6)$$

so that the net effect of plasticity (α) is to increase the standard deviation of the fitness function. In doing so, plasticity directly influences environmental tolerance, defined as the effect of the environment on fitness (see Lande 2014). While this effect is largely an issue of definition, it can have an important consequence that I will now consider.

4. Defining a reaction norm centered on the genotype's optimum environment rather than basing it on a standard environment can alter the effect of new mutations. Comparing the reaction norm equations (3) and (4), it can be seen that the two models only differ in the definition of their $t = 0$ intercept, such that $A_i = b(1-m) E_{i,opt} = bE_{i,opt}/\alpha$. While this may appear to be an unimportant technical detail, it can have important consequences when new mutations arise. In the Chevin et al (2010) model, a mutation of magnitude δ in genotype i results in a breeding value of $A_i + \delta$. The effect of this mutation is to change the environment in which genotype i is best adapted, a change that depends upon the reaction norm. Specifically, the mutation causes a shift in the optimum environment of the new genotype from $\alpha Ai/b$ to $\alpha(Ai+\delta)/b$, a change of $\delta\alpha/b$, so that the effect of the mutation on the shift in the optimum environment increases with the level of plasticity, α. In the current model, this is not the case. It is assumed that a mutation of effect δ would act directly on the trait value, $z'_{i,opt}$, shifting its optimum environment by δ/b along the dashed line shown in Figure 1, i.e. it is assumed that mutational effects evaluated at a genotype's optimum are uninfluenced by plasticity. This difference between the models has the potential to affect their behavior (see Discussion).

Figure 1 shows the reaction norms of 4 genotypes, all of which, under the prevailing conditions, have the same fitness due to their equal deviation D from the optimum trait value. When $m_i = 0$ (the flat reaction norm in Figure 1), then a lag of s_i allelic cycles defines the threshold of extinction (i.e. with absolute fitness = 2 given small N, as discussed above). As m_i increases (i.e. phenotypic plasticity increases), α_i increases, and hence the width of the fitness function increases resulting in an increase in the lag tolerable without extinction. This effect of

varying plasticity (α) depends only on the product αs (see equation 6). Simulations were carried out (as described above) varying this product, and the results are documented with α expressed in terms 1/s.

Plasticity has a cost (Dewitt et al 1998), otherwise individuals would evolve to be equally fit in all environments. The cost of plasticity can take two forms. The first, which is not considered here, is the cost of an inappropriate environmental cue that results in an inappropriate (fitness reducing) phenotypic shift (Gavrilets and Scheiner 1993; Lande 2009). In the present model, the plastic response always shifts the phenotype closer to the current optimum.

The second type of cost is a fitness reduction resulting from the energetic and/or other costs of maintaining the ability to mount a plastic response. In essence, this cost reflects a trade-off between specialist and generalist strategies (e.g. Lynch and Gabriel 1987; Gilchrist 1995); as the range of environmental tolerance increases due to plasticity, the maximum height of the fitness function declines. Chevin et al (2010) included this kind of fitness cost using a weighting of their plasticity parameter. The present model also includes a direct fitness cost (w_c), which is of the form:

$$w_{ci} = (A + 1)/(A + \alpha_i) \quad (7)$$

so that fitness decreases as plasticity (α) increases and the cost (i.e. the fitness loss) of a given level of plasticity increases as the positive constant A decreases. Given equation (7), the cost of plasticity can be represented on a scale of 0 - 1 by 1/(1+A). With this plasticity cost included, we can define the expected absolute fitness of an individual female i as:

$$W_i = w_i w_{ci} f \quad (8)$$

by combining equations (2), (6), and (7), recalling that $\alpha_i s_i$ in equation (6) substitutes for s_i in equation (1).

To investigate the effect of a cost on the evolution of plasticity and adaptation in a changing environment, the parameter A was varied across simulations (A = 0, 2, 4, 8, 32, 128) in which a single locus determined plasticity. The alleles at that locus were an integer series that defined the square of the level of plasticity, α^2. Simulations used s = 1 and were initiated with 50% of the alleles at the zero plasticity level of α = 1, and the remaining 50% uniformly distributed in the integer range $2 \leq \alpha^2 \leq 11$, giving an initial average value of α = 1.80. Alleles mutated up or down one integer with the same mutation rate as the adaptive loci.

Results

1. No-cost phenotypic plasticity and the rate of adaptation.

The effect of a fixed level of no-cost plasticity on population persistence given continuous environmental change was examined assuming a single adaptive trait that was determined by between 1 and 32 genes. The goal was to investigate whether increasing plasticity increased the ability of the population to withstand rapid long term environmental change. The answer was generally a clear "no": plasticity decreased the potential for adaptation under rapidly changing conditions except when the trait being selected was determined by one (or if M was very small, two) loci of large effect (figure 2). When the number of loci was greater than two ($n > 2$), the simulation results showed a clear population-level advantage of low plasticity, since such populations could tolerate a faster rate of environmental change without extinction. For $n \geq 16$, the advantage of low plasticity ($\alpha = 1/s$) relative to the greater plasticity of $\alpha = 3/s$ was consistent, averaging about 2.3 fold across the 25-fold range of M shown (figure 2a). Thus for $n > 2$, no-cost plasticity was detrimental to long-term population persistence.

