DOI: 10.1111/ede.12305

RESEARCH

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Developmental noise and ecological opportunity across space can release constraints on the evolution of plasticity

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Funding information
NSF, Grant/Award Number: 1714550

Abstract

Phenotypic plasticity is a potentially definitive solution to environment heterogeneity, driving biologists to understand why it is not ubiquitous in nature. While costs and constraints may limit the success of plasticity, we are still far from a complete theory of when these limitations actually proscribe adaptive plasticity. Here I use a simple model of plasticity incorporating developmental noise to explore the competitive and evolutionary relationships of specialist and generalist genotypes spreading across a heterogeneous landscape. Results show that plasticity can arise in the context of specialism, preadapting genotypes to later evolve toward plastic generalism. Developmental noise helps a mutant with imperfect plasticity successfully compete against its ancestor, providing an evolutionary path by which subsequent mutations can refine plasticity toward its optimum. These results address how the complex selection pressures across a heterogeneous environment can help evolution find paths around constraints arising from developmental mechanisms.

1 | INTRODUCTION

Phenotypic plasticity is the development or modification of traits in response to environmental cues. Adaptive plasticity is common but not ubiquitous (Palacio-López, Beckage, Scheiner, & Molofsky, 2015), and biologists feel the lack of a predictive theory for when plasticity should evolve when they contemplate the limits to species' resilience to environmental change (e.g., Ashander, Chevin, & Baskett, 2016). Plasticity represents both a product of adaption and a precursor to further change. Plasticity is one solution to environmental heterogeneity, and a number of theorists have pushed toward a synthetic theory that explains when plasticity will emerge and preclude other results like genetically differentiated local adaptation, stochastic bet-hedging, or simply extinction (e.g., Bull, 1987; Scheiner, 2014b; Svardal, Rueffler, & Hermisson, 2011; Tufto, 2015). Plasticity has also been linked to speciation (Pfennig et al., 2010; Schneider & Meyer, 2017), innovation (Moczek et al., 2011), and invasion success (Davidson, Jennions, & Nicotra, 2011), placing it at the core of an emerging, predictive theory of the determinants of the rate of evolution.

Understanding the limits of plasticity is a persistent challenge in evolutionary biology because the issue requires substantial attention to ecology, life-history, and development. Ecology provides both the stick and the carrot, providing both the need for distinct phenotypes across a heterogeneous environment and the informative cues necessary to produce the correct phenotypes. Life-histories can limit the reliability of this information because movement preceding reproduction or metamorphosis can divorce past cues from the organism's adult environment (Scheiner, 2013). The limitations imposed by dispersal from the natal environment depend on the lags and costs intrinsic to the developmental system: behaviors might adjust rapidly to changes in cues, while morphology might be less malleable. Efforts to review the field have produced taxonomies of the various costs that

https://doi.org/10.1111/ede.12305

reduce the realized benefit of plasticity and the constraints that prevent it from arising at all (DeWitt, Sih & Wilson, 1998; Murren et al., 2015). While some costs are obvious—for example, the need to produce a sensory organ—comparative work has mostly failed to find clear costs that make plasticity less competitive than closely related nonplastic specialists (Auld, Agrawal, & Relyea, 2009; Van Buskirk & Steiner, 2009). Attention has, therefore, shifted to understanding how constraints might restrict the evolution of plasticity to certain traits, taxa, and environments.

The complex and poorly understood effects on the evolution of plasticity stemming from these diverse ecological and organismal factors motivate theoretical research. Exploration of extremely simple models can provide structured hypotheses for where constraints might come from and how experiments can be designed to measure which constraints actually matter for explaining the distribution of plasticity across nature. The model presented here stems from a tradition in which modeling of plastic development is made extremely simple in favor of focusing attention on complexity arising from environments and life-histories. Here, an organism's genotype determines the slope and intercept terms of a linear equation, with plasticity emerging as the product of the slope and the difference in cues provided by each environment. While abstract, similar approaches have been successfully used to model the interactions of spatial and temporal heterogeneity with life-histories (Scheiner, 2013), the joint determination of dimorphism by plasticity and genetic differentiation (Leimar, Hammerstein, & Van Dooren, 2006), and the maintenance of genetic variation in plastic traits (De Jong & Gavrilets, 2000). A slope-intercept model codifies the idea that the effects of mutations are biased by some developmental structure, but that this structure can itself evolve to change those biases. The work here combines this simple framework with both spatial structure and developmental instability to articulate new hypotheses about the ecological and developmental factors favoring plasticity.

Developmental instability has been studied within evolutionary biology under names like canalization, robustness, and specific cases such as fluctuating asymmetry. A recent surge of interest in the topic has been inspired by the tremendous growth in capacity to measure and model stochastic heterogeneity at the levels of molecules, cells, and fitnesses in single-cell organisms (Bruggeman & Teusink, 2018; Draghi, 2018; Raser & O'shea, 2005). Stochastic heterogeneity in phenotypes arising from developmental instability has often been viewed as both a direct detriment to fitness and an impediment to the fixation of adaptive mutations (Wang & Zhang, 2011). However, the concept of random phenotypic diversity as an adaption to unpredictable change has a long history in evolutionary thought (Starrfelt & Kokko, 2012). Microbial experiments have been particularly influential by illustrating how cells can use stochastic variability to create a diverse portfolio of phenotypes—e.g., nongrowing, resistant spores and cells competent for natural genetic transformation (Veening, Smits, & Kuipers, 2008). While most attention has focused on understanding how this stochastic variability could function as a form of bet-hedging (Starrfelt & Kokko, 2012), recent experiments point to more subtle benefits to variability and highlight its potential role in stimulating, rather than slowing, adaptation (Bódi et al., 2017).

