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Proc. R. Soc. B 2013 **280**, 20131869, published 25 September 2013

PROCEEDINGS OF THE ROYAL SOCIETY B

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Cite this article: Gomez-Mestre I, Jovani R. 2013 A heuristic model on the role of plasticity in adaptive evolution: plasticity increases adaptation, population viability and genetic variation. Proc R Soc B 280: 20131869. http://dx.doi.org/10.1098/rspb.2013.1869

Received: 18 July 2013
Accepted: 2 September 2013

Subject Areas:
computational biology, developmental biology, evolution

Keywords:
adaptation, developmental plasticity, genetic accommodation, heterogeneous environment, bottleneck, genetic variation

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Electronic supplementary material is available at http://dx.doi.org/10.1098/rspb.2013.1869 or via http://rspb.royalsocietypublishing.org.

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

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An ongoing new synthesis in evolutionary theory is expanding our view of the sources of heritable variation beyond point mutations of fixed phenotypic effects to include environmentally sensitive changes in gene regulation. This expansion of the paradigm is necessary given ample evidence for a heritable ability to alter gene expression in response to environmental cues. In consequence, single genotypes are often capable of adaptively expressing different phenotypes in different environments, i.e. are adaptively plastic. We present an individual-based heuristic model to compare the adaptive dynamics of populations composed of plastic or non-plastic genotypes under a wide range of scenarios where we modify environmental variation, mutation rate and costs of plasticity. The model shows that adaptive plasticity contributes to the maintenance of genetic variation within populations, reduces bottlenecks when facing rapid environmental changes and confers an overall faster rate of adaptation. In fluctuating environments, plasticity is favoured by selection and maintained in the population. However, if the environment stabilizes and costs of plasticity are high, plasticity is reduced by selection, leading to genetic assimilation, which could result in species diversification. More broadly, our model shows that adaptive plasticity is a common consequence of selection under environmental heterogeneity, and hence a potentially common phenomenon in nature. Thus, taking adaptive plasticity into account substantially extends our view of adaptive evolution.

1. Introduction

Understanding the mechanisms of adaptation is the key to understand how life on the Earth has persisted over widely varying environmental conditions resulting in the observed biodiversity, and to understand how organisms would adapt to current global change. Adaptive evolution requires heritable phenotypic variation for selection to act upon, and the standing paradigm that emerged from Modern Synthesis argued that random genetic mutations of fixed phenotypic effects are the only source of heritable phenotypic variation fuelling adaptive evolution [1–3]. Under this scenario, mutations accumulate in populations through various combinations of recurrent mutation, drift, recombination, immigration and selection in heterogeneous environments [4–6]. Selection then acts on this standing genetic variation producing adaptations, and hence the environment acts merely as a sieve for phenotypes.

Nevertheless, there is now ample evidence showing that the environment can also act as a phenotypic inducer so that a single genotype is often capable of expressing alternative appropriate phenotypes in response to different environments [7–9]. This phenotypic plasticity is the consequence of environmentally induced changes in gene expression [10]. Plasticity is often heritable, and it evolves under selection if environmental cues are reliable and gene flow is high among subpopulations [11,12]. Conversely, local adaptation and reduced

plasticity occur when dispersal is low [11] or environmental variation is unpredictable or negligible [13,14].

Extending the paradigm to include adaptive plasticity is a necessary step in evolutionary biology to extend our understanding of the mechanisms of adaptive evolution [15], and there has been a surge of interest in characterizing the evolutionary consequences of environmentally induced variation [16–18]. Previous theoretical studies have greatly contributed to our understanding of different aspects of the evolution of plasticity under particular scenarios, often using complex quantitative genetic models [19–22]. These models have shown that plasticity is advantageous in rapidly changing environments and that it may help colonizing new environments [22], although genetic correlations and costs of plasticity could limit these benefits of plasticity [23,24].

Adaptive plasticity can also result in evolutionary innovations [18]. If sister lineages evolve independently in different stable environments and ancestral plasticity is costly, divergent reaction norms are expected to evolve through selection on genetic modifiers available in the population [2,7,25]. This would lead to genetic accommodation of environmentally induced phenotypes, i.e. adaptive genetic changes in response to selection on the regulation and form of the phenotype [7]. Fixed-effect genes (i.e. not sensitive to environmental input) giving rise to phenotypes with increased fitness in the new environment will be positively selected, and the trait will become genetically assimilated, a particular case of genetic accommodation [7,26]. Thus, whether resulting in novel or canalized phenotypes, or simply in divergent reaction norms, developmental plasticity can foster speciation and diversification [17,27]. Genetic accommodation and assimilation of plasticity have been experimentally demonstrated [28–30] and also inferred from comparative analyses [31,32]. Plasticity is thus a common feature of organisms that is favoured by selection precisely under the same circumstances that maintain standing genetic variation, namely environmental heterogeneity and gene flow among subpopulations [11]. However, historically there has been some reluctance to recognize the importance of phenotypic plasticity in evolution [3,9,21,33,34]. Perhaps simple heuristic models may help illustrating the potential of plasticity in evolution while avoiding the so-often black-box feeling of complex models.