The opposite of this result occurred if the trait was controlled by a single gene of large effect, or by up to 2 loci when the rate of input of beneficial mutations was very low (i.e. below about 1 mutation per locus arising in the population every 25 generations, $M \leq 0.04$; see figure 2). Under these conditions, increasing plasticity by increasing the width of the tolerance curve increased the maximum rate of adaptation. For example, when $n = 2$, decreasing M from 0.2 to 0.008 resulted in an increase in the maximum rate of environmental change tolerated by the more plastic populations ($\alpha = 3/s$ vs. $1/s$) from roughly a 50% disadvantage to a 10% advantage (Figure 2). Examining this effect in more detail (Figure 3), it can be seen that by comparing $\alpha s =$ 0.75, 1, 2, and 3, the disadvantage of plasticity (i.e. T_{min} increasing with increasing plasticity) disappears and turns into an advantage as M decreases from $M = 1$ to 0.2 for $n = 1$, and from $M =$ 0.2 to 0.04 for $n = 2$. Some reversal was also evident (between $\alpha s = 0.75$ to 1) for $n = 4$ when M was very small ($M = 0.008$).

The measure of the cost of natural selection S_{min} ($= T_{min}/n$,), the minimum interval between adaptive substitutions consistent with population persistence, increased with plasticity for $n > 2$ (figure 2b), reflecting the effects on T_{min} noted above, i.e. plasticity decreased the maximum rate of adaptive substitution. The value of S_{min} also declined with M, and decreased to a non-zero asymptote with increasing n (Figure 2b). For example, given $M = 0.2$, when one

beneficial mutation per locus is expected in the population every 5 generations, the asymptotic maximum rate of substitution ($1/S_{min}$) was roughly 1 substitution every 14 generations with αs = 1, while increasing plasticity to αs = 3 gave an asymptotic maximum rate of 1 substitution every 33 generations. Note that both are substantially below Haldane's (1957) proposed threshold of 1 substitution per 300 generations. For multi-locus traits ($n \geq 8$), Haldane's threshold was only exceeded when M was very small (Figure 2b).

An alternative way to measure the adaptive response to environmental change is using the rate of phenotypic change (Lynch and Lande 1993; Bürger and Lynch 1995). In the simulations, the maximum rate of phenotypic change per generation was found to be proportional to the maximum rate of substitution per locus ($=1/T_{min}$) and this relationship was independent of plasticity (Figure 4). The rate of phenotypic change was generally below 10%, as predicted by the previous work, except when M was large (e.g. M = 1, αs = 1, the three uppermost open triangles in Figure 4).

2. The evolution of costly plasticity.

Simulations showed that when the cost of plasticity was low, plasticity was favored by individual selection. This point was illustrated by simulating a situation in which the rate of environmental change was 50% of the maximum (i.e. $1/T_{50}$ where $T_{50} = 2*T_{min}$) defined in the absence of plasticity (α = 1). The cost of plasticity, measured as $1/(1+A)$ (see equation 7), was varied between 0.01 (A = 128) and 1.0 (A = 0). Plasticity ($\alpha > 1$) was inevitably favored whenever the cost was below either 0.11 or 0.20, depending on M and the number of loci (Table 1). For example, when cost was its lowest (= 0.01), the value of α increased in all simulations from its initial average value of 1.80 to a value ranging from 2.57 to 4.27 after 12 allelic cycles (Table 1).

The effect of increasing plasticity was generally to reduce the ability of the population to track the environment through genetic adaptation. This effect was quantified using the observed lag in allelic substitutions (Table 1). When plasticity had a negligible cost, an appreciable lag developed over the 12 allelic cycles of the simulations whenever n was sufficiently large. For example, when M = 0.2 and n = 16, the lag was 3.48, i.e. the average genetic value of the adaptive trait in the population was 8.52 instead of 12. In contrast, when M = 0.04 and n = 1, plasticity increased when the cost was low, but the environmental tracking remained perfect (Table 1).

3. Individual vs. population level advantage.

In the previous section it was shown that, except when the number of loci was small, the spread of plasticity resulted in an increased genetic lag. To further examine the consequences of the spread of plasticity, the allelic cycle was reduced from $2x T_{min}$ to $1.25x T_{min}$, i.e. the rate of environmental change was set at 80% of the maximum consistent with long-term population persistence in the absence of plasticity ($\alpha = 1$). The results showed that the spread of plasticity can lead to extinction, and that this effect is strongest when (a) the flow of beneficial mutations (M) is high and (b) the number of loci determining the adaptive trait is large (Table 2). Thus when $n = 1$, plasticity increased to some limit and environmental tracking remained good even when the cost of plasticity was low. For example, for $M = 0.2$ and a low cost of plasticity (= 0.01) , the lag after 12 cycles averaged 0.96 (Table 2), and after 36 cycles it averaged 0.97 while the plasticity value (α) increased from 3.43 to 3.73. On the other hand, with the same parameters except for more loci controlling the trait ($n = 16$), populations were extinct after an average of 6.2 allelic cycles when a very similar plasticity had evolved ($\alpha = 3.56$).

Discussion

Simulations investigating the interplay between adaptive evolution and plasticity under conditions of continuous environmental change showed several important results. First, as would be expected, very low cost plasticity was always individually favored and spread in the population. It eventually equilibrated at some limit, balanced by the cost. Second, when there was at least a moderate number of loci controlling the adaptive trait (generally $n > 2$), the long-term adaptation of a population was slowed by plasticity, and this slowing of adaptation could drive a population to extinction. Third, the maximum rate of phenotypic change was directly proportional to the maximum rate of substitution per locus ($1/T_{min}$; see Figure 4), and this relationship was independent of the level of plasticity.