Although plasticity and developmental instability have often been considered separately, a recent finding of a positive correlation between these traits in *Arabidopsis* phenotypes (Tonsor, Elnaccash, & Scheiner, 2013) has helped to spark interest in their joint consideration. A recent modeling paper showed how instability arising from genetic factors could evolve, and that evolved robustness in development actually constrained the evolution of plasticity (Draghi, in review). The core result of this paper was that developmental noise loosened pleiotropic constraints preventing plastic mutants from successfully competing against their parent genotypes, allowing a population to evolve a plastic response to environmental heterogeneity. This prior work was confined to the limitations of most traditional population-genetics models: a single well-mixed population reproducing under soft selection. Also, stochastic heterogeneity arose solely from genetic factors in this prior work, making it difficult to isolate the effects of that trait from other traits determined by the same genetic factors. Here I expand upon this study by applying hard selection to a population arrayed across a heterogeneous landscape, and by allowing the environment, rather than the genotype, to control the expression of developmental noise. The model explored here allows plasticity to arise in response to large-scale differences between distinct environments, in the context of stochastic inputs to development that also derive from the environment. While this dual role of the environment on development connects with classical ideas about plasticity (Bradshaw, 1965), its formulation in this spatial model allows for new insights about the origins of genotypes using plasticity to achieve a generalist phenotype. Specifically, the ability of a population to adapt to a novel environment depends on both the aid of developmental noise and the prior emergence of genotypes using plasticity to achieve novel specialist lifestyles. These results show how the developmental biases intrinsic to a very simple form of pleiotropy create a soft constraint on the evolution of an innovative feature, and illustrate how environmental opportunity and evolutionary history can lead to lineages that overcome that constraint.

2 | MODEL AND METHODS

The purpose of this model is to explore the interplay of a very simple developmental system with a more complex ecological framework featuring hard selection, explicit spatial structure, and two types of environmental effects: plasticity and environment-dependent developmental instability. This paper does not attempt to characterize the behavior of this complex model over the range of parameters; rather, the goal is to generate hypotheses about the role of space in the evolution of plasticity, and to illustrate how a developmental system leads to a biased set of mutations that interact in subtle ways with selection across a landscape. To support these goals, I will aim to articulate the model I used as simply as possible, rather than present it in a more general notation.

The spatial environment for a population was modeled as a lattice of cells, each of which could contain up to one organism. These cells were arranged in a rectangle of width W and height H, allowing a maximum of $N = W \times H$ organisms at a time. Each cell was assigned to one of two environments, labeled 1 and 2: these environments differed in their optimal value of the organism's trait, labeled $z_{opt}(1)$ and $z_{opt}(2)$, respectively. The environment also determined which of two values of a cue would be perceived by an organism developing in that cell, providing the developing organism with perfect information about the optimal phenotype for that environment.

Each cell in a landscape was assigned to either Environment 1 or 2 based on a sigmoid function of the x coordinate of that cell's location. Environmental noise was introduced during development into the phenotype, with a magnitude increasing with the y coordinate. This allowed for two forms of stochasticity: dispersal introduced a chance component to where an organism would develop, and environment noise within an environment shaped the resulting adult phenotype.

The probability that a cell would be assigned to Environment 2 was determined as follows:

$$p_2(x) = \left(1 + e^{-2\left(\frac{2x}{W} - 1\right)}\right)^{-1}. \quad (1)$$

A clustering algorithm was then used to produce a variable degree of autocorrelation in space while respecting the expected frequencies derived from Equation (1). This algorithm chose a pair of cells with the same x coordinate and inspected their assigned environments and the eight cells making up their immediate neighbors. If the chosen cells were assigned different environments, and if each was environmentally dissimilar to the majority of its neighbors, then the assignments for those two cells were swapped; otherwise, no change was made. The number of random pairs considered for a potential swap was determined as a Poisson random number with a mean of θN, where θ acts as a clustering parameter. Figures 1a and 2 show examples of the resulting spatial structure when $\theta = 1$.

An organism's genotype consisted of two real numbers corresponding to the slope, a and intercept, b of the canonical equation of a line. The phenotype corresponding to an organism's genotype was determined from three sources: these two genotypic parameters, the cue $c(i)$ associated with the environment assigned to that particular organism's location, and a Gaussian noise term with a mean of zero and a standard deviation $\sigma(y) = 50 + 300(y/H - 1)$. A genotype's phenotype is, therefore, a random variable described by the following equation.

$$z(x, y) = ac(i) + b + N(0, \sigma(y)). \quad (2)$$

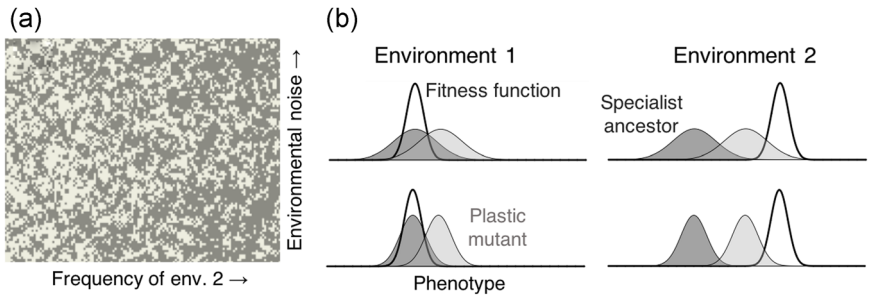

FIGURE 1 (a) A portion of an example landscape showing the distribution of patches of the two environments across the dimensions of the space. The clustering parameter was $\theta = 1$ for this example. The color of each cell indicates the Environment (1 or 2), with the frequency of Environment 2 increasing from left to right. Environmental noise causing developmental instability increases from bottom to top (not illustrated). (b) An illustration of a plastic mutant (phenotypic distributions in light gray) derived from a static ancestor (dark gray). Bold lines indicate the fitness functions in the two environments. Environmental noise is $\sigma = 150$ for the lower examples and $\sigma = 250$ for those on top, representing the change in environment noise along the y-axis of the landscape. For the ancestor, the genotypic parameters are $a = 0$ and $b = 1{,}000$ (see Equation (2)); for the mutant, $a = 0.3$ and $b = 1000$ [Color figure can be viewed at wileyonlinelibrary.com]