Here, we built and analysed a simple heuristic individual-based model comparing adaptive evolution in populations composed of either plastic or non-plastic genotypes. We examine how adaptive plasticity evolves under common scenarios assumed to maintain non-environmentally dependent standing genetic variation, and then examine how plasticity affects adaptive evolution because of the role of the environment as a phenotypic inducer. We simulated population dynamics under contrasting combinations of environmental stochasticity, occurrence of genetic changes, levels of plasticity and costs of plasticity. We specifically explored the conditions under which genetic assimilation occurs, and the relationship between plasticity and standing genetic variation. There is also evidence that in some organisms epigenetic marks allow induced phenotypes themselves (and not just the ability to produce them) to be inherited across multiple generations [35,36], but that is not the scope of this study. Here, we focus only on plastic genotypes that inherit the ability to produce different adaptive phenotypes according to perceived environmental cues.

We used the model to test the following predictions: (i) during rapid environmental change or when facing a novel environment, plasticity improves the persistence of populations and reduces the severity of bottlenecks; (ii) plasticity contributes to the maintenance of standing genetic variation within populations; (iii) by increasing population persistence and maintaining genetic variation, plasticity 'buys time' for appropriate genetic variants of fixed phenotypic effect to appear by mutation and (iv) costs of plasticity result in genetic assimilation (i.e. loss of plasticity) if heterogeneous environments stabilize.

2. The model

This model description follows the Overview, Design concepts and Details protocol for describing individual- and agent-based models [37–39]. The model is implemented in NetLogo v. 5.0.3 [40] (NetLogo is freely downloadable from http://ccl.northwestern.edu/netlogo/download.shtml) and available in the electronic supplementary material (Model.txt).

— *Purpose.* The main purpose of the model is to explore the consequences of phenotypic plasticity in adaptive evolution. This is done by simulating population persistence and genetic evolution under environmental change. Simulations are run separately for *non-plastics* and *plastics*. *Non-plastics* evolve by selection on random genotypic mutations with fixed phenotypic effects. *Plastics* evolve exactly in the same way, but also through selection on mutations conferring phenotypic plasticity (figure 1)

— *Entities, state variables and scales.* Environmental conditions are simulated by the variable *environment*. The entities of the model are asexual individuals of two kinds: either *non-plastics* or *plastics*. Each individual has a given *genotype* and a *phenotype*. *Plastics* also have a *plasticity-range* that allows them to improve their match with the *environment*. The *match* is an individual variable calculated as 1 - |*phenotype* - *environment*|, which shapes individual survival and reproduction (see below). The amount of *plasticity-range* used by the individual to improve its phenotypic match with the environment is the *used-plasticity*. For instance, a *genotype* of 0.7 in an *environment* of 0.8 with a *plasticity-range* of 0.2 would only need to use 0.1 of its *plasticity-range* to produce a perfectly matching *phenotype* (i.e. *used-plasticity* = 0.1). Thus, while *plasticity-range* is an inherited trait of the individual, *plasticity-used* is a value recorded by the model when the individual develops. One time step of the model corresponds to one generation, and generations are non-overlapping. See table 1 for variable definitions and range of parametrized values.

— *Process overview and scheduling*. See a schematic diagram in figure 1. At birth, individuals inherit from their parent's a *genotype* and (if *plastics*) a *plasticity-range*. Both genetic features mutate in the same way (see '*mutation*' below). *Non-plastics* develop a *phenotype* equal to their *genotype*. *Plastics*, however, use their *plasticity-range* to fit their *phenotype* as much as possible to the *environment* (see '*development*' below). *Non-plastics* and *plastics* have a mortality probability according to their realized *match* to the *environment* (see '*die-by-mismatch?*' below). Subsequently, they can die by negative density-dependence (see '*die-by-negative-density-dependence?*' below). Moreover, *plastics* could die by costs of maintaining a given *plasticity-range* and the costs of the *plasticity-used* (see '*die-by-plasticity-costs?*'