In the absence of phenotypic plasticity, we expect that, provided the environmental change is not too extreme or too rapid, species will generally adapt (Lynch and Lande 1993; Bürger and Lynch 1995; Gomulkiewicz and Holt 1995; Lande and Shannon 1996; Stockwell *et al*. 2003). Haldane (1957) highlighted the classic case of industrial melanism in the peppered moth (see Cook 2003), but there are now many examples of rapid evolution in natural populations (see Hendry and Kinnison 1999; Reznick and Ghalambor 2001; Rice and Emery 2003; Hairston *et al.* 2005). For quantitative traits, the rate of adaptation can be expressed in terms of phenotypic

standard deviation per generation, and Bürger and Lynch (1995) calculated that the maximum sustainable rate of evolution for such a trait was about 10% of a phenotypic standard deviation per generation, noting that other factors might reduce this closer to 1%.

Given this previous modeling of adaptation and environmental change based on phenotypic measures, it was important to establish that the results of the present model were not affected by the shift to measures of genetic change. The simulation results were indeed consistent with the prior work, with a maximum phenotypic change of about 10%; the fastest sustainable rates of substitution occurred with a high input of beneficial mutations ($M = 1$) when the rate of phenotypic change reached 10-15% per generation (Figure 4). In addition, the rate of phenotypic change was proportional to the maximum rate of substitution per locus (the reciprocal of the allelic cycle, $1/T_{min}$) and not to the overall rate of substitution ($1/S_{min}$). This was expected because the allelic cycle (T) reflected the overall strength of selection, since the fitness effect per locus decreased proportionally as the number of loci increases (see equation 1). The slope of the relationship deviated only slightly from the expectation of 1 (= 0.935), due to a curvature in the relationship when the rate of phenotypic change was very high (around 10% per generation).

Selection over short periods typically exploits pre-existing genetic variation, whereas longer term evolution increasingly depends on the accumulation of new mutations (Barton and Keightley 2002). However, a major concern is that if environmental change drives a high rate of evolution over a period longer than about 15-20 generations (which is certainly likely given global warming), then genetic variability would erode and the probability of extinction would increase (Hendry and Kinnison 1999). The results of Bürger and Lynch (1995) support this view. By incorporating the stochastic loss and gain of genetic variation, they showed that the resulting increase in the variance of the adaptive response reduced the maximum rate of phenotypic evolution by at least an order of magnitude compared to the earlier estimate of Lynch and Lande (1993).

Given this background, there has been substantial interest over the last few years concerning the role of plasticity in responding to climate change (Gienapp et al. 2008; Hendry et al 2008; Visser 2008) and the empirical data support a strong involvement of plasticity in the response to climate change (Merila and Hendry 2013). Theory has also been developed to predict how plasticity effects adaptation. For example, the models of Chevin and Lande (2009) and Lande (2009) demonstrate how plasticity is beneficial in enabling a population to survive and then

adapt to an abrupt environmental change. However, the outcome is less clear when the environmental change is continuous over many generations. Chevin et al. (2010) found that plasticity was always beneficial for population persistence, while in the present analysis, under most conditions, the exact opposite was observed. The difference probably arose from the manner in which phenotypic plasticity was built into the models. As noted earlier, Chevin et al. (2010) considered a plastic response that was defined relative to the breeding value of a genotype evaluated in some standard environment (set as the environment at $t = 0$). The plastic response provided a boost to the phenotype proportional to the recent state of the environment. As noted by the authors, this proportional response appeared to compensate for any plasticity-related drop in the effectiveness of natural selection acting to adapt to environmental change.

In apparent contrast, the model used here assumed that a genotype's plastic response was proportional to the difference between the current state of the environment and the environmental state optimal for the genotype. However, both models can be expressed in the same terms (see equations 3 and 4). The only difference is that the intercept (at $t = 0$) in the model of Chevin et al (2010) is a genotype-dependent constant (A_i) whereas in the present model it is the product of the genotype-dependent constant ($E_{i,opt}$) and (1-m) (= the reciprocal of plasticity $1/\alpha$). As described earlier, a mutation that increases the genotype by a fixed trait value (δ) has a different effect in the two models. In the present model, a shift in optimum environment resulting from the mutation is always δ/b (i.e. a shift along the dashed line shown in Figure 1), because the genotypic value (and hence the effect of a mutation) is defined in its optimum envionment. In the Chevin et al (2010) model the equivalent shift is $\alpha\delta/b$, because the genotypic value (and hence the effect of a mutation) is defined at $t = 0$ so that a mutation's effect is amplified by the slope of the reaction norm in defining where it intersects the line defining the trait/environment optimum. The result is that in their model, as plasticity increases, mutations have a larger effect in tracking the environmental optimum and therefore likely to promote adaptation. This amplification of mutational effects with increasing plasticity is likely to be driving the favorable effect of plasticity on long-term adaptation found by Chevin et al (2010). Thus the biological issue distinguishing the models appears to be whether adding an average mutation to a genotype typically results in a shift in the optimum environment of the genotype that is independent of plasticity or that increases with plasticity. If the shift is independent of