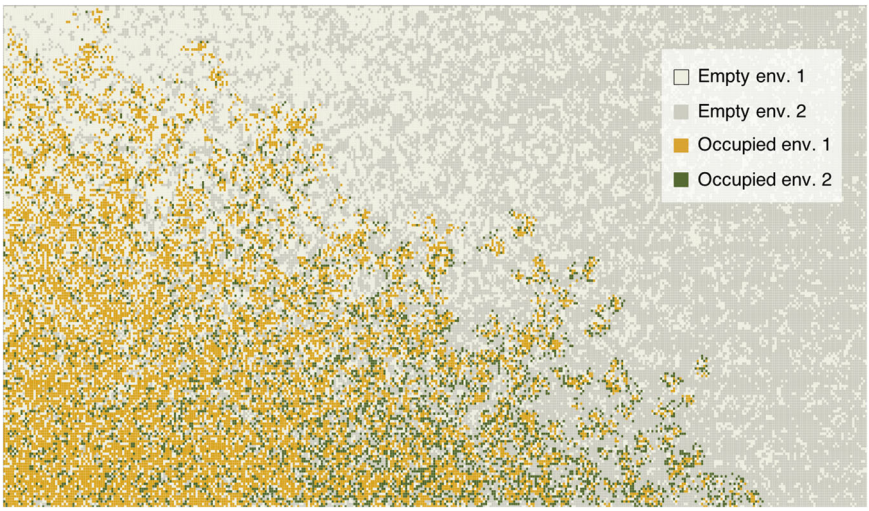

FIGURE 2 Colonization of an example landscape by the ancestral Environment-1 specialist. Snapshot of the adults in the population after 1,000 generations, showing a quasiequilibrium distribution without evolution [Color figure can be viewed at wileyonlinelibrary.com]

The distribution of phenotypes produced by a given genotype, therefore, depends on environmental factors in two ways. A genotype with some degree of plasticity ($a \neq 0$) will produce a different mean phenotype in Environment 1 compared to its average in Environment 2 (Figure 1b). In addition, the y coordinate shapes the variability of development but not its mean; this stochastic influence occurs regardless of the type of environment or the organism's genetic values.

An organism's fitness is assigned via a Gaussian function comparing its phenotype with the optimum for its environment i. Throughout the simulations reported here, $z_{\text{opt},1}$=1000, $z_{\text{opt},2}$= 2000, $c_1 = 1000$, $c_2 = 2000$, and σ^2_{opt}=5,000. Fitness w is therefore:

$$w = \exp\left(\frac{-(z - z_{\text{opt},\,i})^2}{2\sigma^2_{\text{opt}}}\right). \tag{3}$$

After birth, an organism disperses based on a Gaussian movement kernel with a variance of 2.5. If the organism disperses to an occupied cell or off the margins of the landscape it is lost from the population; if it lands in an empty cell it develops to adulthood as governed by its genotype (Equation (2)). Fitness determines fecundity and reproduction is semelparous and asynchronous. A generation is defined by permuting the list of all cells and processing each cell in order according to the following algorithm: if a cell is occupied, that organism produces a Poisson-distributed number of offspring with a mean equal to 10 times the fitness of the organism; these offspring then disperse, and the focal organism is removed.

Organisms are haploid and reproduce asexually with mutation at a rate $\mu = 0.001$/individual/generation. This rate was chosen to balance two constraints: a very high rate can allow a lineage to acquire two or mutations in rapid succession, allowing pleiotropic constraints to be avoided with unrealistic ease. A rate that is too low simply slows the evolutionary dynamics and inflates the needed computation time. By capping the rate at an average of one mutation per 1,000 generations we err toward the side of computational inefficacy and allow selection to act effectively on individual mutations.

A mutation affects either the a or b parameter with equal probability. Mutations in the a parameter are Gaussian with a standard deviation of 0.4; mutations in the b parameter are also Gaussian with a standard deviation of 500. Because genotypes are fully linked, each individual has a single lineage of ancestors. Genealogies of every new mutant genotype are recorded during each simulation such that lines of descent can be unambiguously reconstructed and traced. The spatial location on the landscape at which a new mutation originates is also recorded.

We can write a useful equation for the relative fitness of a genotype in a given environment, averaging over the distribution of phenotypes it would be expected to produce. The integral of Equation (3) with the Gaussian distribution of phenotypes defined by Equation (2) is given below:

$$\begin{aligned}\omega(\mu, \sigma^2) &= \frac{1}{\sigma\sqrt{2\pi}} \int_{-\infty}^{\infty} \exp\left(-\frac{(z - z_{\text{opt}}(1))^2}{\sigma^2_{\text{opt}}}\right) \\ &\quad \exp\left(-\frac{(z-\mu)^2}{\sigma^2}\right) dz, \\ \omega(\mu, \sigma^2) &= \exp\left(-\frac{(\mu_{\text{opt}} - \mu)^2}{2(\sigma^2_{\text{opt}} + \sigma^2)}\right) \frac{\sigma_{\text{opt}}}{\sqrt{\sigma^2_{\text{opt}} + \sigma^2}}.\end{aligned} \tag{4}$$

Simulations and analysis scripts were written in R and will be made available in a Data Dryad repository.