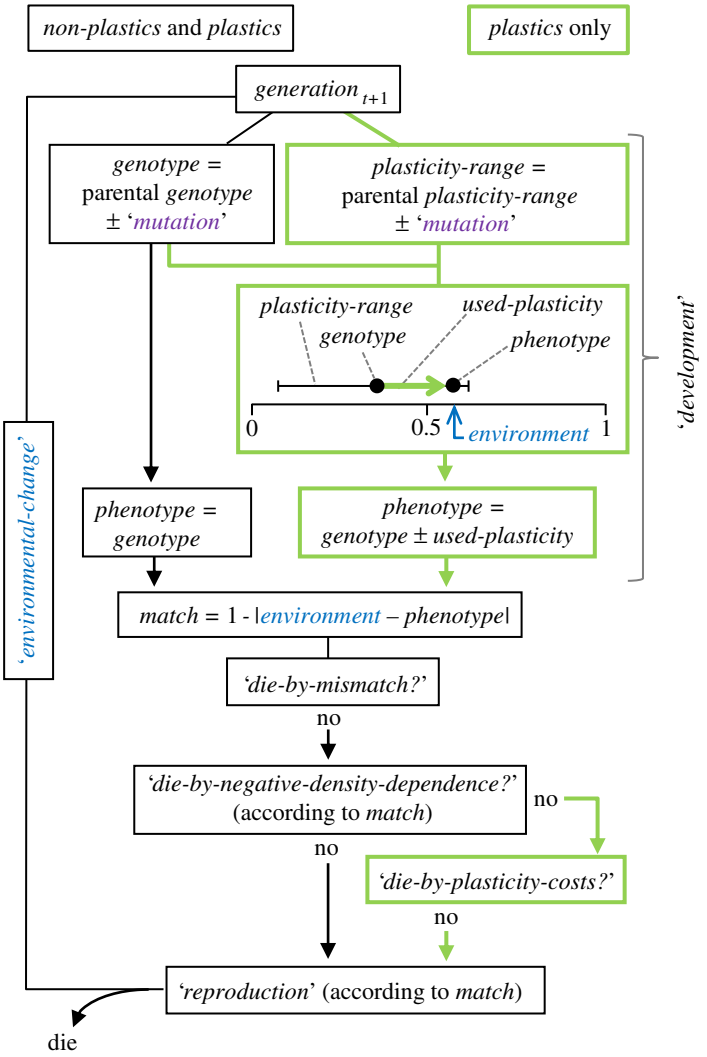

Figure 1. Schematic of the individual-based model comparing adaptive evolution in populations composed of *plastic* or *non-plastic* genotypes. They are all clonal organisms with no recombination so that *non-plastic* genotypes map directly into phenotypes and their odds of surviving and reproducing depend on the *match* with the *environment*. By contrast, *plastic* genotypes can respond to the *environment* modifying their *phenotype* to reduce the mismatch to the extent that their plasticity-range allows. In both cases, the *environment* acts as a selective factor, but for *plastic genotypes* it is also a phenotypic inducer. (Online version in colour.)

below). These two costs of plasticity are commonly identified in the literature on developmental plasticity as 'maintenance costs' and 'production costs' and correspond to the presumed costs of maintaining a sensory machinery and actually producing alterations on the phenotype, respectively [23,24]; see the electronic supplementary material. Surviving individuals reproduce (see '*reproduction*' below) and die immediately after. The *environment* is updated before the new generation is born, starting the cycle again. The *environment* is thus updated between the death of generation t and the birth of generation $t+1$ (see '*environmental-change*' below). In this way, newborns can adjust (if *plastics*) their *phenotype* according to the *environment* where they will live until death; and this is the *environment* that will affect their survival and reproduction.

— *Design concepts.* Evolution (changes in population mean/variance values of *genotypes*, either *plastic* or *non-plastic*, and *plasticity-range*) and other population dynamics (e.g. stability, bottlenecks and extinction) emerge from the combined effects of heredity, phenotypic plasticity (for *plastics* only), natural selection (differential survival and reproduction of individuals) and demographic (density-dependence) processes. Also, population genetic variability (either *genotype* or *plasticity-range*) is not imposed at initialization, but emerge during the first 100 generations when the population evolves under a mildly fluctuating *environment* (see '*environmental-change*' below). Note that the *genotype* and the *phenotype* could potentially take any real value, but in simulations tended to remain between 0 and 1 because of the selection imposed by the *environment* and the initialization conditions (i.e. *genotype* = *phenotype* = 0.5; see figure 2 insets and figure 3*c*). Stochasticity affects environmental change, mutation, survival probability and reproduction. We recorded the number of individuals at the end of 300 generations (100 of them being the initialization generations). For illustrative purposes, we also recorded for some model runs longitudinal (e.g. environmental fluctuations, population size dynamics, mean population *genotype*, *phenotype* and *plasticity-range*) and transversal data (e.g. *genotype* of each individual) across and within generations, respectively.

— *Initialization.* Simulations were initialized with *environment* = 0.5 and 100 individuals (either *mutants* or *plastics*). All individuals started with *genotype* = *phenotype* = 0.5. *Plastics* started with *plasticity-range* = 0.

— *Input.* The model does not have any external input; the *environment* is updated according to internal model rules.

— *Submodels*

Table 1. Variables and parametrization. All variables and parameters can take continuous values.

	initialization	constraints during simulations	description
parameters			
Std-Dev-environment-change	(0.04 – 1)	initialization value	determines the degree of environmental stochasticity $environment_{t+1} = environment_t + N \sim (0, Std\text{-}Dev\text{-}environment\text{-}change)$
plasticity-costs	(0 – 1)	initialization value	determines whether plasticity carries a load reducing odds of survival and reproducing
mean-mutational-change	(0 – 0.002)	initialization value	determines both the probability of occurrence of genetic changes and their effect size on the *phenotype*
emergent values			
environment	0.5	[0,1]	expresses the environmental conditions on a single dimension, the same one used to describe the *phenotype*, the *genotype* and the *plasticity-range*
non-plastics and plastics			
phenotype	0	—	phenotypic value expressed in the same dimension as the environment
genotype	0	—	in the absence of plasticity, the *phenotype* = genotype
match	n.a.	—	absolute difference between the phenotypic value and the environmental value; the phenotype is optimized if match = 1 $1 - \lvert environment - phenotype \rvert$
plastics only			
plasticity-range	0	—	the maximum phenotypic adjustment that a *genotype* is capable to increase *match*
used-plasticity	n.a.	$0 \leq used\text{-}plasticity \leq plasticity\text{-}range$	amount of the *plasticity-range* that is actually used by an individual during development

