Received: 11 May 2023 | Accepted: 7 July 2023

DOI: 10.1111/ede.12452

RESEARCH ARTICLE

WILEY

Niche construction and the environmental term of the price equation: How natural selection changes when organisms alter their environments

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Funding information
John Templeton Foundation, Grant/Award Number: 61369; Konrad Lorenz Institute for Evolution and Cognition, Grant/Award Number: Visiting Fellowship; National Institute of Food and Agriculture, Grant/Award Number: 2019-33522-30064

Abstract

Organisms construct their own environments and phenotypes through the adaptive processes of habitat choice, habitat construction, and phenotypic plasticity. We examine how these processes affect the dynamics of mean fitness change through the environmental change term of the Price Equation. This tends to be ignored in evolutionary theory, owing to the emphasis on the first term describing the effect of natural selection on mean fitness (the additive genetic variance for fitness of Fisher's Fundamental Theorem). Using population genetic models and the Price Equation, we show how adaptive niche constructing traits favorably alter the distribution of environments that organisms encounter and thereby increase population mean fitness. Because niche-constructing traits increase the frequency of higher-fitness environments, selection favors their evolution. Furthermore, their alteration of the actual or experienced environmental distribution creates selective feedback between niche constructing traits and other traits, especially those with genotype-by-environment interaction for fitness. By altering the distribution of experienced environments, niche constructing traits can increase the additive genetic variance for such traits. This effect accelerates the process of overall adaption to the niche-constructed environmental distribution and can contribute to the rapid refinement of alternative phenotypic adaptations to different environments. Our findings suggest that evolutionary biologists revisit and reevaluate the environmental term of the Price Equation: owing to adaptive niche construction, it contributes directly to positive change in mean fitness; its magnitude can be comparable to that of natural selection; and, when there is fitness $G \times E$, it increases the additive genetic variance for fitness, the much-celebrated first term.

KEYWORDS
adaptive plasticity, eco-evo feedbacks, ecosystem engineering, genotype by environment interaction, habitat choice

1 | INTRODUCTION

Starting with the work of R. A. Fisher, evolutionary genetic theory has provided the guiding framework for investigating key factors in the process of adaptive evolution. Within this framework, two key features of populations have been most prominent: mean fitness, W, and the additive genetic variance for fitness, V_g. Mean fitness is often considered a summary measure of the degree to which an organism is adapted to its current environment, and because the additive genetic variance of fitness governs the rate and magnitude of gene frequency change in response to natural selection, this component of genetic variance has a direct relationship to change in mean fitness (Hill & Kirkpatrick, 2010). In his "Fundamental Theorem of Natural Selection," R. A. Fisher (1930) specified this relationship:

> *The rate of increase in fitness of any organism at any time is equal to its genetic variance in fitness at that time*. (Fisher, 1930, p. 37)

Based on this central insight, along with supporting results from artificial selection studies in animal and plant breeding (Crow, 2010; Hill & Kirkpatrick, 2010), the additive genetic variance in fitness has held center stage in evolutionary biology, both in theory and in practice, for nearly a century. Thus, although the "fit" between organism and environment is widely acknowledged to be the source of fitness variation among individuals within populations, it is the genetic component of this fit, not the environmental component, that has been the primary focus of evolutionary theory (see Hill & Kirkpatrick, 2010; Queller, 2017). (Although fitness tends to be highly context dependent, Fisher famously eliminated context-dependence from his definition of genetic variance by including contextual effects by a such as dominance, epistasis, and frequency-dependence with environmental effects on fitness; Ågren, 2021; Crow & Nagylaki, 1976; Frank & Slatkin, 1992; Kimura, 1958; Queller, 2017; Wade & Goodnight, 1998; Wright, 1980).

The contribution of change in the environment to change in mean fitness has been more difficult to identify and formalize, and it tends to be ignored in most theoretical treatments. A component of the change in mean fitness owing to environmental change was not explicit in Fisher's "Fundamental Theorem," although he discussed how its effects were likely to act in opposition to those of natural selection. Fisher saw natural selection and its positive effect in increasing a population's mean fitness (W) as self-limiting, since a population's improving adaptedness would cause a concomitant process of intensifying competition for dwindling environmental resources:

> *Against the action of Natural Selection in constantly increasing the fitness of every organism.... is to be set off the very considerable item of the deterioration of its inorganic and organic environment* ...[because] *an increase in numbers of any organisms will impair its environment*. (Fisher, 1930, pp. 45–46)

In recent decades, evolutionary biologists have relied on the Fundamental Theorem as expressed by the Price Equation (Price, 1972) to mathematically partition the change in mean fitness into effects of natural selection and of environmental change (see discussion and references in Lively & Wade, 2022). The second term of the Price Equation offers an opportunity to provide a formal description of the effect of environmental change on changing mean fitness. However, until recently (see e.g., Gandon & Day, 2009; Lively & Wade, 2022; Wade, 2022), little has been done with this second term for a variety of reasons. In part, this is because it is generally posited that generational changes in the environment affecting W tend to be near zero—that is, it is assumed that environmental changes are both small and trendless (i.e., without direction), such that environmental variation at the individual scale can be viewed as noise. In part, it is because any systematic changes in mean fitness caused by environmental change in a particular direction, such as the increasing oxygen concentration of the Earth's atmosphere over geological time or present-day climate change, are seen as governed by processes lying outside the bounds of evolutionary theory in the domain of ecology. And, in part, it is because environmental change at the scale that influences individual fitness is often considered to be too rapid and transient to be relevant on the gradualist timescale of evolutionary genetic change (see Carroll et al., 2007 for discussion). For these reasons, the second term in the Price Equation describing the impact of the environment's change on mean fitness change has generally been ignored. Indeed, this step is considered essential to maintaining the simplifying focus on (additive) genetic variation that is the "hallmark" of fundamental evolutionary theorems, as explained in a recent, synthetic review:

> ...All of these theorems, in their usual forms, require the assumption that the second term of Equation (1) *[the Price equation] is near 0 or at least that it can usefully be set aside as being of secondary interest*. (Queller, 2017, p. 347)

although such foundational theorems

> *...will be inexact to the extent that there are forces other than selection, such as mutation or environmental change*. (op. cit., p. 349)

There are indications, however, that a reassessment of this secondary role for the second term might be in order. Day and Bonduriansky (2011) used the framework of the Price Equation to extend the concept of inheritance to include extra-genetic mechanisms (e.g., DNA methylation, parental effects [see also Wade, 2022], and cultural inheritance). In the context of interspecific interactions Day et al. (2020) and Lively and Wade (2022) showed that, when other species are a key component in the environment of a focal organism, coevolution can be viewed as feedback between the Price Equations of different but interacting species. In pairwise species interactions, the adaptive evolution of one species may usefully be seen as a change in the environment of the other, and vice versa. When viewed in this way, changes in the environment of one species due to selective change in the other can have as large or larger effects on mean fitness than those arising from selection on the species itself (Day et al., 2020; Lively & Wade, 2022). This unexpected result reveals that a nonzero second term can have a substantial impact on evolutionary change in W, suggesting that empirically plausible sources of directional environmental change merit theoretical attention.