3 | RESULTS

3.1 | Model dynamics without evolution

Simulations began with ancestral organisms that lacked plasticity and were perfectly adapted to one of the two environments, referred to as Environment 1 or the ancestral environment. Moving east across the landscape the frequency of patches of this ancestral environment declines (Figure 1a) in favor of Environment 2, the novel environment, to which the original genotype was poorly adapted. Figure 2 shows an example of the spread of the ancestral genotype in a simulation without the potential for evolution (mutation rate $\mu = 0$). As in all the simulations discussed here, individuals were initially placed on the western edge of the landscape and moved across it via dispersal of offspring. This distribution represents a quasi-steady-state, as some of the unoccupied clusters of Environment 1 might be colonized by rare long-distance migrants given sufficient time. However, this snapshot does illustrate key aspects of the spatial distribution of the original specialist. Notably, developmental stochasticity reduces fitness (see Equation (4)) which, under these hard-selection conditions, causes the population density to decline along a latitudinal cline. The combination of the latitudinal gradient of environment noise magnitude with the lateral gradient in the frequency of Environment 2, to which this genotype is poorly adapted, creates a roughly diagonal range margin for this genotype. Finally, note that autocorrelation among the patches of each environment leads to small enclaves of the population in areas surrounded by the unfavorable Environment 2, as well as numerous uncolonized patches of Environment 1.

3.2 | Evolution of model generalists and specialists across space

Evolutionary simulations with mutations in both genetic parameters produced several distinct phenotypes. Figure 3 shows the origination points of successful mutants of two types: specialists on the novel environment, and plastic generalists that use both environments. A genotype whose fitness in Environment 2 is at least four times greater than its fitness in Environment 1 is considered a specialist on Environment 2; specialists on Environment 1 are defined similarly, and generalists are then defined as the intermediate cases. These mutants are drawn from 100 replicate simulations, and a mutant was classified as successful if it rose to at least 2% of its population—qualitative patterns were not sensitive to the value of this threshold (data not shown). Both types of mutants can thrive across a range of origins in the x coordinate, though both show some clustering toward the right half of the middle. However, in the y coordinate new specialists tend to arise in areas with lower developmental noise, while plastic generalists show an opposite trend and overall show more variability in where they flourish.

The role of environmental noise in the origination of successful plastic generalists can be understood by first appreciating how noise shapes both the fitness and the realized resource utilization of a genotype. The mutants in Figure 3 are assigned to the categories of generalists and specialists based on their predicted niche, given the level of environmental noise present where each mutant arose. While the basic pattern in Figure 3 is robust to the arbitrary fourfold cutoff applied (data not shown), the role of environmental noise in this determination is intrinsic to the model and deserving of more explication.

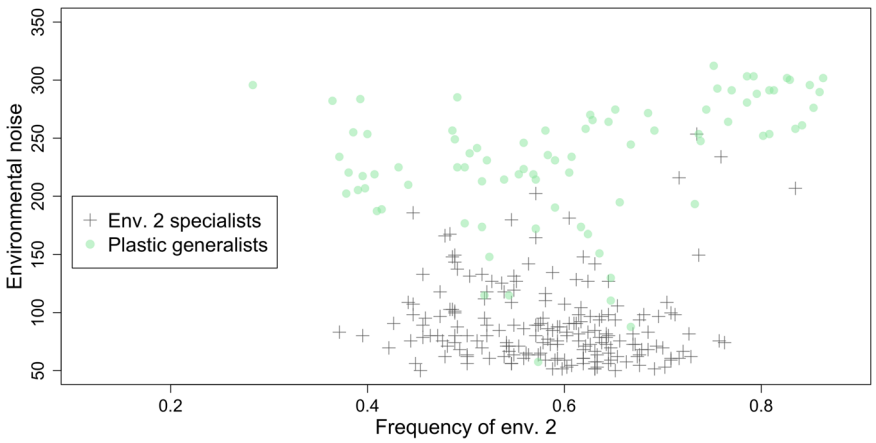

FIGURE 3 Novel specialists and plastic generalists arise from different areas of a heterogeneous landscape. The figure comprises the results of 100 replicate evolutionary simulations; points depict successful mutants that specialized in Environment 2 or showed a significant, plastic ability to use both environments. Mutants were defined as successful if they achieved a maximum frequency of at least 0.02 during the simulation (see Section 2) [Color figure can be viewed at wileyonlinelibrary.com]

Therefore, Figure 4 illustrates how two generalist mutants compare with their ancestors across the range of environmental noise encountered in the simulated landscape. Changing the slope parameter a in the nonplastic ancestor can improve fitness in Environment 2 but produces maladaptation in Environment 1; when developed with greater environmental noise, the benefit of this mutation is improved and the cost lessened, both in absolute terms and in comparison to the ancestor (Figure 4b). This mutation would, therefore, be deleterious in low-noise environments and neutral or beneficial at higher levels of developmental noise. Another pattern visible in this example is that the nonplastic ancestor is invariably much more fit in Environment 1 than in Environment 2, and the plastic mutant is always a relative generalist, with about equal potential performance in each environment. A second example slightly complicates this picture: a highly plastic mutant can function as a specialist for the novel Environment 2 at low levels of noise, but display more equitable fitness across both environments at very high levels of noise (Figure 4c,d). This example also illustrates a hypothetical pathway toward the evolution of an adaptively plastic generalist: a plastic mutant might initially function as a specialist in Environment 2, then serve as an ancestor for a generalist with equal plasticity (equal a) but more equitable performance across environments.