— '*environmental-change*'. During the first 100 generations of a simulation, the *environment* tightly fluctuates around 0.5. This is achieved by changing the *environment* towards 0.5 by increasing (or decreasing) the *environment* by a pseudorandom number extracted from a normal distribution with mean = 0.5 and variance arbitrarily fixed at 0.01 to ensure small fluctuations of the *environment* around 0.5. For the next 200 generations, the *environment* fluctuates every generation according to the value of a pseudorandom number extracted from a normal distribution with zero mean and *Std-Dev-environment-change* variance. To test the adaptive response to rapid directional changes and the role of costs of plasticity in causing genetic assimilation, we also modelled a scenario in which the *environment* fluctuates during the first 100 generations as in the other simulations, but then rapidly drift upwards in steps of 0.015 from 0.5 to 1, then remaining at 1 for the rest of the simulation.

— '*reproduction*'. Each individual produce *match* × 2 individuals, rounded to the nearest integer; i.e. they produce either 0, 1 or 2 individuals according to their *match*.

— '*mutation*'. The *genotype* and the *plasticity-range* (if *plastics*) inherited from the parent mutate by extracting a pseudorandom number from an exponential decay distribution with mean *mean-mutational-change* (see the electronic supplementary material). This number is either added or extracted to the inherited trait with equal probability. In this way, we are jointly modelling the probability of mutation and the magnitude of its effect on the phenotype. Given the many sources and kinds of mutations, we preferred this approach over simply modelling a per base per generation substitution rate (see the electronic supplementary material).

— '*development*'. *Non-plastics* develop a *phenotype* = *genotype*. *Plastics*, however, use their *plasticity-range* to produce a *phenotype* as close as possible (given their *plasticity-range*) to the *environment*. The amount of *plasticity-range* eventually used is called *used-plasticity* (i.e. $0 \leq used\text{-}plasticity \leq plasticity\text{-}range$).

— '*die-by-mismatch?*'. Individuals can die because of a low *match* with the *environment*. They do so with probability 1—*match*, i.e. extracting a pseudorandom number from a uniform distribution from 0 to 1, dying if this number is > *match*.

— '*die-by-negative-density-dependence?*'. *Plastics* and *non-plastics* die because of negative density-dependence when (before reproduction) population size is above 100 individuals. The dying individuals are those with

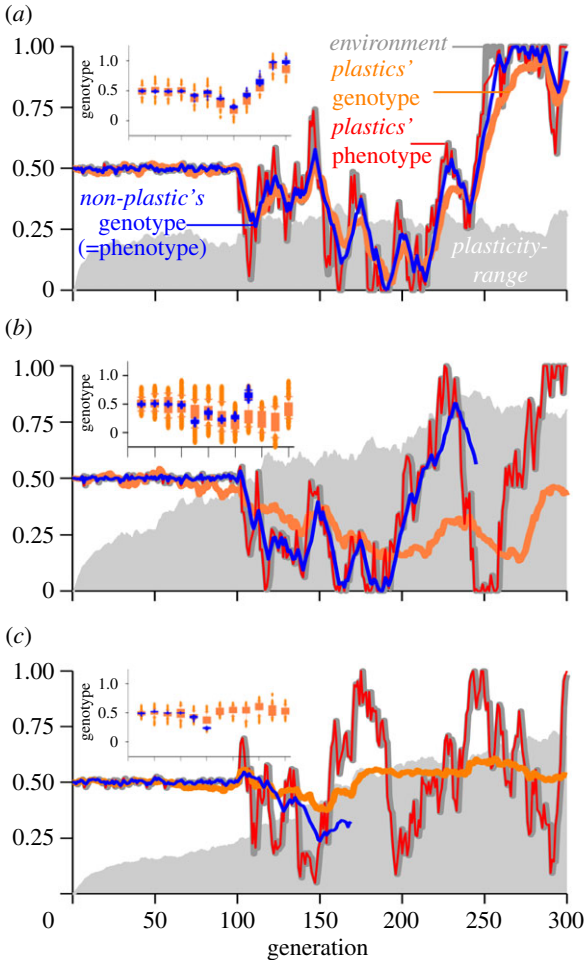

Figure 2. Examples of adaptive evolution of *plastic* and *non-plastic* populations under medium-low environmental fluctuations (*Std-Dev-environment-change* = 0.1) and different scenarios of *mean-mutational-change* and *plasticity-costs*. (*a*) At high *mean-mutational-change* and high *plasticity-costs*, *plastics* performed similar to *non-plastics*. Here, a high *mean-mutational-change* allowed both populations to closely track the *environment*. *Plasticity-range* was reduced compared with scenarios with lower costs but maintained owing to environmental fluctuations. (*b*) Under high *mean-mutational-change* but with low *plasticity-costs*, plasticity allowed a close phenotypic *match* to the *environment* and the persistence of the *plastic* population, but often *non-plastics* went extinct as shown in this example. (*c*) Under low *mean-mutational-change* and low *plasticity-costs*, *plastic genotypes* produced *phenotypes* that closely matched the *environment* while their genotypic values were intermediate across environmental fluctuations, and plasticity increased. *Non-plastic* genotypes could not adapt fast enough and quickly went extinct. At any given time and in all scenarios, genotypic variation was higher in the *plastic* population than in the *non-plastic* one. This is shown in inset boxplots in each panel, where blue boxes depict genetic variation of the *non-plastic* population and orange boxes that of the *plastic* population, sampled every 25 generations.

lower *match* with the *environment* (note that in any given model run all individuals are either *plastics* or *non-plastics*, so there is no competition between these types).