An ubiquitous feature of organisms is the way their life-histories and behaviors alter their encounters with the environment (Brodie, 2005; Odling-Smee et al., 2003; Sultan, 2015), a property broadly termed *niche construction* (Lewontin, 1985, 2000). There are three general categories of niche constructing activities: organisms may preferentially move from one environment to another; they may modify their environments; and they may develop differently in alternative environmental conditions in ways that mediate their experience of those conditions. A general theory of such niche-constructing traits might be developed using the second term of the Price Equation. In this paper, we present a simplified version of what such a theoretical treatment of niche-constructing traits might look like and what insights it might provide. Inevitably, organisms change their environment in negative ways, including with respect to population growth rate. That is, some "niche constructing" activities are neutral or maladaptive for the organism, for instance resource depletion or the release of waste and other metabolic byproducts. However, other familiar activities of organisms well-studied by evolutionary, behavioral and ecosystem ecologists comprise *adaptive* modifications of the environment, such as relocating to more favorable habitat patches, building thermally protective burrows, and plastic development of defenses that prevent predation (such that they experience their environment as predator-free). These adaptive niche constructing traits require that an organism has the capacity to assess its environment and subsequently alter its behavior, life-history, development or physiology in functionally appropriate ways. In a population with a given spatial distribution of environments, all three aspects of adaptive niche construction can be viewed as altering the frequency distribution of environments that a population encounters. Moreover, they alter the distribution of environments in a way that increases mean fitness by increasing the relative frequency of the population's encounters with more favorable environments. In other words, niche constructing traits of organisms may contribute to a nonzero, directional environmental change and thereby contribute to the second term in the Price Equation.

Because niche constructing traits (henceforth *NCTs*) change the distribution of environments experienced by a population, it seems useful to ask: what is the impact of such traits on selective evolution of both the niche constructing traits themselves *as well as* other genes in the population? That is, just as changing the effective population size (N_e, see Falconer & Mackay, 1996) or changing the mating system (say from outcrossing to selfing) affects the evolution of all genes in a genome (Charlesworth, 2009), a trait that affects the distribution of environments that a population experiences also affects the rest of the genome. To highlight this feature of adaptive niche construction, our focus is two-fold: (1) *the evolutionary origin* of a capacity to adaptively alter the experience of environments (i.e., the evolution of NCTs); and, (2) *the selective consequences for other genes* of such a capacity. We use a simple 2-environment, haploid population-genetic model to consider each of three different categories of adaptive niche constructing traits:

(1) traits that change the frequency of environments encountered by allowing individuals to move between them (*habitat choice*, Donohue, 2005; *relocation niche construction*, Odling-Smee et al., 2003);
(2) traits that change the actual frequency of environments by converting a low fitness environment into one of higher fitness (*ecosystem engineering*, Jones et al., 1997; *perturbation niche construction*, Odling-Smee et al., 2003; *habitat construction*, Sultan, 2015);
(3) traits that alter the frequency of the environments that individuals experience by means of adaptive phenotypic plasticity or polyphenisms (*experiential niche construction*, Chiu, 2019; Sultan, 2015).

We use the Price Equation to examine the process of adaptation to spatially variable environments by *NCTs* and how the evolution of such traits affects other genes. To do this, we take advantage of the structure of the Price Equation. Namely, we hold the additive genetic variance (i.e., the first term) constant and alter the second, environmental variance term, just as Fisher held the second term of his Fundamental Theorem constant and altered the first. We use this procedure and our genetically simplified models to ask two questions: (1) how do adaptive NC traits evolve in response to variable environments; and, (2) what are the consequences of adaptive NC for other traits undergoing natural selection in response to that same variable environment, particularly in the widespread case when there is genotype-by-environment interaction (G × E) for the fitness effects of those genes rather than strictly parallel genic fitness differences across environments (see Des Marais et al., 2013; Fry et al., 1996).

Conceptually, our approach shares much in common with other models that have investigated local adaption to a varying environment together with individual choice of particular habitat patches (e.g., via habitat choice, Edelaar et al., 2008, 2017; Ravigné et al., 2009), organismic changes to the environment ("environmental engineering" or habitat construction, e.g., Edelaar & Bolnick, 2019; Laland et al., 2016; Odling-Smee et al., 2003, 2013), or adaptive plasticity that mediates the organism's phenotypic encounter with its environment. These three ways of modifying the distribution of selective environments that an organism experiences were nicely summarized by Edelaar et al. (2017) as either the organism changing its environment to match its phenotype (habitat choice or habitat construction), or the organism changing its phenotype to match its environment (adaptive plasticity) (see also Odling-Smee et al., 2003; Sultan, 2015; and references therein). Our starting point or null hypothesis is local adaptation occurring via natural selection acting on a non-habitat-choosing, non-niche-constructing, non-plastic set of two alternative genotypes in an environment that includes two possible states (E_1 and E_2) (see Edelaar et al., 2017 for another modeling approach). Average fitness for the two-genotype "population" is higher in E_1 than it is in E_2, as might be the case for habitat patches that differ in some aspect of environmental quality relevant to the organisms in question such as levels of a key resource or predator presence versus absence. The cases we investigate vary with respect to the presence or absence of genotype-by-environment interaction (G × E) for fitness and the type of fitness G × E (scale G × E or crossover G × E, Figure 1). We use our models to ask: How does the adaptive process change when we introduce the genetic possibility that an organism can either choose its habitat, modify its habitat, or or modify its phenotype in response to its habitat?

In the sections below, we (i) introduce the Price Equation and (ii) describe how mean fitness changes in a spatially variable environment with and without G × E, in the absence of niche constructing traits. Here, we show that even where genotypic fitnesses trade-off between alternative environments (i.e., fitness G × E of the crossover type), mean fitness, W, changes solely as a function of the additive genetic variance for fitness (V_{Add}).

In the subsequent three sections, we introduce in turn each of the NC traits—habitat choice, habitat construction, and adaptive plasticity—and show how each alters the null model. In each of these sections we show how the NC trait interacts with the distribution of environmental fitness variation to accelerate, retard, or leave unchanged the null process of local adaptation to biotic or abiotic factors by natural selection. We find that the greater the difference in mean fitness between environments, the stronger is the selection favouring the evolution of niche constructing traits. Thus, adaptive niche construction can be understood as the capacity of an organism to reduce the deleterious effects of environmental variation by altering the distribution of environments it experiences toward environments with higher mean fitness. This is an important departure from the classic results of studies that characterize selective response in spatially varying environments where the distribution of environments is considered as fixed or given. Interestingly, such studies have found that adaptation to a fixed distribution of fitness-altering environments can lead to a *decrease* in mean fitness (reviewed in Felsenstein, 1976).[1]

Finally, we evaluate the impact of the NCTs on evolutionary dynamics at other trait loci. We find that, as the experienced frequency of alternative environments is changed by the evolution of adaptive niche constructing traits, the additive genetic variance of all other (non−niche constructing) trait loci can increase when those loci show genotype by environment interaction for fitness. This increase in additive genetic variance accelerates the process of adaption to the environments favored by niche construction. In this way, the evolution of an adaptive capacity to alter the distribution of environments experienced by an organism can contribute to the rapid refinement of alternative adaptations to different environments, depending on the precise pattern of genotype by environment fitness variance. One of our central findings is that adaptive NCTs contribute importantly to additive genetic variation for other traits—the very quantity captured by the canonical First Term of the Price Equation—so as to accelerate their rate of selective response.