The examples in Figure 4 motivate caution in attempting to infer the niche of a model organism based solely on its genotypic parameters. Specifically, a specialist in a low-noise environment can behave as a generalist in a high-noise context, and plasticity can be adaptive by producing a specialist on the novel environment, rather than a generalist. Bearing these caveats in mind, we can use these niche predictions to formulate hypotheses about how environments interact with the developmental system to facilitate the evolution of plasticity. Figure 5 shows two example populations in which, according to these predictions, generalism arose and came to exclude both the ancestor and evolved specialists for Environment 2. In both examples, the ultimate descendants show slopes approaching one and intercepts approaching zero, which should produce optimal plasticity at any level of environment noise. In each case, these highly plastic genotypes result from a number of refining mutations, forming a chain of

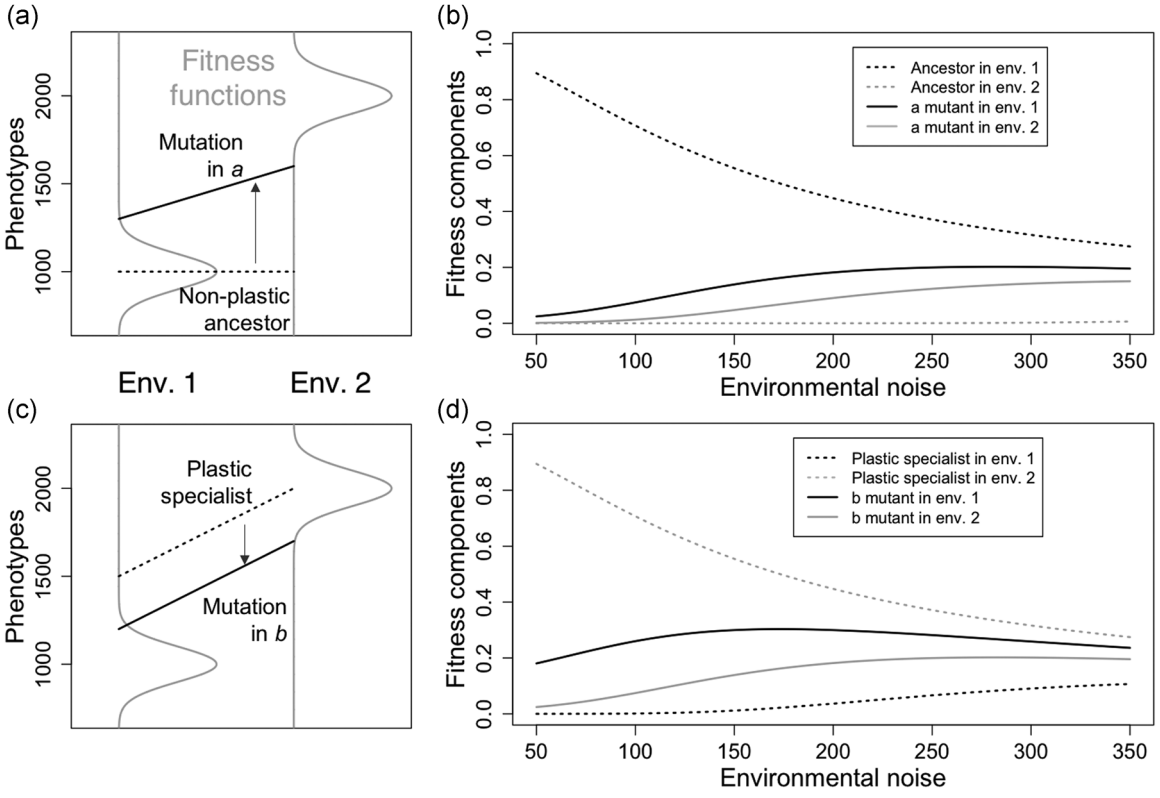

FIGURE 4 (a) Illustration of the reaction norms of the ancestral specialist (dotted line) and a potential mutant in which the a parameter has increased from 0 to 0.3. Gray lines show the fitness function in each environment. (b) Fitness in each environment for both mutants over the range of values of environmental noise. Values were calculated based using Equation (4) with $\sigma^2_{\text{opt}} = 5{,}000$. (c) As in (a) but the Environment-2 specialist has parameters $a = 0.5$ and $b = 1{,}000$, and the mutant changes the value of b to 700. (d) Fitness in each environment for each genotype shown in (c)