— '*die-by-plasticity-costs?*'. With the same approach, *plastics* can also die first with probability = *plasticity-range* × *plasticity-costs*, and then also with probability = *used-plasticity* × *plasticity-costs*. That way, increased plasticity costs penalize separately plasticity maintenance and plasticity use. Maintenance is associated with the ability of being plastic, i.e. *plasticity-range*; the broader the range of possible phenotypes, the highest the cost. Production costs, however, are the costs incurred when actually altering the phenotype (i.e. *used-plasticity*; see the electronic supplementary material).

(a) Simulations

Simulations for *non-plastics* and *plastics* are run independently but using the same pseudorandom generator seed to make results fully comparable. For each group, we ran a total of 200 simulations for each of the 4056 combinations of 26 (equally spaced) values for *Std-Dev-environment-change*, 26 different values for *mean-mutational-change* and six values of plasticity-cost i.e. a total of 811 200 model runs (see table 1 for parameter details). For each of the 4056 parameter combinations, we calculated (separately for *non-plastics* and *plastics*) population size at the end of the simulations and the cumulated population size along the 200 generations after initialization. Note that we run 200 simulations for each of the 4056 parameter combinations for *plastics* and *non-plastics* although parametrizations only differing in the *plasticity-cost* value do not affect *non-plastics*. This way results from *plastics* were directly comparable with simulations (with same pseudorandom generation seeds) for *non-plastics*. To test hypothesis (iv) regarding genetic assimilation in a novel environment, we also modelled a scenario with an abrupt directional environmental change, which then stabilized (see above). This could represent either the colonization of a novel habitat, or a rapid environmental transformation such as those occurring as a consequence of global change across the world.

3. Results

During the first 100 generations of the model runs, the *environment* was forced to remain close to 0.5 and the initial generation had *genotype* = 0.5 and *plasticity-range* (if *plastics*) = 0. In all simulation runs, *plastic* and *non-plastic* populations survived these initial generations, generating standing genetic variation and (in *plastics*) variation in *plasticity-range*. As plasticity costs increased, population size during the first 100 generations of initialization was lower for *plastics* than for *non-plastics* (see examples for intermediate plasticity costs in figure 3*b*), indicating that under low environmental fluctuations, plasticity costs may outweigh the benefits of plasticity.

(a) Adapting to a fluctuating environment

Afterwards, when the *environment* was allowed to vary stochastically along 200 generations, the *plastic* and *non-plastic* populations began evolving to adapt to the changing *environment*. Both *plastic* and *non-plastic* populations were capable of persisting over simulated environmental fluctuations provided that the *mean-mutational-change* was high, but population viability was severely compromised as environmental fluctuations increased (figures 2 and 3). At low environmental fluctuations, *plastics* always performed slightly worse than *non-plastics* during the next 200 generations (figure 3*a*, and first panel of figure 3*b*). This also supports the idea that plasticity even at low plasticity costs has demographic consequences when occurring at low environmental fluctuations.

Selection favoured increased plasticity during bouts of rapid, recurrent or wide environmental shifts (figure 2 main