2 | MODELING APPROACH

2.1 | The Price Equation

We use the Price Equation to track adaptive evolution from generation to generation (defined as simply the mean fitness given the environment after selection, minus the mean fitness given the environment before selection). Following Price (1972) and Frank and Slatkin (1992), we use prime notation such that W′ and E′ refer respectively to mean fitness and the average environment in the next generation. The total change in fitness in one generation thus equals:

$$\Delta W = W'|E' - W|E. \quad (1)$$

Next, by adding and subtracting the term W′|E, we can create two separate terms from Equation (1), one quantifying the effect of natural selection on W, and the other quantifying the effect of environmental change on W. Each term contributes to the total change in fitness:

$$\Delta W = (W'|E - W|E) + (W'|E' - W'|E). \quad (2a)$$

$$\Delta W = \Delta W_{\text{Natural selection}} + \Delta W_{\text{environment}}. \quad (2b)$$

If the environment is held constant sensu Fisher (1930), the first term ($\Delta W_{\text{Natural selection}}$) equals the additive genetic variance, and the genic effects (i.e., the effect of A vs. a) on fitness remain constant. The second term, $\Delta W_{\text{environment}}$, contains environmental effects plus all of the genetic complications (dominance, epistasis, etc.) (Fisher, 1930). Note that as these factors change E to E′, they will also cause changes in W when (a) change in fitness due to the environment is nonzero ($\Delta W_{\text{environment}} \neq 0$), or (b) the change to E′ changes the genotypic fitness differences that cause selection (i.e., $\Delta W_{\text{Natural selection}}$). Importantly, just as Fisher derived $\Delta W_{\text{Natural selection}}$ by imagining that the frequencies of alternative environmental conditions within a population remains constant (a step repeated in standard models that set E′ = E), in our characterization of the second term, $\Delta W_{\text{environment}}$, we imagine that the gene frequencies in the first term are kept constant.

In the next section, we introduce a two-allele, haploid model of evolution in two environments (i.e., in a population with spatial environmental variation) with and without G × E for fitness and use this simple model to derive both terms of the Price Equation, $\Delta W_{\text{Natural selection}}$ and $\Delta W_{\text{environment}}$.

2.2 | G × E for fitness

We introduce genotype by environment interaction (G × E) for fitness to our model, a pattern of genotypic variation that is "a general feature" of continuously varying traits such as fitness in natural systems (Barton & Turelli, 1989, p. 345, see also Des Marais et al., 2013; Gupta & Lewontin, 1982; Haldane, 1946; Sultan, 2015 and references therein; e.g., Fry et al. [1996] determined that at least 50% of naturally occurring new mutations in *Drosophila melanogaster* lab populations exhibited fitness G × E). We draw on the "norm of reaction" concept to illustrate the presence or absence of G × E using a pair of hypothetical haploid genotypes (Falconer & Mackay, 1996; Scheiner, 1993). Then, for each of the possible patterns of fitness variation for the pair of genotypes (shown in Figure 1), we partition the total variance in fitness into its components of genetic, environmental and gene-by-environment (G × E) interaction (Feldman & Lewontin, 1975; Lewontin, 1974). Following Lewontin (1974), for each of the four patterns of fitness variation we show how the additive genetic component of fitness variation is affected by changes in the distribution of environments.

A norm of reaction depicts the phenotypes produced by a single genotype, when that genotype is reared across a specified set of environments (see Sultan & Stearns, 2005). Our approach here is to use a very simple definition of genotypes, alternative haploid alleles A and a, and compare their fitness phenotypes across a similarly simplified set of alternative environments, E_1 and E_2. To draw the clearest relationship between natural selection and gene frequency change, the phenotype of our reaction norms is genotypic fitness; in each environment the genotype with the higher average fitness value will be favored by natural selection. Each panel shows how patterns of genotypic fitness and hence selection change when the genotypes encounter E_1 versus E_2.

Consider first, Figure 1a. Average fitness for the set of two genotypes is higher in E_1 than it is in E_2, as would occur for different quality habitat patches for this organism.

Just as the genic fitness effect, s (or t), is defined as half of the difference in fitness between genotypes A and a within environment E_1, we have defined the environment effect on fitness, e, equal to half the difference in fitness between environments E_1 and E_2. This means that substituting E_1 for E_2 would increase mean fitness by an amount equal to (2e), in the same sense that substituting the A allele for the a allele increases mean fitness by (2s) in E_1 (Figure 1a). (Note that this fitness difference between environments becomes important when we introduce NCTs, which for instance allow organisms to move from E2 to the more favorable E1 where they have higher fitness).

In Figure 1a, the genotypic fitness effect, s, is the same in both environments, resulting in parallel norms of reaction: there is no fitness G × E. Nonparallel norms of reaction indicate G × E for fitness, where the fitness effect of genotypes and/or the fitness difference between them changes depending on the environment. In panels (c) and (d), the genotypic effects are, respectively, (2s) in E_1 but 0 in E_2, and 0 in E_1 and (2s) in E_2. These panels depict *scale G × E*, where the size of genotypic fitness differences changes from one environment to another. Figure 1b depicts "crossover G × E," where the fitness ranking of genotypes is environmentally dependent (i.e., genotypic effects change sign in the different environments). For simplicity here, in panel (b) we show gene effects of equal magnitude but differing in sign. Below, however, we are more general, using effects of different sign and magnitude (s in E_1 and -t in E_2).

For each panel in Figure 1, we calculate the total variance in fitness (V_W) and partition that variance into its three components (Table 1): (1) the additive genetic variance in fitness ($V_{Additive}$); (2) the environmental variance in fitness ($V_{Environment}$); and (3) the