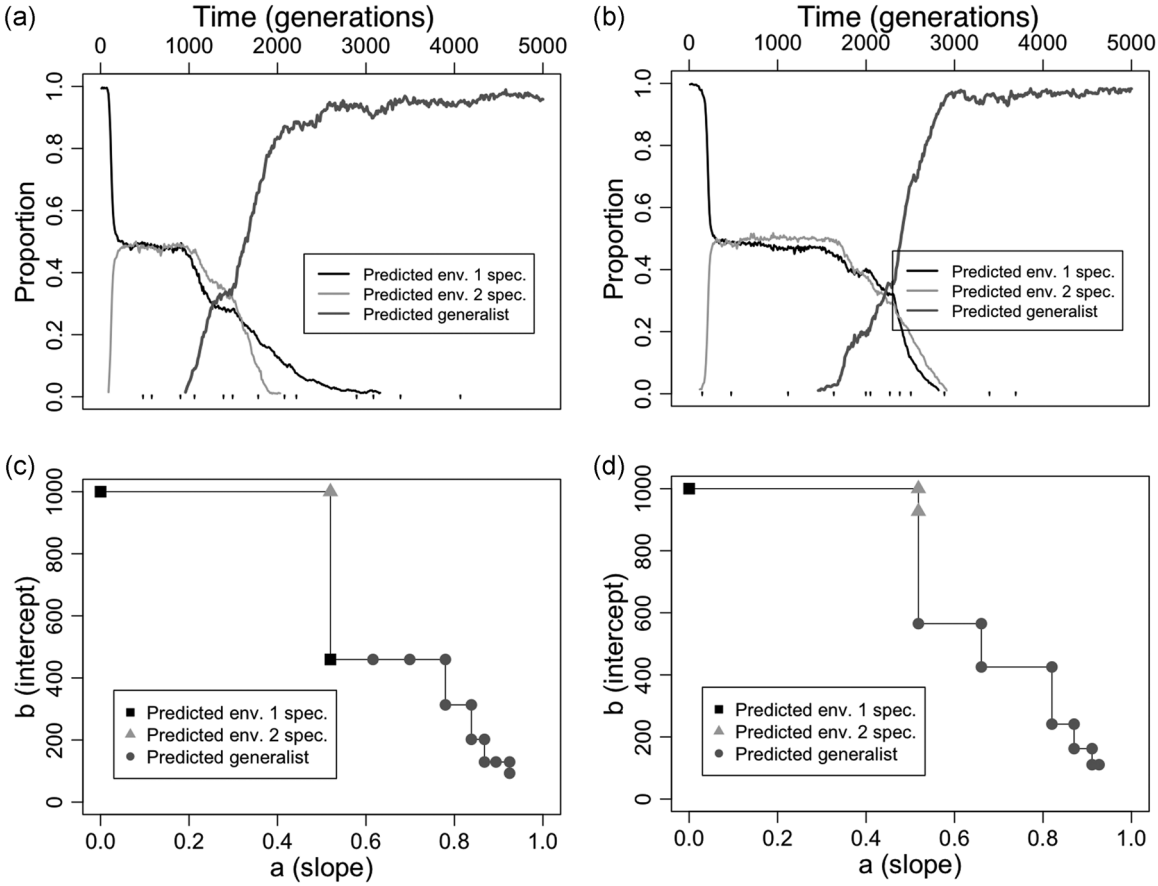

FIGURE 5 (a,b) Frequencies of predicted specialists and generalists over time for two example populations in which generalism evolved. Predicted niches are based on a genotype's comparative fitnesses in each environment for the level of environment noise at which it evolved. (c,d) Genetic parameters for the genotypes along the line of descent of the most common genotype at generation 5,000 for the two populations in (a,b). Symbols indicate the predicted niche of each genotype; the origination time of each mutation is indicated by the tick marks in (a) and (b)

ancestors that are predicted to derive fitness from both environments. However, the predecessors to these plastic generalists are more varied and suggest a complex dynamic. In both examples, a plastic genotype predicted to be an Environment-2 specialist is ancestral; in one example, a derived Environment-1 specialist forms a bridge between this ancestor and the first plastic generalist.

3.3 | Developmental and ecological prerequisites for the evolution of plastic generalists

To investigate the evolutionary processes suggested by these examples, I first quantified how often evolved specialists on Environment 2 were plastic, as opposed to carrying a mutation in their intercept parameters. Out of 187 observed Environment-2 specialists that achieved a maximum frequency of at least 0.02, 131 (70%) had changes only in the a parameter, indicating that they had evolved to become plastic. Of the remainder, 47 showed change in the intercept term but not the slope, while nine showed changes in both parameters. Specialists in the novel environment could, therefore, evolve in ways that introduced either strong environmental sensitivity or maintained the ancestor's insensitivity. These 187 successful specialists were spread across all 100 replicate populations, suggesting that distinct specialist genotypes temporarily coexisted or arose serially in some replicates. To examine the causal influence of specialist genotypes on the evolution of plasticity I quantified how often generalism evolved, and whether its evolution was strictly dependent on the presence of a plastic specialist ancestor. Thirty-nine of the 100 replicate populations evolved to be dominated by generalists (i.e., the combined proportion of predicted generalists exceeded 0.5). In every case, the line of descent included an evolved Environment-2 specialist that was plastic, rather than a specialist evolved via a mutant in the intercept parameter. This supports a model in which the particular type of novel-environment specialist that happens to evolve in a population determines whether plasticity can

readily emerge. I next evaluated whether the pattern seen in Figure 5a,c was general: did the line of descent typically include a secondarily evolved specialist on Environment 1? This was the case for 82% (32/39) of the instances of successful generalism.

The finding that a secondarily evolved specialist on the initial environment often served as a stepping-stone to generalism is explicable genetically by inspection of Figure 3: a large decrease in the intercept term of a plastic Environment-2 specialist can produce a genotype that it best-adapted to Environment 1 but poised to also exploit Environment 2 after a subsequent mutation increases the slope parameter. However, the emergence of such genotypes is ecologically puzzling because the ancestral genotype is perfectly adapted to Environment 1 and would presumably exclude less well-adapted, secondarily evolved competitors from that niche. One hypothesis that might resolve this paradox is that the actual niche of this supposed specialist might be more general. A second hypothesis is that these genotypes are not advantageous, even locally, but represent deleterious mutations that never achieve high frequencies, and are only found on the line of descent because their enable a second, beneficial mutation conferring an adaptive generalist phenotype. Finally, inspection of Figure 2 suggests a third hypothesis: secondarily evolved specialists may be growing within clusters of Environment 1 that are inaccessible spatially to the bulk of the ancestral population.

To better understand these ecological dynamics of competition among genotypes, I traced the population size and realized niche of each genotype on the line of descent of those populations that evolved a high frequency of generalism. The realized niche, measured as the proportion of a genotype's reproductive output derived from Environment 2, was calculated directly from the fitnesses of all individuals of that genotype, rather than predicted as described above. Viewed through this lens, a genotype's niche could change as it spread through the landscape and experienced competition with other lineages. Figure 6 shows the peak population size of each genotype that was classified as a secondarily evolved Environment-1 specialist, as well as realized niche of that genotype when it was most prevalent. It is evident that some of these putative specialists are actually deriving a substantial proportion of their fitness from Environment 2, and these genotypes also tend not to reach substantial numbers. However, other examples produce substantial subpopulations primarily on patches of Environment 1. Inspection of the spatial locations of these more abundant mutants suggested that they primarily arose near the range margins of the ancestral specialist and spread along or expanded those margins.

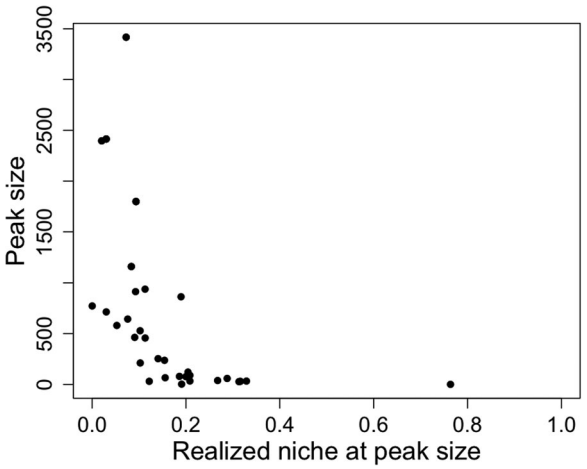

FIGURE 6 Maximum subpopulation size of secondarily evolved Environment-1 specialists and their realized niche, defined as the proportion of their fitness derived from Environment 2, at that peak

However, the edge of a genotype's range was sufficiently labile to make it difficult to quantify this observation.