476 plasticity then the current model indicates that increasing plasticity can be detrimental to a
477 population over the long term.
478 In both the present model and that of Chevin et al (2010), plasticity ensured that fitness was
479 maintained over a larger range of environmental conditions, and, not surprisingly, it has been
480 shown here that this ability to maintain fitness is always individually advantageous when there is
481 zero cost. In the present model, increasing plasticity increased a genotype's tolerance, but it also
482 resulted in an increasing population-level lag in adaptation as the rate of environmental change
483 increased, indicating a drop in the effectiveness of natural selection. Thus over the long-term,
484 plasticity was disadvantageous. The only exception was found when adaptation relied on one or
485 two loci of large effect.
486 Why was plasticity found to be advantageous when the number of loci (n) determining the
487 adaptive trait was small? The reason is almost certainly bet hedging. When n is large, the
488 variance in the flow of beneficial mutations is much less than when only one or two loci are
489 involved. Thus while plasticity may still impose a cost on the effectiveness of natural selection
490 when $n = 1$, there will be times when the waiting time between mutations is unusually high. If
491 this happens when the environment is changing rapidly, the population will lack the variation to
492 adapt and will decline to extinction unless individuals exhibit substantial plasticity, enabling
493 them to survive this atypical (but inevitable) period. A similar effect due to the stochastic nature
494 of mutation was observed in the model of Bürger and Lynch (1995).
495 It is expected that plasticity has a cost. (DeWitt 1998). The cost of responding to
496 inappropriate environmental cues was not considered here; however, the possibility of a
497 continuing fitness cost due to the need to maintain the ability to mount a plastic response was
498 included. Chevin et al (2010) showed that when plasticity has such a cost there is a threshold
499 value above which the population would go extinct if plasticity ever became that high; however
500 their model did not consider the possibility of plasticity itself evolving. In the current model,
501 when plasticity was free to evolve, plasticity typically increased to an intermediate optimum,
502 and, if the rate of environmental change was initially close to the extinction threshold of a
503 population, then the evolution of plasticity could drive the population to extinction (Table 2).
504 This sets up an interesting group selection scenario (*sensu* Nunney 1985) with individual
505 selection acting to increase plasticity, but with population-level selection acting on the emergent
506 property of extinction to decrease it. However, it is very unlikely that the population-level

507 selection would ever be successful at suppressing the individual effect (see Nunney 1999). As a
508 result we are left with the likelihood that given directional environmental change, individual
509 selection will favor increased plasticity and that as a general rule selection for this form of
510 plasticity will make the population more vulnerable to extinction.
511 This extinction effect adds to a number of possibilities whereby it is possible for adaptive
512 evolution to promote "Darwinian extinction" (Webb 2003). However, the effect observed here is
513 distinct from the examples usually identified. Some of these situations, such as the "ecological
514 traps" discussed by Schlaepfer et al (2002) result from abrupt environmental changes that create
515 maladaptation, which, given enough time, would be resolved by further adaptive change, Most
516 other examples involve frequency-dependent selection, with genotypes interacting to determine
517 fitness (Parvinen 2005; Rankin and Lopez-Sepulcre 2005), such as competition for some form of
518 limited resource. In the present case, the spread of plasticity is a response to a gradually changing
519 environment which does not directly affect on the fitness of others in the population; instead, it
520 reduces the effectiveness of another population-level process, genetic adaptation. An more
521 analogous effect appears to be the adaptive reduction of dispersal in a metapopulation tha can,
522 under some conditions, lead to extinction (Gyllenberg et al 2002).
523 The detrimental effect of increasing plasticity is of particular concern in small populations
524 which inevitably have small values of M and hence vulnerable to rapid long-term environmental
525 change. Assuming a high beneficial mutation rate of 10^{-5} /locus /generation, a population with a
526 carrying capacity of 2000 has $M = 0.04$. For example if an adaptive trait is determined by 16 loci,
527 when plasticity is low ($\alpha s = 1$) the maximum rate of substitution is one substitution per 52
528 generations; however when plasticity is higher ($\alpha s = 3$) the maximum rate is more than halved to
529 one substitution per 116 generations. Thus if plasticity generally evolves by broadening the range
530 of environmental conditions that individuals can tolerate, then the possibility that the evolution
531 of increased phenotypic plasticity may increase extinction risk should be considered in the
532 environmental planning process. In particular, it may require maintaining larger populations of
533 threatened species.

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Table 1: Adaptation and the spread of plasticity as a function of the cost of plasticity when the rate of environmental change is 50% of the maximum consistent with population persistence (i.e. at a rate of $1/T_{50}$ where $T_{50} = 2T_{min}$ defined for $\alpha = 1$). The values shown are the lag in allelic substitutions and (in parentheses) the average value of plasticity (α) after 12 allelic cycles of environmental change (i.e. $12T_{50}$ gens). The cost of plasticity was defined as $1/(1+A)$, so the maximum cost is 1.00 when $A = 0$ (see eqn. 4). For all simulations, $s = 1$.

			$M = 0.04$			
cost of plasticity (T_{50})	0.01	0.03	0.11	0.20	0.33	1.00
1 locus (1140 gens)	0.00 (2.57)	0.00 (1.77)	0.00 (1.40)	0.00 (1.00)	0.00 (1.00)	0.00 (1.00)
4 loci (732 gens)	0.29 (2.45)	0.32 (1.73)	0.17 (1.00)	0.13 (1.00)	0.08 (1.00)	0.13 (1.00)
16 loci (1160 gens)	1.20 (3.13)	1.07 (2.91)	0.08 (1.00)	0.13 (1.00)	0.11 (1.00)	0.11 (1.00)
			$M = 0.2$			
cost of plasticity (T_{50})	0.01	0.03	0.11	0.20	0.33	1.00
1 locus (190 gens)	0.56 (3.33)	0.89 (3.20)	0.00 (1.49)	0.00 (1.00)	0.00 (1.00)	0.00 (1.00)
4 loci (178 gens)	2.39 (3.65)	2.18 (3.56)	0.32 (1.37)	0.25 (1.00)	0.18 (1.00)	0.17 (1.00)
16 loci (510 gens)	3.48 (4.27)	1.59 (3.11)	0.20 (1.00)	0.17 (1.00)	0.20 (1.00)	0.18 (1.00)