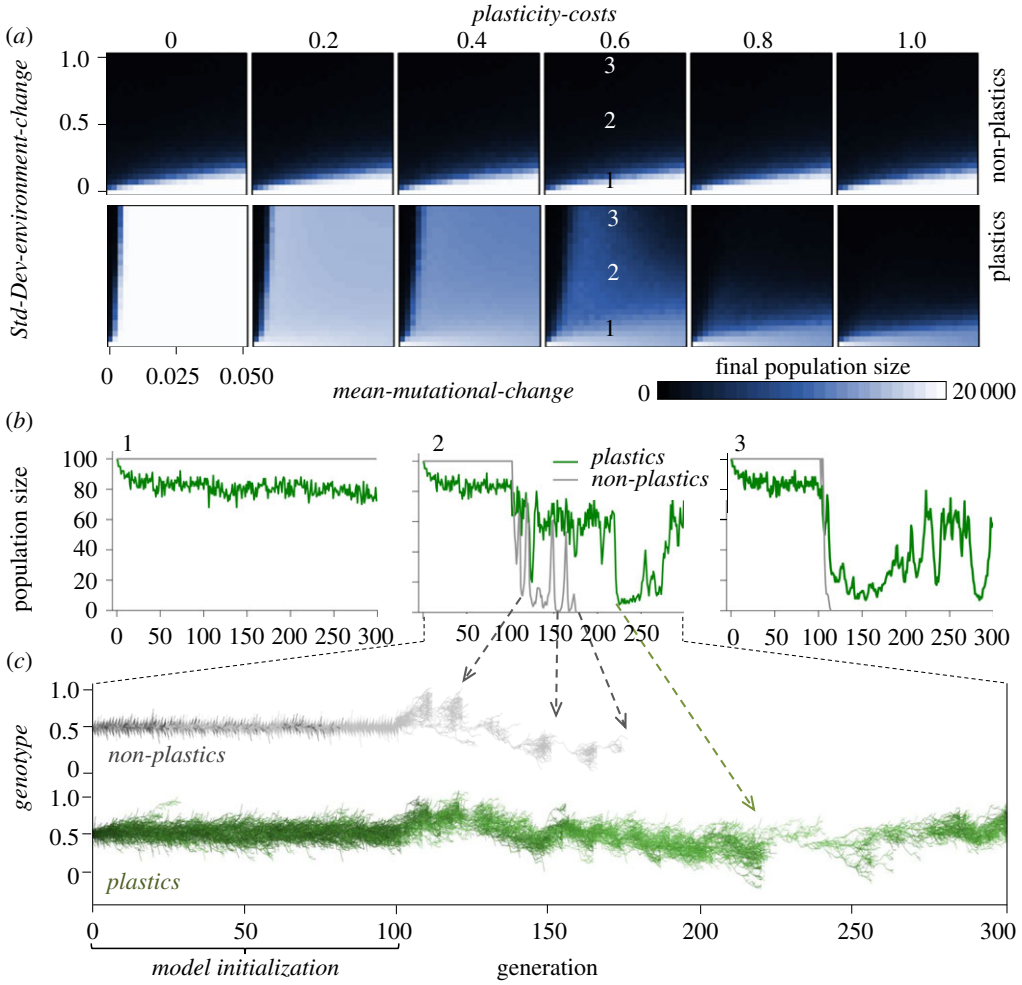

Figure 3. (*a*) Results for population size for populations composed of either *plastic* or *non-plastic* genotypes from simulations sweeping over all parameter combinations of environmental stochasticity (*Std-Dev-environment-change*), mutation rate (*mean-mutational-change*) and *plasticity-costs*. Populations composed of *plastic* genotypes persisted over a much broader range of environmental stochasticity than populations of *non-plastic* genotypes, unless *plasticity-costs* were high, in which case they performed worse than *non-plastic* genotypes. (*b*) Examples of population dynamics for *plastic* and *non-plastic* populations at different levels of environmental stochasticity and *mean-mutational-change* = 0.04 and *plasticity-costs* = 0.6; panel numbers relate (*a*) and (*b*). (*c*) Example of clonal lineages trajectories (each line is a lineage) according to *genotype* and (for plastics) *plasticity-range* (lighter green colour depicts higher *plasticity-range*). Note that only very plastic lineages survived the strongest population bottleneck (as shown in corresponding (*b*) panel).

panels), often being the most plastic genotypes the ones that persisted (see examples in figures 3*c* and 4*a*). Costs of plasticity reduced the effectiveness of the plastic response and when taken to the extreme ultimately made *plastic* genotypes evolve analogously to *non-plastic* ones (figure 3*a*). Except in such scenarios of extreme costs of plasticity, *plastic genotypes* always showed a better phenotypic *match* to the *environment* than *non-plastic* ones, even at high *mean-mutational-change* (figure 2 main panels).

At higher *Std-Dev-environment-change*, selective sweeps of poorly matched *genotypes* were more frequent and resulted in population bottlenecks (figure 3*b*), reducing the likelihood of persistence for both *plastic* and *non-plastic* genotypes (figure 3*a*). Population viability of *non-plastics* was restricted to low environmental fluctuations and high *mean-mutational-change* (figure 3*a*). *Plastic genotypes*, however, experienced attenuated population bottlenecks because a greater fraction of *genotypes* within the population were capable of expressing appropriate *phenotypes*, confirming our first prediction (figures 2 and 3). Plasticity allowed the persistence of populations even at low rates of *mean-mutational-change* and high environmental fluctuations, unless *plasticity-costs* were high (0.7 and above; figure 3*a*).

The maintenance of an average greater population size and alleviation of bottlenecks also contributed to increased genetic variation in the *plastic* populations (figure 2 insets). Moreover, because large *plasticity-ranges* allowed *genotypes* that would otherwise have had a poorly fitted *phenotype* to improve their *match*, the effect of selection was buffered and higher genotypic diversity within populations was retained in plastic populations at all times, confirming our second prediction. The strong genetic response to selection of *non-plastics*, however, resulted in a better *match* between average *genotype* and the *environment* for *non-plastic* than for *plastic genotypes* (figure 2). Consequently, in fluctuating environments, plasticity allowed the *phenotype* to closely match the *environment* while slowing down the genotypic response to selection (figure 2). At low *plasticity-costs*, the average genotypic value was maintained around the average value of the environmental conditions experienced throughout the simulations while at the same time retaining large genotypic variance (figure 2*b*,*c*). In consequence, low plasticity-costs allowed increased plasticity to evolve (figure 2*b*,*c*), leading to a higher *genotype* variance (figure 2*b*,*c* insets) and thus increasing the chances that appropriate genetic variants of fixed phenotypes arose by mutation.