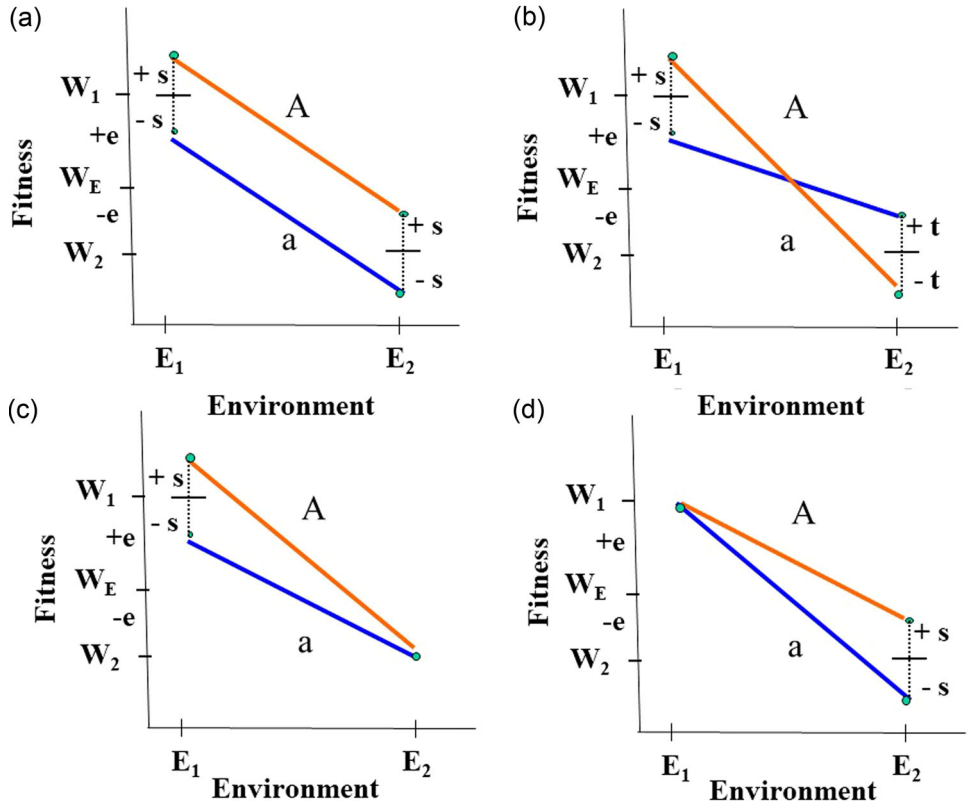

FIGURE 1 A schematic diagram of four possible fitness reaction norm distributions for the genotypes A and a in two different environments, E1 and E2. In (a), there is no genotype-by-environment interaction but there is a genotypic effect on fitness (s) and an environmental effect on fitness (e). In (b), there is a fitness trade-off or crossover G × E; and, in (c and d), there is scale-type G × E for fitness where either E1 or E2 is the selective environment, respectively, and the alternative environment is nonselective (i.e., the genotypes have the same fitness in the nonselective environment). See text for further discussion. [Color figure can be viewed at wileyonlinelibrary.com]

TABLE 1 **Table of fitness variances for the four possible cases shown in Figure 1:** The Columns show the three components ($V_{Additive}$, $V_{Environment}$, $V_{G\times E}$) of the total variance in fitness ($\mathbf{V_W}$), which is the sum of those components, for each of the four sets of norms of reaction shown in the panels of Figure 1.

	(a) No G × E	(b) Crossing G × E	(c) Scale G × E	(d) Scale G × E
$V_{Additive}$	$(2s)^2(pq)$	$(2Z)^2(pq)$	$(2f_1s)^2(pq)$	$(2f_2s)^2(pq)$
$V_{Environment}$	$(2e)^2(f_1f_2)$	$(f_1f_2)\{2e + (p\text{-}q)(s + t)\}^2$	$(f_1f_2)\{2e + (p\text{-}q)(s)\}^2$	$(f_1f_2)\{2e + (p\text{-}q)(s)\}^2$
$V_{G\times E}$	0	$4(pq)(f_1f_2)(s + t)^2$	$4(pq)(f_1f_2)(s)^2$	$4(pq)(f_1f_2)(s)^2$

Note: The parameter, Z, equals ($f_1s - f_2t$) and is the average genic effect for the crossing-type G × E shown in Figure 1b. Note that the average fitness, W_E, which is common to each of the genotypic fitnesses in each environment, does not appear in the components of variance.

genotype × environment variance in fitness ($V_{G\times E}$). Total variance in fitness equals the sum: $V_W = V_{Additive} + V_{environment} + V_{G\times E}$.

We now calculate how the gene frequencies evolve (Δp_A where p_A is the frequency of the A genotype and q_a is the frequency of the a genotype) and how mean fitness evolves (ΔW) given the norms of reaction in Figure 1a. In all cases, we let the frequency of Environment 1 be f_1 and that of Environment 2 be f_2 where $(f_1 + f_2) = 1$. For the moment, we assume that the two genotypes experience environments E_1 and E_2 in proportion to the existing frequency of the two environments (i.e., there is no niche construction, which we introduce in the section below).

With the reaction norms shown in Figure 1a (no G × E) and these assumptions, W_A, the mean fitness of genotype A averaged across both environments equals

$$W_A = f_1(W_1 + s) + f_2(W_2 + s) \quad (3a)$$

$$= (f_1W_1 + f_2W_2) + (f_1 + f_2)s \quad (3b)$$

$$= W_E + (f_1 - f_2)e + s \quad (3c)$$

where W_E is the average fitness and $W_1 = W_E + e$ and $W_2 = W_E - e$. A similar derivation gives us $W_a = W_E + (f_1 - f_2)e - s$.

The *average genotypic fitness*, W_G is $(p_AW_A + q_aW_a) = W_E + (f_1 - f_2)e + (p_A - q_a)s$, and the *relative fitness* of genotype A, w_A, equals (W_A/W_G). After selection, $p'_A = (p_A)(w_A)$. The change in frequency by natural selection, Δp_A, is the difference, $(p'_A - p_A)$:

$$\Delta p_A = (p_Aw_A) - (p_A)(W_G/W_G) \quad (4a)$$

$$= \{p_A(W_E + (f_1 - f_2)e + s) - p_A(W_E + (f_1 - f_2)e + [p_A - q_a]s)\}/W_G \quad (4b)$$

$$= (2s/W_G)(p_Aq_a). \quad (4c)$$

The mean fitness *after selection* is $W'_G = W_E + (f_1 - f_2)e + (p_{A'} - q_{a'})s$, so that

$$\Delta W_{Natural\ selection} = \{W_E + (f_1 - f_2)e + (p_{A'} - q_{a'})s\} - \{W_E + (f_1 - f_2)e + (p_A - q_a)s\} \quad (5a)$$

$$= ([p_A + \Delta p_A] - [q_a + \Delta q_a])s - (p_A - q_a)s \quad (5b)$$

$$= (2\Delta p_A)s \quad (5c)$$

$$= (2s)^2(p_Aq_a/W_G). \quad (5d)$$

From Table 1, row 1, we see that $\Delta W_{Natural\ selection} = V_{additive}/W_G$ as expected from Fisher's Fundamental Theorem. Both Δp_A and ΔW_G are functions of the effect of a gene substitution, 2s, and the genetic variance, pq. The value of $\Delta W_{Environment}$ is 0, because the environment frequencies, f_1 and f_2, do not change.

In Figure 1b, there is crossover G × E for fitness. Here, the average effect of the A allele on fitness, $Z_A = (f_1s - f_2t)$, is an explicit function of the environmental frequencies. When environment 1 is more frequent ($f_1 >> f_2$), the effect of the A allele on fitness in E_1 determines its overall effect on fitness, and vice versa when $f_2 >> f_1$. The changes in gene frequency and in mean fitness by natural selection are now equal to:

$$\Delta p_A = (2Z_A/W_G)(pq) \text{ and}$$

$$\Delta W_{Natural\ selection} = (2Z_A)^2(pq/W_G).$$

However, because the environment frequencies, f_1 and f_2, do not change, $\Delta W_{Environment}$ remains equal to 0 even in this case with trade-offs in genotypic fitness between environments.