Table 2: The spread of plasticity driving extinction when the rate of environmental change is 80% of the maximum consistent with population persistence when $\alpha = 1$ (i.e. $1/T_{80}$). The values shown are the lag in allelic substitutions and (in parentheses) the average value of plasticity (α) after 12 allelic cycles of environmental change (i.e. $12T_{80}$ gens). "Extinct": all 6 simulations went extinct within 12 cycles. If this was not the case, but extinction was 100% by 60 cycles, then the percent extinction after 12 cycles is shown. The cost of plasticity was defined as $1/(1+A)$, and $s = 1$ throughout.

cost of plasticity			$M = 0.04$			
(T_{80})	0.01	0.03	0.11	0.2	0.33	1
1 locus (713 gens)	0.03 (2.71)	0.00 (1.87)	0.00 (1.38)	0.00 (1.00)	0.00 (1.00)	0.00 (1.00)
4 loci (458 gens)	1.84; 33% (3.09)	1.41; 0% (2.49)	0.32 (1.29)	0.20 (1.00)	0.07 (1.00)	0.25 (1.00)
16 loci (1038 gens)	extinct	extinct	1.19; 33% (2.18)	0.21 (1.00)	0.16 (1.00)	0.20 (1.00)
cost of plasticity			$M = 0.2$			
(T_{80})	0.01	0.03	0.11	0.2	0.33	1
1 locus (119 gens)	0.96 (3.43)	0.88 (3.30)	0.03 (1.73)	0.00 (1.05)	0.00 (1.00)	0.00 (1.00)
4 loci (111 gens)	extinct	extinct	extinct	1.11; 17% (2.00)	0.30 (1.00)	0.21 (1.00)
16 loci (319 gens)	extinct	extinct	extinct	extinct	0.27 (1.00)	0.27 (1.00)

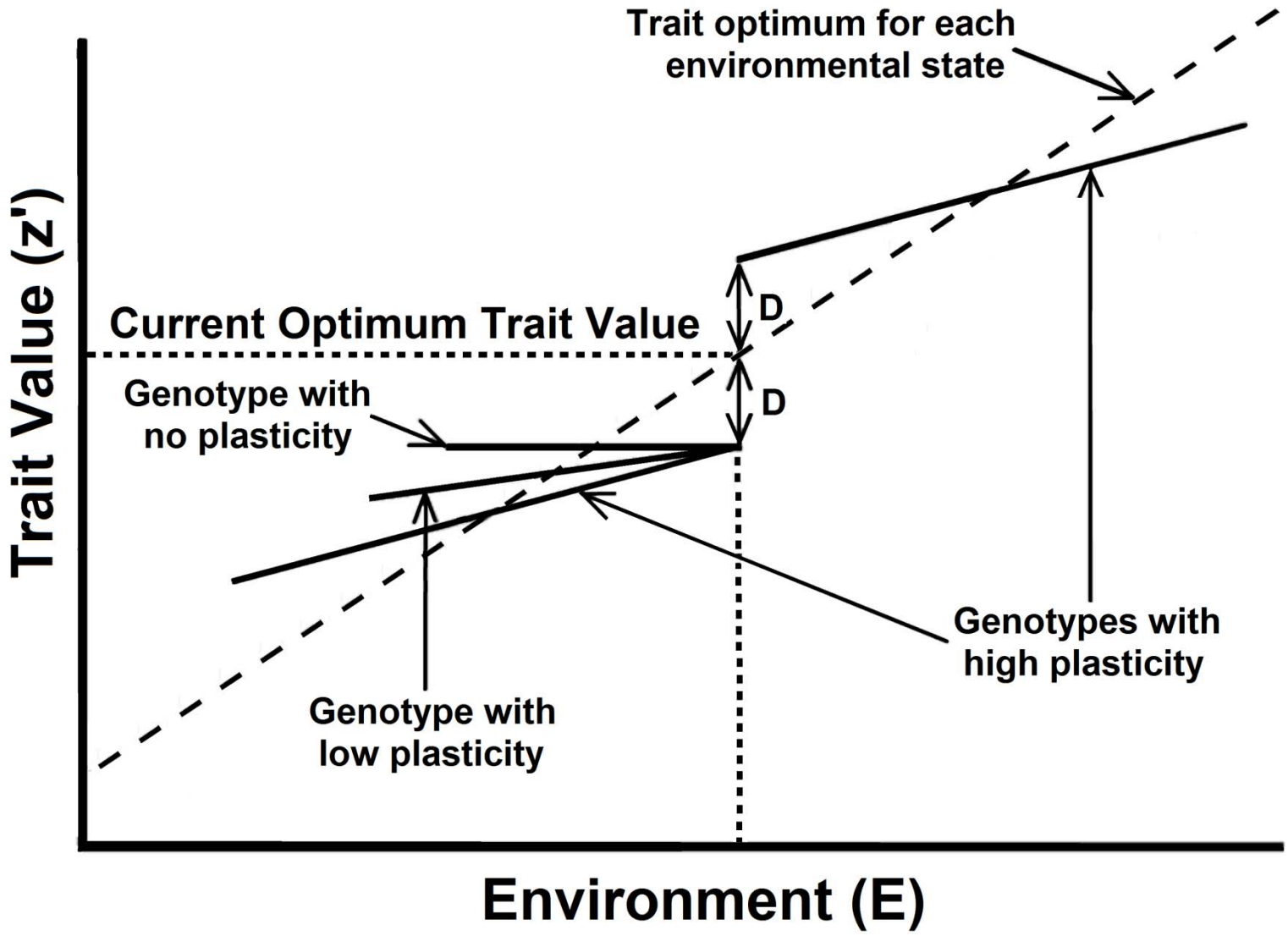
Figure Legends.