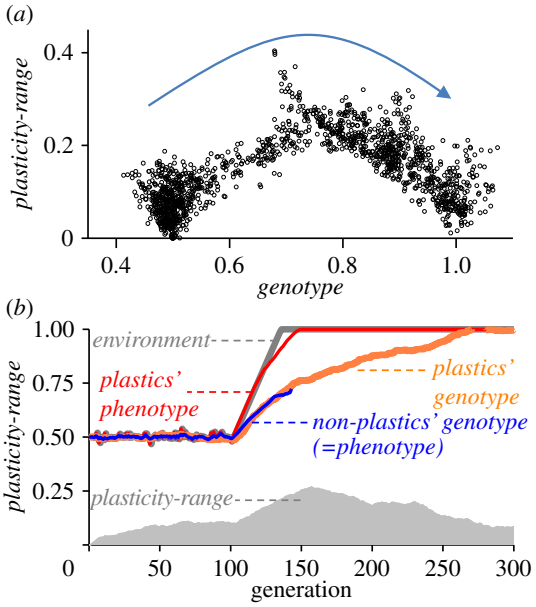

Figure 4. Example of model run for a scenario of directional environmental change, where environment changed abruptly from 0 to 1 and then stabilized at 1 with *mean-mutational-change* = 0.005 and *plasticity-costs* = 0.7. (*a*) Shows for *plastic* individuals their position in the *genotype* versus *plasticity-range* space. The arrow indicate the pass of time (in generations), beginning with all individuals with *genotype* = 0.5 and *plasticity-range* = 0 (initialization conditions) and ending at the end of the simulation with individuals with *genotypes* close to 1 and reduced *plasticity-range*. (*b*) Same as in figure 2. It is shown how plasticity increased temporarily under selection and the plastic population expressed well-matched phenotypes, allowing the population to persist over enough generations to allow genotypes to slowly evolve towards the new optimum. Once the environment stabilizes, plasticity is rapidly reduced owing to costs of plasticity, causing genetic assimilation.

(b) Environmental stabilization and genetic assimilation

To test the prediction that costs of plasticity result in loss of plasticity upon environment stabilization, we simulated a fast environmental transition from *environment* = 0.5–1, followed by *environment* stabilization at 1, such as it would occur for instance owing to human activity or if a population was to enter a distinct ecological region (figure 4). As in previous analyses (figures 2 and 3), our model exploration showed that adaptation to the novel *environment* in the *non-plastic* population depended on *mean-mutational-change* relative to environmental change (results not shown). Also, if the environment changed too abruptly given their mean-mutational-change, the *non-plastic* population failed to adapt and went extinct. Plastic genotypes, however, managed to persist even with a low *mean-mutational-change* and despite rapid transitions to the novel *environment*. It was possible because their *plasticity-range* allowed them to manifest *phenotypes* that better matched the *environment* at any given time. As shown in figure 4*a*, *plasticity-range* was strongly positively selected during the abrupt environmental change and only the most plastic *genotypes* survived the sharp environmental transition, because only very plastic *genotypes* were capable of producing extreme *phenotypes*. Nevertheless, plastic *genotypes* lagged substantially behind their *phenotype* (figure 4*b*). In other words, plasticity bought time for adaptive, fixed (i.e. non-*environment* sensitive) genetic changes to occur because individuals expressed the appropriate *phenotype* soon but it often still took the *genotype* many generations to match the *environment* (figure 4*b*). When costs of plasticity were high and the new *environment* remained stable, plasticity quickly decreased to background levels maintained by mutation, resulting in genetic assimilation of the environmentally induced phenotypes (figure 4*a*,*b*).

4. Discussion

With this simple heuristic model, we integrated adaptive plasticity into an explicit population genetic framework and examined some fundamental consequences of plasticity in adaptive evolution. We found that fluctuating or rapid directional environmental change strongly selected for plastic genotypes. This result is in accordance with previous modelling approaches [22,41,42], especially when environmental fluctuations are modelled to act after development but before selection [43]. In our model, increased plasticity allowed genotypes to produce phenotypes better matching the changing environmental conditions at each generation, hence showing a high potential for rapid adaptation to new environments. This relationship between plasticity and adaptive potential to novel environments has been suggested in some cases, as in invasive plant species having greater plasticity than non-invasive ones [44]; plasticity mediating rapid adaptation to introduced predators in zooplanktonic species [45]; or adaptations to climate change in birds [46].

Plasticity led to faster phenotypic modifications of whole populations because adaptive phenotypes were induced concurrently by environmental cues available to all individuals, instead of requiring the time for beneficial mutations to spread throughout the population by differential survival and reproduction [7]. This allowed populations composed of plastic genotypes to suffer fewer and lesser demographic bottlenecks despite steep fluctuations in the environment (figures 2 and 3).