For the cases of scale G × E for fitness (Figure 1c,d), there is no selection in E_1 or in E_2, respectively. Setting $t = 0$, the value of Z_A for E_1 becomes (f_1s) for Figure 1c. Similarly, Z_A becomes (f_2s) for Figure 1d. With this change and using the entries in Table 1, we find that $\Delta p_A = (2f_1s/W_G)(pq)$ and $\Delta W_{Natural\ selection} = (2f_1s)^2(pq/W_G)$ for case c and $\Delta p_A = (2f_2s/W_G)(pq)$ and $\Delta W_{Natural\ selection} = (2f_2s)^2(pq/W_G)$ for case d. Since the environment frequencies, f_1 and f_2, do not change, $\Delta W_{Environment}$ equals 0 in these two cases of G × E as well. Because there is only one selective environment in each case, and because both f_1 and f_2 are less than 1, the additive genetic variance for fitness in cases c and d is *reduced* relative to the strictly additive case (panel a) (cf. Table 1). And, importantly, the fitness effect is now an explicit function of f_1 and f_2, the distribution of environments E_1 and E_2. That is, leaving the gene frequencies constant (so that $[p_Aq_a]$ is constant), the additive genetic variance for fitness contributed by the A locus changes if f_1 and f_2 change. However, thus far we have considered only scenarios where the environmental distribution remains constant.

In summary, the expressions for Δp_A and ΔW_G change as the norms of reaction for genotypes at the A locus change (Figure 1):

Panel a: $\Delta p_A = (2\,s/W_G)(p_Aq_a)$ and $\Delta W_G = (2\,s)^2(p_Aq_a/W_G)$

Panel b: $\Delta p_A = (2[sf_1 - tf_2]/W_G)(p_Aq_a)$ and $\Delta W_G = (2[f_1s - f_2t])^2(p_Aq_a/W_G)$

Panel c: $\Delta p_A = (2\,sf_1/W_G)(p_Aq_a)$ and $\Delta W_G = (2\,sf_1)^2(p_Aq_a/W_G)$

Panel d: $\Delta p_A = (2\,sf_2/W_G)(p_Aq_a)$ and $\Delta W_G = (2\,sf_2)^2(p_Aq_a/W_G)$.

The smaller the effect of a gene substitution on fitness ($2Z_A$), the slower are the rates of change of Δp_A and ΔW_G. Comparing panels a, c, d, and b (Figure 1), we see a decline in the effect of a gene substitution from $2s > 2\,sf_1$, $2sf_2 > 2(f_1s - f_2t)$, respectively. In *none* of these cases do the frequencies of E_1 and E_2 change, that is, $E' = E$, and the second term of the Price Equation, $\Delta W_{Environment} = W'|E' - W|E$, must be zero by definition. *When we add the capacity for niche construction to the model in the next section, we are adding a mechanism for changing E to E′.* Doing so changes the action of selection in *all* cases with G × E (Figure 1b–d). Below, we show how the capacity to change the distribution of environments evolves, thereby *adding* an environmental term to the Price Equation as well as changing the long-studied additive genetic variation for fitness (i.e., at other loci), which is its first term. Specifically, we first show how NCTs evolve at a niche constructing locus, C. And, after discussing the evolution of NCTs, we then show how their evolution *also* changes the additive variance for fitness at the A locus.

3 | MODEL OVERVIEW

In a spatially heterogeneous population with more and less favorable habitat patches, organisms may change the frequency of the environments they experience by means of three types of adaptive niche constructing traits or NCTs: choosing their habitat, changing their habitat, or adjusting phenotypically to better suit a given habitat. First, habitat choice changes the distribution of environments an organism experiences from f_1 and f_2 (without choice), to f_1' and f_2' as a result of choosing one environment over the other (Figure 2). The second type of NCT, habitat construction, renders a suboptimal environment more similar to a favorable one. This happens, for example, when desert beetles build trenches perpendicular to prevailing winds to collect moisture from the air that doubles moisture content within the dune ridges they inhabit (Seely & Hamilton, 1976), or the desert rhubarb plant builds its own "mini-oasis" via specialized leaves that capture rainwater and funnel it rootward (Lev-Yadun et al., 2009). As a result, such adaptive habitat-constructing activities also change an organism's distribution of environments, from f_1 and f_2 before it alters its habitat to f_1' and f_2' afterwards. Lastly, we can consider adaptive plasticity in response to an environmental demand as a form of niche construction that effectively changes the environment the organism experiences (*experiential niche construction*, Sultan, 2015; see Chiu, 2019; Walsh, 2015). For example, predator-induced plastic defenses (Kishida & Nishimura, 2004; Petrusek et al., 2009) transform the environment an organism experiences to one that has fewer or no predators. Similarly, a flooded plant that plastically elongates its stem to maintain access to aerial oxygen experiences a plentiful O_2 environment rather than a hypoxic one (Voesenek et al., 2006).

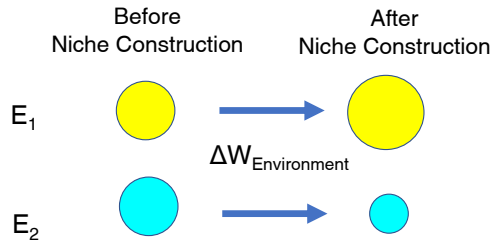

FIGURE 2 A schematic diagram showing the effect of niche construction on the relative frequencies of two environments, E1 (favorable) and E2 (unfavorable); the change in size of the circles represents the change in frequency. This causes a positive change in mean fitness, $\Delta W_{Environment} > 0$. See text for further discussion. [Color figure can be viewed at wileyonlinelibrary.com]

For each of the three categories of adaptive niche constructing traits, we first ask two questions: *What kind of change in the experienced distribution of E_1 and E_2 is favored by selection*?; and *What determines the strength of selection favoring the evolution of the NCT*? We find that, for all NCTs, it is the difference in fitness between environments, the quantity $(W_1 - W_2)$, that governs the evolution of adaptive niche construction. When plasticity is modeled as epistatically regulated, however, its evolution is governed instead by the phenotypic fitness tradeoff between the alternative environments. We next ask: *How does evolution of these adaptive NCTs alter selective dynamics at other trait loci in the organism*?

3.1 | Niche constructing traits and their evolution

i. *The capacity for adaptive habitat choice*: Consider a haploid locus with two alleles, C and c, which occur in frequencies p_C and p_c, respectively. Let individuals bearing the no-choice allele c choose environments E_1 or E_2 at random at the start of a generation. That is, c individuals experience environments E_1 and E_2 in frequencies f_1 and f_2, respectively. In contrast, let individuals with the C allele exert a choice favoring E_1 over E_2 and let m represent the strength of this habitat preference. As a result, C individuals experience E_1 as though its frequency were $f_1' =$

$f_1(1+m)/(1+mf_1)$ and E_2 as though its frequency were $f_2' = f_2/(1+mf_1)$. This is different from the other habitat choice treatments where habitat choice is assumed to be genetically fixed (e.g., Ravigné et al., 2009, p. E145). We adopt this type of "choice" model because we are investigating the evolution of the "capacity for habitat choice" as opposed to optimizing its mean value, so we include the fact that individuals will not make this choice 100% of the time.