Figure 1: Modeling phenotypic plasticity in a changing environment. The reaction norms of four genotypes are shown, with each reaction norm (with slope mb) centered at the genotype's fittest phenotype, which is placed on the dashed line (slope b) that defines ideal genotype-environment adaptation. The current state of the environment (vertical dotted line) defines the current optimum trait value. All four genotypes are equidistant (D) from that optimum, and therefore have the same fitness under the prevailing conditions (excluding any costs of plasticity). The genotype lacking plasticity (horizontal line, i.e. $m = 0$) has a fixed trait value, and consequently has a narrow range of conditions within which it less than D from the optimum (the length of the line), while the two genotypes with high plasticity have a much wider equivalent range. The two high plasticity genotypes have equal fitness under the prevailing conditions shown, even though one is optimally adapted at a lower environmental state and the other at a higher state.

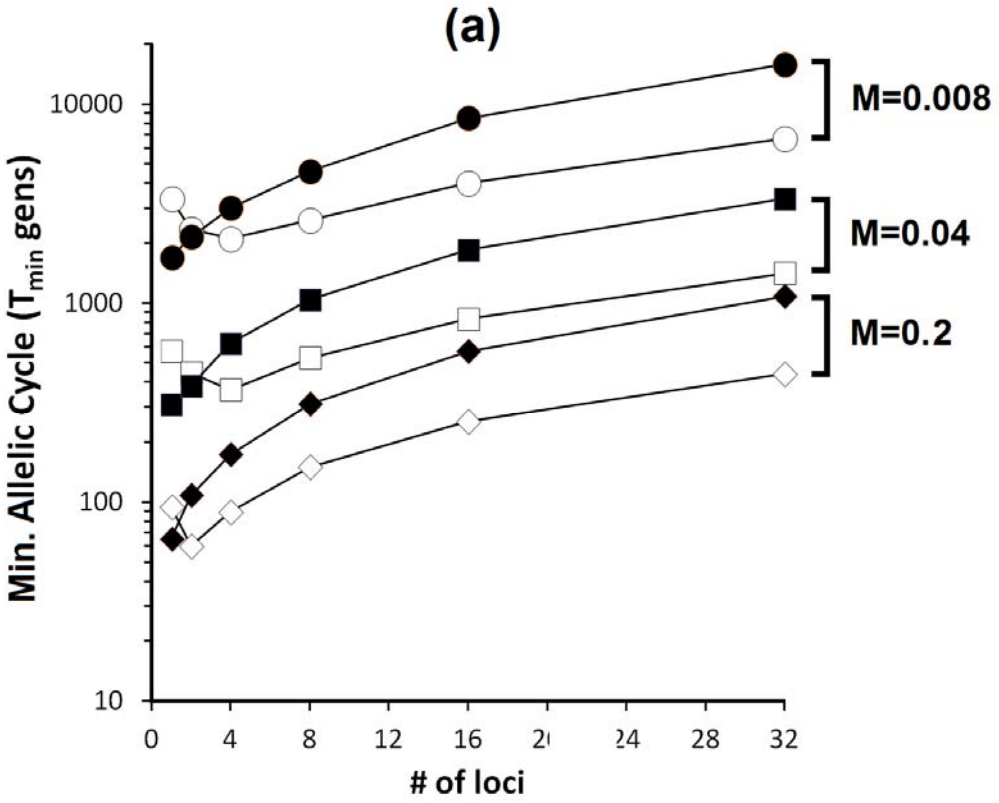
Figure 2: Minimum interval between allelic substitutions compatible with population persistence with and without adaptive plasticity given different numbers of loci (n) and different numbers of beneficial mutations expected to arise in the population /locus/generation, M (= $2Ku$). Open symbols: low plasticity, $\alpha = 1/s$; Closed symbols: high plasticity, $\alpha = 3/s$. (a) The minimum allelic cycle (T_{min}) on a log scale. (b) The same data shown on a linear scale as the minimum interval between substitutions (S_{min}). M was varied by altering K (carrying capacity) with u (beneficial mutation rate) = 10^{-5}. Each data point was based on >>100 simulations (see Methods).