An important result emerging from this model is that adaptive plasticity contributes to the maintenance of genetic variation within population (figure 2 insets) in two ways. First, plastic populations had higher genetic variation because plasticity shielded a broader range of genotypes from purifying selection by allowing them to express well-matched phenotypes. Second, plasticity reduced the effect of genetic drift as a consequence of maintaining greater population sizes (i.e. by reducing population bottlenecks). This result is supported by a very different modelling approach that has also recently proposed that plasticity tends to lead to populations with greater mutational and standing genetic variance [47].

It has often been debated whether plasticity fosters evolution by facilitating adaptation to novel environments or rather impede divergence by shielding genetic variation from divergent selection [17,48,49]. We show that plasticity allows phenotypically cryptic (or unexpressed) genetic variation to build up within populations by conferring similar fitness to distinct genotypic variants (see also [18,50]). Adaptive plasticity also allows otherwise imperilled populations to persist until appropriate genetic variants appear (figures 2 and 4). Moreover, the accumulated genetic variation can be rapidly released and manifested in the face of further environmental or mutational changes, enabling rapid adaptive divergences [6,17,51,52]. Our study suggests that plasticity facilitates adaptation to novel environments by allowing a synchronic

phenotypic shift in response to the environment, while at the same time maintaining genetic variation that would otherwise be selected out (figure 2 insets), even though phenotypic plasticity slows down the response to selection (figures 2 and 4*b*).

Overall, shielding of genetic variation by plasticity may only be a transient effect of an otherwise rapid process of adaptation to divergent environments by genetic accommodation, as we found that plastic genotypes always showed a greater adaptive potential to a changing environment (figures 2–4). Congruently, there are many cases of rapidly diversifying groups of species where genetic accommodation of plasticity is likely to have been the main driver for divergence [53], as in sticklebacks [54,55], anole lizards [56] or arctic charrs [57]. Rapid adaptive transitions between environments are more easily achieved by plastic than non-plastic genotypes (figures 3 and 4), and we show that genetic assimilation of induced phenotypes and the associated loss of plasticity will occur if costs of plasticity are high and the environment stabilizes (figure 4).

Plasticity costs have been elusive and difficult to measure empirically [58–60], but there is evidence for plasticity costs from plants to invertebrates and vertebrates [61–63]. Moreover, patterns of evolution of plasticity are often congruent with theoretical expectations of the consequences of costs of plasticity, namely reduced plasticity under stable environmental conditions. American spadefoot toads, for instance, have evolved a canalized accelerated larval development with respect to the slow but plastic development ancestral to the group as a result of their adaptation to ephemeral desert ponds [31]. Accelerated development has become nearly genetically assimilated, and plasticity has been lost to a great extent in desert spadefoot toads so they are no longer capable of long larval periods [31,64]. Such translation of ancestral environmentally induced changes in development within populations into adaptive constitutive divergences among taxa is a clear path connecting micro- and macroevolution [2,7,31].

Because environmental variation is the rule in nature [65] and it often selects for adaptive plasticity [16,18,66], the evolutionary paradigm needs to be extended to include environmentally dependent regulation of gene expression as a heritable source of phenotypic variation, whether genetic or epigenetic [9,35,67–69]. Whether the incorporation of adaptive plasticity constitutes an extension of the paradigm emerged from the Modern Synthesis or a new paradigm, may ultimately be better evaluated retrospectively. To some extent, adaptive plasticity simply extends and strengthens the current paradigm, as it improves our understanding of the maintenance of genetic variation in populations, facilitates rapid adaptive shifts between adaptive peaks and helps explaining the adaptive radiations and recurrent parallel speciation. However, at the same time, accounting for adaptive plasticity expands the Modern Synthesis paradigm in several meaningful aspects that may warrant a new paradigm. Our model illustrates these aspects in a fairly simple and intuitive way. First, during organismal development, the environment acts as a phenotypic inducer in addition to its traditional role as a mere selective sieve. This is important because environmental induction may act simultaneously on most genotypes in a population inducing synchronous phenotypic shifts in the direction of the new local adaptive optimum. Second, plasticity increases the match of the phenotype to the environment, reducing bottlenecks and hence increasing population viability. Last, plasticity contributes to the maintenance of genetic variation within populations both by shielding many genetic variants from selection and by reducing genetic drift, and can become quickly accommodated between lineages evolving in divergent environments.

In this line of thought, our model shows the high relevance of plasticity to evolution and population ecology, while at the same time it shows that incorporating plasticity is conceptually as simple as acknowledging the fact that genotypes may have the potential to use environmental information to express better fit phenotypes. Other central tenets of mainstream evolutionary thought (i.e. random mutation and selection of phenotypes according to environmental conditions) evidently remain unchanged. The simple addition of environmentally sensitive adaptive gene regulation, however, provides a demonstrated mechanism for swift adaptation to rapidly changing environments that may have often lead to lineage diversification and evolutionary innovations.

Acknowledgements. We thank P. Edelaar, S. Sultan, J. Moyá and C. M. Herrera, and two anonymous reviewers for their comments and suggested improvements to the model.

Funding statement. This research was supported by the Ramón y Cajal Program of the Spanish Ministerio de Ciencia e Innovación (MICINN) to I.G.M. (RYC-2008-03519) and to R.J. (RYC-2009-03967).

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