The change in experience of the environment, Δf_1, equals the difference $(f_1' - f_1)$ and the change in experience of E_2 must equal - Δf_1 so that the experienced environmental frequencies sum to 1. We find:

$$\Delta f_1 = \{f_1(1+m)/(1+mf_1)\} - f_1 \quad (6a)$$

$$= (m)(f_1 f_2)/(1+mf_1). \quad (6b)$$

This formulation of habitat choice is analogous to a function for mate choice (Kirkpatrick, 2010; Wade, in prep). It has the conceptual advantage that it "looks like" and has an effect analogous to a selection coefficient acting on the frequency of environments. If we let $m > 0$, then we are saying that E_1 is favored over E_2 and, if $m < 0$, then E_2 is favored over E_1. There is no habitat choice and $\Delta f_1 = 0$ when $m = 0$. The term $(1+mf_1)$ is the average preference and by dividing by it, we insure that $(f_1' + f_2') = 1$.

In this version of our model, we are not going to add a separate fitness cost of exerting habitat choice, even though such a cost might be reasonable in some circumstances. We are also assuming that alleles at the NC locus are randomly associated with alleles at the A locus discussed above, at least at first (i.e., there is no linkage disequilibrium). Once selection has acted, however, alleles at the NC and A loci are not randomly associated since independent selection acting on each locus causes "selective interference" (i.e., negative linkage disequilibrium) between them (Feldman et al., 1996). Because this effect is small relative to direct selection acting at each locus (derivation not shown), we do not discuss it further.

If the allele C spreads through a population, we can say that the population has evolved the *capacity for habitat choice*. Now that we have a genetic model of the capacity for habitat choice and we know how much it changes the experienced frequency of environments, we now ask, *will it evolve by natural selection and, if so, how strong might that selection be*? Does a change in experience of E_1 of magnitude Δf_1 increase or decrease the fitness of the habitat-choosing genotype, C? The answer depends upon the norm of reaction for fitness. Consider the additive norm of reaction (Figure 1a), where W_{E1}, mean fitness in E_1, equals $(W_1 + [p_A - q_a]s)$ and is greater than W_{E2} which is $(W_2 + [p_A - q_a]s)$. Note that mean fitness in both environments is a function of (1) $(W_1 - W_2) = 2e$, the environmental effect on fitness shared by all genotypes experiencing an environment (i.e., like 2s, the effect on fitness of an A for a gene substitution, 2e is the effect of a substitution of E_1 for E_2) and (2) the average genotypic effect on fitness within that environment (i.e., the $[p_A - q_a]s$ term). Because genotypes C and c experience environments E_1 and E_2 with different frequencies, they will have different fitnesses as a result of habitat choice. Remembering that $W_E = (f_1 W_1 + f_2 W_2)$, the fitness of genotype c is $W_c = (f_1\ [W_1 + f_2 W_2) = (W_E + [p_A - q_a]s) = W_{mean}$. That is, genotype c has a fitness equal to W_{mean} as a result of its random experience of E_1 and E_2. The fitness of genotype C is $W_C = (f_1' W_{E1} + f_2' W_{E2}) = W_{mean} + \Delta f_1 (W_1 - W_2) = W_{mean} + 2e\Delta f_1$. The average fitness, W_G equals $(p_C W_C + q_c W_c) = W_{mean} + 2ep_C\Delta f_1$. As a result, the change in frequency of p_C by the selection resulting from habitat choice will equal

$$\Delta p_C = (p_C W_C/W_G) - p_C(W_G/W_G) \quad (7a)$$

$$= (p_C q_c)(2e\Delta f_1/W_G). \quad (7b)$$

This means that the selection coefficient of C equals $(2e\Delta f_1)$, the product of how much NC changes the environmental distribution and the fitness effect of that change, that is the substitution of a fraction of the E_1 for E_2. If $\Delta f_1 > 0$ *and* $(W_1 - W_2) = 2e > 0$, then genotype C is choosing the environment with higher fitness and, by doing so, achieving higher mean fitness for itself relative to the alternative allele c, which experiences the environments at random. (This is similar to the movement of an organism toward a peak on a fitness landscape; Tanaka et al., 2020). For this case, $\Delta p_C > 0$ and the capacity to choose one's habitat is adaptive and increases in the population. If C should prefer E_2 the lower fitness environment, then the product, $(-2e\Delta f_1)$, will be less than 0, and "poor" habitat choice will not evolve. The increment in fitness (Equation 6b) achieved by exerting habitat choice depends upon m, the strength of the preference for E_1; upon $(f_1 f_2)$ the variance of the environmental distribution; and upon $2e = (W_1 - W_2)$, the environmental effect on fitness. When the action of natural selection within environments is independent of environmental context, as it is for the simple additive, no G × E case (Figure 1a), then it is only the environment's effect on fitness, 2e, that governs the sign and the magnitude of selection for habitat choice. Next we ask: *How does introducing G × E change this evolutionary scenario*?

Adding G × E to the norm of reaction for fitness (Figure 1b,c or d) changes selection acting on habitat choice. It *does not change* Δf_1, rather it changes the fitness consequences of $\Delta f_1$for both the NC locus and for the A locus (shown in the next section). Consider the trade-off norm of reaction (Figure 1b), where W_{E1}, mean fitness in E_1, equals $(W_1 + [p_A - q_a]s)$ and is greater than W_{E2} which is $(W_2\text{-}[p_A - q_a]t)$. Note that mean fitness in both environments is a function of (1) the environmental effect on fitness shared by all genotypes experiencing that environment (i.e., the W_1 or W_2 terms) and (2) the average genotypic effect on fitness within that environment (i.e., the $+[p_A - q_a]s$ or $-[p_A - q_a]t$ terms). The different experience of environments E_1 and E_2 results in different fitnesses for the genotypes C and c. Remembering that $W_E = (f_1W_1 + f_2W_2)$ and that $Z_A = (f_1s - f_2t)$, the fitness of genotype c is $W_c = (W_E + [p_A - q_a]Z_A)$. The fitness of genotype C is now $W_C = W_E + (p_A - q_a)Z_A + \Delta f_1\{(W_1 - W_2) + (p_A - q_a)(s + t)\}$. The average fitness, W_G equals $(p_CW_C + q_cW_c) = W_{mean} + p_C\ \Delta f_1\ \{(W_1 - W_2) + (p_A - q_a)(s + t)\}$. As a result, the change in frequency of p_C by the selection resulting from habitat choice equals

$$\Delta p_C = (p_CW_C/W_G) - p_C(W_G/W_G) \quad (8a)$$

$$= (p_Cq_c)(\Delta f_1\{(2e) + (p_A - q_a)(s + t)\}/W_G). \quad (8b)$$

With G × E, there are two components to the selection coefficient affecting C: (1) $(2e\Delta f_1)$, the product of how much its choice changes the environmental distribution and the environmental fitness effect; and, (2) the product of how much its choice changes the environmental distribution and the net effect on selection within environments once it does so. A key issue for the evolution of habitat choice when there are both environmental effects on fitness as well as within-environment genetic effects on fitness will *be which is larger*, the environmental effect on fitness, 2e, or the net within-environment genetic effects, (s + t). This is similar to the issue of the difference between genes with major and minor effects on fitness. If the environmental effect on fitness is larger than that of genes within the environment, and if habitat choice increases fitness (i.e., if $\Delta f_1 > 0$ *and* (2e) >0), then genotype C is favored and the capacity to choose one's habitat evolves.