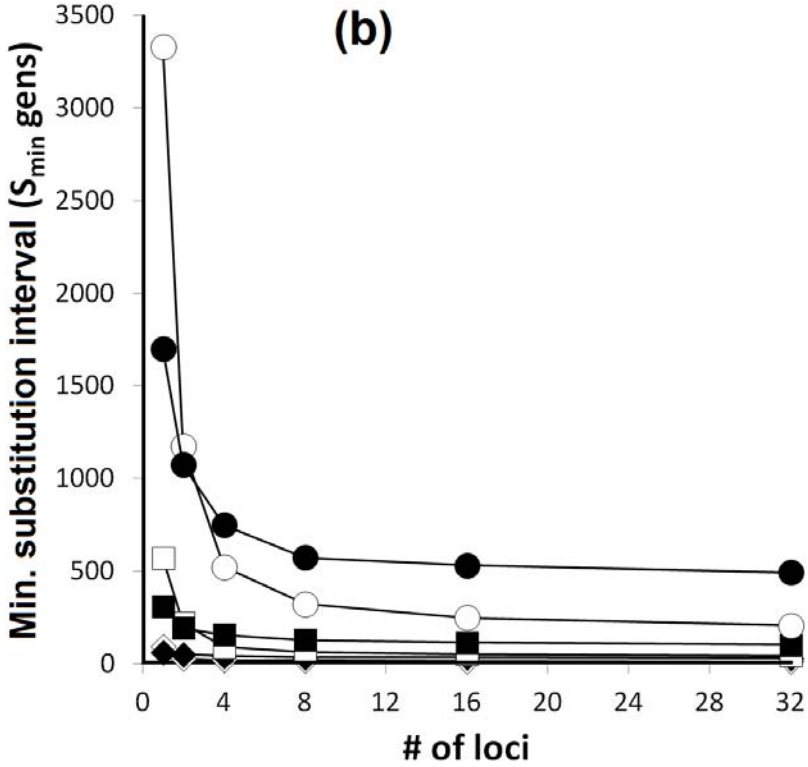
Figure 3: Transition of the effect of plasticity (s) from retarding to accelerating population adaptation as the number of adaptive loci (n) decreases and M is small. Plasticity levels (from lowest to highest): striped symbols, $\alpha = 0.75/s$; open symbols, $\alpha = 1/s$; grey symbols, $\alpha = 2/s$; and black symbols, $\alpha = 3/s$. Relative to figure 2, data added for $M = 1$, and for plasticity levels $\alpha = 0.75/s$ and $2/s$.

Figure 4: Relationship between the maximum rate of environmental change consistent with population persistence and the resulting rate of phenotypic change. The data are shown on a log scale with a reference line of slope 1, which defines direct proportionality. The least squares slope is 0.935 ± 0.40 (95% confidence). Data are from the simulations shown in Figure 2a, plus for $M = 1$ ($s = 1$ and 3) from Figure 3. The symbols are defined as in earlier figures.

