Electronic Supplementary Material

A heuristic model on the role of plasticity in adaptive evolution: plasticity increases adaptation, population viability and genetic variation

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Model details
— mean-mutation-change

Mutation rate is often used to describe the probability of change per base-pair per generation, and mutations affecting quantitative trait loci are typically low, ranging from $10^{-9}$ to $10^{-6}$ [1, 2]. Mutation rate, however, varies widely not only among different organisms [3], but among populations [3], sexes within populations [4] and among genomic regions [5, 6]. Moreover, single nucleotide changes in protein-coding sequences are but one of many possible genetic changes of evolutionary importance, because a large fraction of heritable phenotypic changes can be due to changes in regulatory sequences, epimutations, or even changes in the state of gene networks [7-10]. In fact, whereas phenotypic variants can sometimes be traced back to unique changes in the sequence of protein-coding genes [11, 12], they are often instead mediated by changes in regulatory sequences [13] or epigenetic modifications [8, 14, 15]. However, deciding the relative importance of these sources of heritable phenotypic variation to the evolutionary process is well beyond the scope of this study and is not critical for testing our hypotheses.

Instead, we have jointly modelled the probability of a genetic change, and its effect size on the phenotype, and we have called it mean-mutational-change. In this way, mean-mutational-change is a parameter used to simulate the magnitude of mutational change on the genotype (for plastics and non-plastics) and on the plasticity-range (for plastics only). This approach is more inclusive of genetic sources of variation and only assumes that regardless of the nature of the genetic change occurred (single base-pair substitution, recombination, addition/deletion of transcription factors, epimutation, etc), genetic changes causing small phenotypic effects are more likely than genetic changes causing large phenotypic effects. This is in accordance with the literature on quantitative trait loci (QTLs), where a few QTL of large effect and an increasing number of additional modifier QTLs of smaller effects are commonly
reported [16, 17]. It is also in accordance with Fisher’s geometric model of mutational effect sizes [18].

Consequently, we used a negative exponential function with mean \( \text{mean-mutational-change} \) to describe the probability of occurrence of genetic changes of a certain size. This distribution captures the idea that larger effects of genetic mutation upon the resulting phenotype are very rare. This is calculated as \((\text{mean-mutational-change}) \ast \ln(x)\), where \(x\) is a floating point pseudorandom number between 0 and 1.

Upper figure shows the resulting histogram for \( \text{mean-mutational-change} = 0.025 \), and lower figure shows the same for \( \text{mean-mutational-change} = \)

\[ 0.05 \]

--- plasticity-costs
The evolution of plasticity is thought to be dependent upon costs of plasticity, which could be of very different nature. The most general notion of costs of plasticity is that of maintenance costs, i.e. energetic costs associated to the maintenance of sensorial mechanisms and regulatory mechanisms enabling the potential for changing the phenotype according to the perceived environment [19, 20]. In addition to maintenance costs, plastic genotypes are also expected to incur in production costs or costs incurred during the process of phenotypic change that exceed those experienced by genotypes of fixed phenotypes. In our view these two are the two most general costs of plasticity that can be expected, and would apply to all kinds of phenotypic plasticity, regardless of the trait involved, the type(s) of cue(s) triggering the phenotypic change or the ontogenetic stage of the organism. Discussion of the existence and relative importance of more particular cases of costs and limits of plasticity can be found in [19-21]. In our model, we introduced maintenance and production costs of plasticity. Once the phenotype is developed, individuals have a mortality probability according to their realized match to the environment (see 'die-by-mismatch?' in main text). Costs intervene because plastics have two additional potential mortality causes: costs of maintaining a given plasticity-range and the costs of the plasticity-used (see 'die-by-plasticity-costs?' in main text).

Literature cited