Cases of scale G × E are special cases where there is only one selective environment and the selective environment either has a higher or lower environmental effect on fitness (panels C and D, respectively).

ii. *The capacity for adaptive habitat construction*: Here, our haploid niche constructing locus also has two possible alleles, C and c, occurring in frequencies p_C and q_c, respectively. Let individuals bearing the no-construction allele c, experience environments E_1 and E_2 in frequencies f_1 and f_2, respectively. In contrast, let individuals with the C allele modify E_2 to become like the selective environment of E_1, thereby decreasing the experienced frequency of E_2 from f_2 to $f_2' = (f_2 - \Delta f_2)$ and increasing the experience of environment E_1 from f_1 to $f_1' = f_1 + \Delta f_2$. In this expression for Δf_2, our parameter, m, which was the strength of habitat choice above, takes on a different meaning. Here it denotes the degree to which E_2 becomes like E_1 or, equivalently, the fraction of E_2 that is experienced as E_1 by C genotypes. Note that whereas m, representing choice, can take values greater than 1, here m is defined as the degree or fraction of mitigation of the deleterious environment in the direction of the favorable environment and, as such, cannot exceed 1. The rest of the derivation proceeds as above replacing $+\Delta f_1$ with $-\Delta f_2$. It is harder to argue that habitat engineering is cost-free as we did for habitat choice above and we return to this assumption in the Discussion.

iii. *The capacity for adaptive plasticity*: Niche construction by adaptive phenotypic plasticity is the capacity to adjust the developmental, physiological, or behavioral phenotype in ways that improve the organism's experience of a given set of external conditions ("experiential NC," Chiu, 2019; Sultan, 2015; see also Walsh, 2015). Plastic adjustments take place in response to specific environmental conditions or "cues" that are transduced into biochemical signals to cells and tissues (see references in Sultan & Stearns, 2005; Sultan, 2015). Like other types of niche construction, plasticity can be maladaptive as well as adaptive (i.e., increase the experienced frequency of a lower-fitness environment, which in the case of plasticity would entail producing a phenotype that exacerbates rather than mitigates an environmental stress). We envision two ways that plasticity might evolve as an adaptive NCT.

A simple case could be modeled by a plasticity gene (analogous to the genes posited above) "for" the capacity to express an environment-specific, adaptive phenotype in response to suboptimal conditions. Such a C gene would have two pleiotropic effects, both aligned to facilitate adaptation to variable environments: (1) the ability to perceive such conditions; and, (2) the ability to induce expression of a phenotype partially or wholly adapted to the suboptimal environment. Individuals with a C allele can adaptively adjust their phenotype—for example by, extending their root systems, or re-tooling oxygen-processing tissues—so as to experience their poor environment as a more favorable one. As with the C gene for habitat choice or habitat construction, the plasticity C

gene effectively increases the frequency with which individuals experience the more favorable environment. In this case, the m parameter represents the extent to which the phenotypic adjustment effects this change. Hence, NCT via simple plasticity evolves under selection according to the same dynamics as described above, owing to the mean fitness difference between environmental states.

The capacity for adaptive plasticity may also evolve by means of environment-specific epistasis (Lehner, 2011), through the acquisition of the ability of an environmentally sensitive gene to regulate the expression of a second, previously acquired gene. In such systems, epistatic (gene–gene) interactions evolve that regulate environment-specific or *conditional* expression of traits whose fitness impacts are either positive or negative, depending on the environment. In the evolution of pathogen resistance in plants, for example, genes conferring pathogen resistance often reduce plant growth and reproductive fitness. The *subsequent evolution* of fine-scale regulation of resistance genes, restricting their expression to certain environments, times or tissues, is important to reducing the fitness costs of defense (Karasov et al., 2017; MacLean & Vogwill, 2015). Though complex, this G × G × E mode of gene regulation, in which certain genes turn other genes on or off in environmentally specific ways, is prevalent in natural systems (Lehner, 2011) and characteristic of animal and plant hormones as environment-sensitive regulators of gene expression (reviewed by Dufty et al., 2002; Lema & Kitano, 2013; Sultan, 2015).

We consider the niche-constructing locus C as a hormone-like regulatory gene and the A locus as the regulated or responding gene. We can imagine alternative alleles at locus C, one allele with the ability to control the expression of either the A or the a allele depending upon the environment, and the alternative allele leaving expression at the A locus as depicted in the four patterns of fitness shown in Figure 1. Alternatively, we could hypothesize different kinds of A locus alleles, some capable of joining the control of an existing regulatory gene (for instance by acquiring a new binding site).

Consider first the case without G × E for fitness (Figure 1a) and allow the gene combination, aC, to be neutral instead of deleterious in E_1. (If we had allowed the gene combination aC to be neutral instead of deleterious in E_2, we would replace f_1 with f_2 below.) This results in the following four genotypic fitnesses, each averaged across E_1 and E_2:

$$W_{AC} = W_E + s + (f_1 - f_2)e \quad (9a)$$

$$W_{Ac} = W_E + s + (f_1 - f_2)e \quad (9b)$$

$$W_{aC} = W_E - f_2 s + (f_1 - f_2)e \quad (9c)$$

$$W_{ac} = W_E - s + (f_1 - f_2)e. \quad (9d)$$

Overall mean fitness, W_{Mean} equals $\{W_E + (f_1 - f_2)e + (p_A - q_a)s + p_C q_a f_1 s\}$. It is greater than W_{Mean} without C by the positive increment in fitness equal to $(p_C q_a f_1 s)$. This increment is, by definition, a three-way interaction $(G_C \times G_a \times E_1)$ interaction for fitness.

Thus, the frequency of the C allele increases

$$\Delta p_C = (p_C q_c)(q_a f_1 s / W_{Mean}), \quad (10)$$

and the rate of evolution at the A allele is reduced from $(2s)(p_A q_a / W_{Mean})$ to

$$\Delta p_A = (p_A q_c)(2s - q_a f_1 s)/W_{Mean}. \quad (11)$$

Note that Z_A decreases from (2s) to $(Z_A - Z_C)$, where $Z_C = (q_a f_1 s)$, so that this type of NCT relaxes selection at the A locus. Moreover, these two evolutionary effects have the same form for both cases with scale G × E for fitness: whenever C improves the performance of an otherwise deleterious allele in either environment, it is favored by natural selection *and* it relaxes selection at the locus whose fitness it alters.