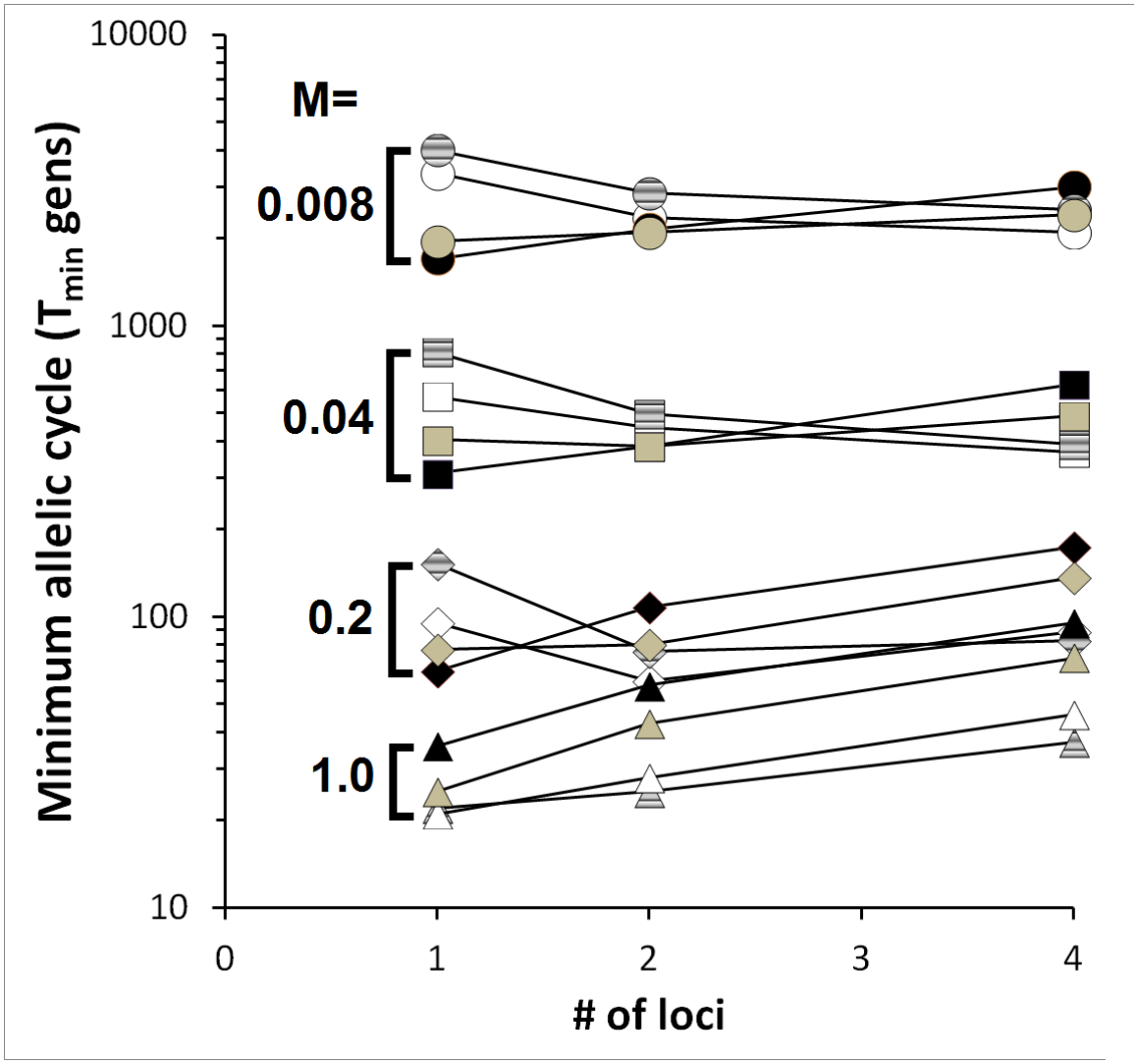

Trait optimum for each
environmental state
Trait Value (z')
Current Optimum Trait Value
D
D
Genotype with
no plasticity
Genotypes with
high plasticity
Genotype with
low plasticity
Environment (E)

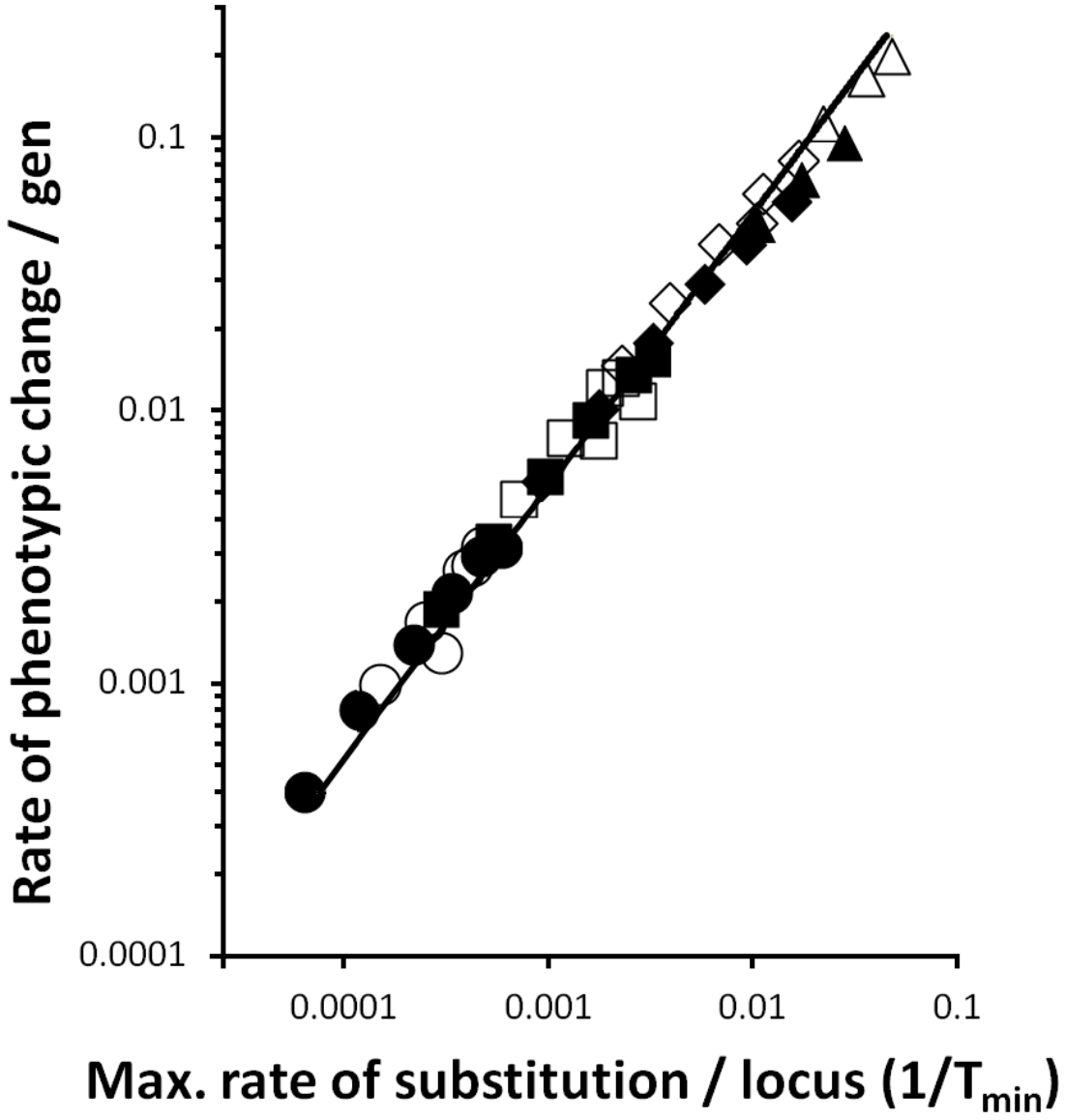
2


(a)
Min. Allelic Cycle (T_{min} gens)
10000
1000
100
10
0
4
8
12
16
20
24
28
32
# of loci
M=0.008
M=0.04
M=0.2


(b)
Min. substitution interval (S_{min} gens)
3500
3000
2500
2000
1500
1000
500
0
0
4
8
12
16
20
24
28
32
# of loci

2


10000
1000
100
10
Minimum allelic cycle (T_{min} gens)
M=
0.008
0.04
0.2
1.0
0
1
2
3
4
# of loci


Rate of phenotypic change / gen
0.1
0.01
0.001
0.0001
0.0001
0.001
0.01
0.1
Max. rate of substitution / locus (1/T_min)

2