Finally, I sought to clarify whether predicted generalists did, in fact, gain fitness by using both environments. Figure 7 plots the realized niche measurements for each genotype on the line of descent, using the two example populations plotted in Figure 5. These examples are representative of the qualitative pattern seen in other populations that evolve generalism: successive generalist genotypes tend to favor one environment or the other and can coexist for moderate periods of time alongside related generalists with different biases. Each line represents a single genotype, meaning that vertical movement of a line represented ecological, not evolutionary change; specifically, a range shift or expansion that changes the distribution of environments encountered by that subpopulation. These figures illustrate that successive refinement of the dominant generalist genotype occurs in the context of dynamic repartitioning of the niche through both evolution and range shifts.

4 | DISCUSSION

A primary result here that extends and supports a finding from related previous work (Draghi, in review) shows that developmental noise can actually aid the evolution of plasticity under certain circumstances. While this previous study demonstrated this argument in a highly simplified ecological context, the results shown here show that noise can stimulate the evolution of plasticity even when the fitness costs of noise reduce the local population size in high-noise environments. Although many more organisms reside in low-noise environment

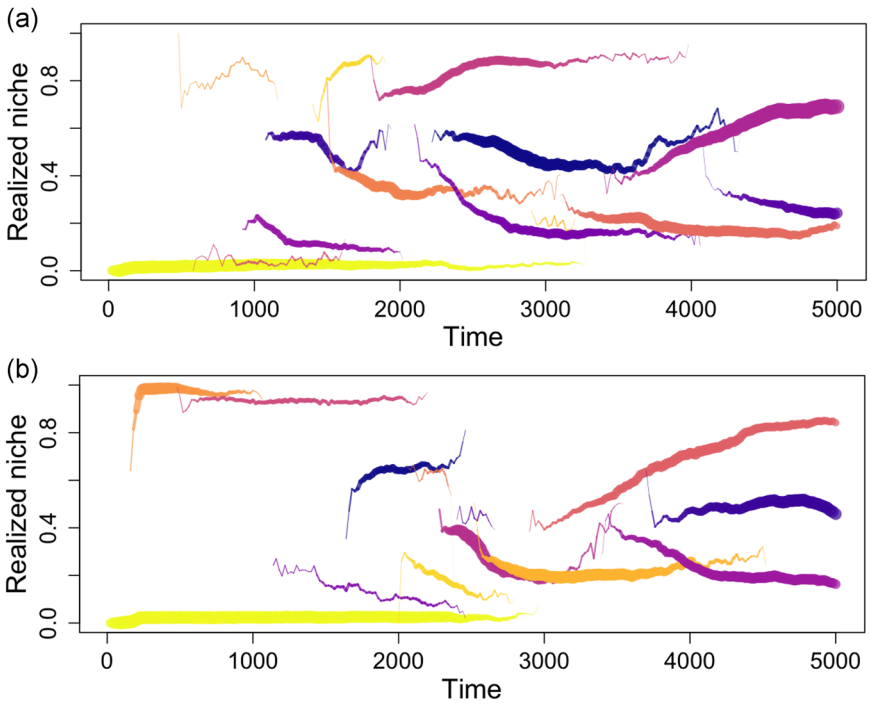

FIGURE 7 Realized niches, defined as the proportion of their fitness derived from Environment 2, of the genotypes on the line of descent of the dominant members of the final populations. (a) and (b) show the same populations as Figures 5a,c and 5b,d, respectively. Widths of each line are proportional to the square root of that genotype's frequency, and serve as a scaled index of abundance of each genotype [Color figure can be viewed at wileyonlinelibrary.com]

(Figure 2), and those environments are the most effective incubators of novel specialists (Figure 3), the association between noise and the origination of plasticity is still quite strong (Figure 3). A second, weaker pattern is that successful plastic mutants are more likely to arise in areas where the novel environment is quite common, again despite the paucity of organisms that live and reproduce in these areas. A general prediction emerging from these results is that variation in how selection acts on new, innovative mutations may dominate over the influences of demography, perhaps making innovations more likely at a population's periphery rather than in the more populous core.

While only a few other models of the evolution of plasticity have focused on developmental noise or instability, their conclusions and approaches have been quite different (Scheiner, 2014b). One factor that might account for this difference is that developmental noise is often viewed as an inherent cost of plasticity (DeWitt et al., 1998; Scheiner, Caplan, & Lyman, 1991; Tonsor et al., 2013). This viewpoint is based on the idea that the sensitivity to the environment required for plasticity will necessarily introduce more noise into development via any randomness or variation in the cue. One assumption of this argument is that noise is always disfavored because the resulting developmental instability produces a mismatch between the genetically determined phenotype and the optimum phenotype to which the population has adapted. However, random variation in phenotypes can be beneficial when environmental optima are difficult to predict, and this type of adaptive response has been studied extensively under the umbrella of the term "diversifying bet-hedging" (Frank & Slatkin, 1990; Starrfelt & Kokko, 2012). A number of recent papers have combined these to examine how bet-hedging, developmental instability, and phenotypic plasticity interact. Scheiner and Holt (2012) showed that extreme plasticity could evolve as a form of bet-hedging, and follow-up modeling work showed that developmental instability and plasticity could act as mutually exclusive strategies (Scheiner, 2014a, 2014b). Other recent approaches have examined how uncertainty in cues affects the relative value of bet-hedging and plasticity (Donaldson-Matasci, Bergstrom, & Lachmann, 2013) and empirical work has begun to disentangle how genotypes might vary in the degree to which they use each strategy (Simons, 2014). Other approaches have modeled how a bet-hedging benefit allows new regulatory connections to evolve even in the absence of a correlation between that regulatory signal and optimal phenotypes (Maxwell & Magwene, 2017; Wolf, Silander, & van Nimwegen, 2015). These latter studies provide a perspective on the relationship between developmental noise and plasticity that complements the results here: in both, developmental instability provides a form of generalism by allowing at least some individuals to exploit several of the resources that they encounter. However, a key departure from these studies is that the results here emphasize how developmental noise helps a mutant with imperfect plasticity successfully compete against its ancestor, providing an evolutionary path by which subsequent mutations can produce a more refined form of plastic generalism.

Here I created a scenario in which the ancestral genotype was well-adapted to one environment but poorly adapted to a second environment that was concentrated at the margins of its range. This set-up, along with the strong limitation of linear dependence between cues and plastic responses, created a major constraint on the evolution of plasticity: any mutant that changes plasticity will have pleiotropic effects, and in the initial genotype such a pleiotropic mutant must decrease fitness in the ancestral environment. The scenario of a well-adapted population encountering a new environment is found elsewhere in the literature on plasticity: for example, Via (1987) examined this case in a quantitative-genetics model that also included developmental instability. While environmental noise played a significant role in helping plastic generalists evolve in the results presented here (Figure 3), the genotypes of evolved specialists on the novel environment emerged as a critical ingredient in how evolution bypassed constraints on plasticity. Plasticity was one mutational pathway by which organisms could drastically change their phenotypes to match the optimum of the novel environment, and lineages that took this path to specialism laid the foundations for the further evolution of genotypes that applied that plasticity to a generalist niche (Figures 4, 5, and 7). Starting with a well-adapted specialist reorients the question being answered by the model: rather than generally addressing how plasticity can evolve, the results speak more directly to how generalism can evolve via plasticity from a specialist ancestor. The hypotheses generated by these results help demonstrate the potential value of taking this more niche-centric approach to familiar questions about constraint and pleiotropy.

The dynamics of the evolution of plasticity as explored in this model are linked to the genetic underpinning specified for the plastic response: separate slope and intercept terms. While this decomposition of the determinants of plasticity has a long pedigree, it can hardly be motivated biologically. Moreover, the decision is not without consequence: for example, constructing a linear reaction norm from three variables changes the evolution of genetic assimilation (Ergon & Ergon, 2017). One solution to this dilemma is to study more complex models with flexible, emergent functions linking cues to phenotypes (e.g., Draghi & Whitlock, 2012). As deployed here, the core concept that the two-parameter model is intended to capture is constraint via pleiotropic correlations between the expression of the same trait across different environments.

Evaluating the role of spatial structure is a key motivation for this model, and its importance is evident in the role of secondarily evolved Environment-1 specialists in the evolution of plasticity. This particular pathway to plastic generalism seems to benefit from the existence of a margin of underexploited patches of the ancestral environment, surrounded by clusters of the novel environment. One potential follow-up could examine how autocorrelation in the placement of patches of the two environments shapes the range margin, and therefore affects the evolution of plasticity. A more detailed examination of the behavior of the population at its margin would also benefit by exploring the dispersal parameter as well as variants of the model that allowed for long-tailed dispersal kernels. A second ecological dimension deserving of more investigation is the demographic effects of environmental noise. As seen in Figure 2, higher noise leads to a lower population density, which may partially explain why newly evolved specialists tend to arise and succeed near the southern margin of the landscape (Figure 3). However, higher densities also equate to greater competition, and a future study could clarify the role of these demographic considerations in the evolution of specialists and generalists. While previous models have considered the impact of hard selection on the evolution of specialists and generalists (e.g., Van Tienderen, 1991), studies of an explicit landscape could lead to new insights about the interactions of space and demography.

The question of developmental biases in evolution relates to the issue of the evolution of phenotypic plasticity both directly and conceptually. On the most basic level, plasticity represents a sensitivity of the developmental processes to the environment; the evolution of plasticity requires a change in how information is processed during development that must bias how mutations can affect traits. Understanding plasticity is, therefore, one avenue toward a larger comprehension of how genotype-phenotype maps are shaped by evolution, and in turn, direct its course by biasing the spectrum of mutational effects on phenotypes. One way to appreciate this bias is to examine the role of pleiotropy in this model: regardless of the genotype, any mutation in the slope or intercept parameter is clearly pleiotropic. However, the degree of constraint imposed by this pleiotropy changes with the specific, quantitative nature of that pleiotropy, with plastic Environment-2 specialists able to find mutations that are pleiotropic but still adaptive.

Conceptually, plasticity is often viewed as an ideal solution to the problems of heterogeneity across environments, and studies focus on the constraints that prevent nature from realizing this ideal. This conceptual framing mirrors that of developmental variability and bias, in which pleiotropy and constraints are viewed as deviations, requiring explanations, from an ideal, isotropic

distribution of mutational effects (Gould, 2002). Each viewpoint references an impossible ideal which perhaps limits our ability to see that constraints and biases are intrinsic to developmental systems, and not pathologies in need of comparison to a version of biology without development. An alternative is to study how variability and plasticity arise out of the assembly of genetic elements to answer adaptive challenges. Here I address both how constraints can emerge from development and how selection in a complex landscape can find ways around those constraints, showing how constraints can be relevant without being absolute.

ACKNOWLEDGMENTS

The author thanks Holly Kindsvater and the editors of this special issue.

CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

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How to cite this article: Draghi J. Developmental Noise and Ecological Opportunity Across Space Can Release Constraints on the Evolution of Plasticity. *Evolution & Development*. 2019;e12305. https://doi.org/10.1111/ede.12305