With crossover G × E for fitness, it may often be the case that the increment in fitness owing to plasticity is greater than $q_a f_1 s$, when such gene regulation not only saves a fitness cost $(q_a f_1 s)$ but also free up resources to be diverted toward reproductive fitness, thereby gaining s_{aC1}, an environment-specific epistatic fitness effect. The total increment in fitness here would be $(q_a f_1 s + s_{aC1})$, increasing the strength of selection on the C allele. Adaptation to E_1 by the A locus would also be accelerated if $s_{aC1} > q_a f_1 s$.

3.2 | How the presence of niche constructing traits alters evolution at other trait loci

In this section, we revisit evolution at the A locus (see Figure 1 and Table 1) in light of evolution of an NC trait. We have already shown how NCTs and their evolution creates a positive second term, $\Delta W_{Environment}$, of the Price Equation and discussed how such evolution alters the distribution of *experienced* environments. By changing the distribution of environments, an NCT also changes Z, the additive effect on fitness of alleles at other loci, whenever those loci show genotype by environment interaction effects for fitness. We investigate such changes in this section from the perspective of the A

locus and show how the change in Z_A caused by habitat choice, niche construction or simple plasticity depends on the reaction norms of A locus genotypes (Figure 1). Because both Δp_A and $V_{Additive}$ are functions of Z_A, evolution of an NCT may also change the first term of the Price Equation, the additive genetic variance for fitness.

For each type of reaction norm in Figure 1, we calculated Z_A, the magnitude of the A allele's effect on fitness, as the average effect across environments; that is, $Z_A = (f_1)(\text{effect on fitness in } E_1) + (f_2)(\text{effect on fitness in } E_2)$. We also showed earlier that Z_A declined from $2s > 2sf_1$, $2sf_2s > 2(f_1s - f_2t)$ from panels a to c and d, to b (Figure 1). As an NCT changes f_1 and f_2, the frequencies of E_1 and E_2, it changes those average values of Z_A which are functions of f_1 and f_2, in some cases changing the order of the effects.

The case with no G × E for fitness (Figure 1a) is the simplest. Here, the effect on fitness of the A allele is independent of environment and $Z_A = (f_1)(s) + (f_2)(s) = s$, for all frequency distributions of f_1 and f_2. This is the only case where the evolution of the NC C allele has no effect on evolution at the A locus. And, for this reason, it serves as a common reference point for the relative value of Z_A before and after NCT evolution. (Here, we are treating the two terms of the Price Equation as though one or the other were held constant, ignoring the small effect of selective interference between simultaneously selected loci, where selection creates negative $LD = -(s)(e\Delta f_1)(p_Cq_c)(p_Aq_a)/W^2$. The complete dynamics of gene frequency change for this two-locus model [Δp_A, Δp_C, and ΔLD_{AC}] will be presented elsewhere [Fogarty and Wade in prep.]).

For reaction norms b, c, and d, Z_A changes as evolution of the NC C allele causes change in f_1 (i.e., $\Delta f_1 \neq 0$). For Figure 1c, Z_A increases from $(2sf_1)$ to $(2s[f_1 + \Delta f_1])$ and, in Figure 1d, Z_A decreases from $(2sf_2)$ to $(2s[f_2 - \Delta f_1])$. Figure 1b, exhibits the most interesting change owing to NCT evolution. Here, Z_A *always increases*, changing from $2(f_1s - f_2t)$ to $(2s[f_1 + \Delta f1] - 2t[f_2 - \Delta f_1])$; that is, an increase of $2(\Delta f_1)(s + t)$. Because the $V_{Additive}$ equals $(2Z_A)^2(p_Aq_a)$, an increase in Z_A *always* increases the first term of the Price Equation.

4 | KEY RESULTS

1. *NC genes spread because they alter the actual or the experienced distribution of environments in a way that increases mean fitness.* We have conceived of NC traits as those traits that change an environment of lower mean fitness into one of higher mean fitness. Adaptation at the NC locus increases mean fitness ($\Delta W_{Environment} > 0$) by substituting the experience of the favorable environment for that of the other, deleterious environment (to the degree specified by m).
2. *As NC genes spread and alter the actual or the experienced distribution of environments, they change the way that other genes adapt to the environment.* By changing the distribution of environments, NC traits can change the selective pressures that cause adaptation at other loci, as here, by changing Z_A, the *average effect on fitness* at the A locus. This change in selection depends upon the fitness relationship between a gene and its environment, which we depict as a fitness reaction norm. Whenever a non-NC locus has G × E for fitness, its contribution to the additive genetic variance for fitness is changed by adaptive evolution at the NC locus (see Table 2). A change in Z_A changes both in the rate of evolution at the A locus (Δp_A) as well as the rate of evolution of mean fitness by natural selection, $\Delta W_{Natural\ selection}$, since $V_{Additive}$ is a function of $(2Z_A)^2$. There are two circumstances under which NC *increases* $\Delta W_{Natural\ selection}$: (1) when the favored environment by NC has a larger local effect on fitness than the disfavored environment; and, (2) whenever there is crossing G × E or a fitness trade-off between environments. Our finding here lends further support to the conclusions of Laland et al. (1999), who examined the effect of NC alleles on evolution at other loci using two-locus population genetic models.
3. *As NC genes spread, they reduce the environmental components of fitness variance, $V_{Environment}$ and $V_{G\times E}$, and increase overall heritability*. As shown in Table 2,

TABLE 2 **Table of the changes in fitness variance components as a result of *niche construction* for the norms of reaction shown in Figure 1.**

	(a) No G × E	(b) Crossing G × E	(c) Scale G × E	(d) Scale G × E
$V_{Additive}$	0	+	+	–
$V_{Environment}$	–	–	–	–
$V_{G\times E}$	0	–	–	–

Note: In constructing this table, we used the expressions in Table 1, and assumed that $f_1 = f_2 = 0.5$. That is, we assumed a maximum value for $V_{Environment}$ and $V_{G\times E}$ to emphasize the effects of niche construction on the components of fitness variance. The entries in the columns are +, 0, or – indicating, respectively, that a variance component has *increased* (+), *remained the same* (0), or *decreased* (–) relative to its value (see Table 1) before niche construction. Note that, in all cases, the environmental variance *decreases* from its initial value because NCT evolution, which is driven by the variance in fitness owing to the environment, depletes this component of the total fitness variance. Note also, that when $V_{G\times E} > 0$, it too always *decreases* in response to NCT evolution. There are two circumstances where NCT evolution increases $V_{Additive}$: (1) whenever there is Crossing G × E for fitness (column B); and, (2) whenever the NCT favored environment is the most selective environment (column C).

Abbreviation: NCT, niche constructing trait.

NC traits change the distribution of environments E_1 and E_2 away from intermediate frequencies and toward "fixation" of the most favorable environment (i.e., toward $f_1 = 1$ in our model). In so doing, they reduce $V_{Environment}$ and $V_{G \times E}$ toward 0 (see also Clark et al., 2020; Fogarty & Wade, 2022). Because h_A^2, the narrow-sense heritability of fitness at the A locus in in our model, equals the ratio ($V_{Additive}$/[$V_{Additive} + V_{Environment} + V_{G \times E}$]), adaptive niche construction *always* increases the narrow-sense, heritability of fitness (i.e., including in the absence of G × E).

5 | DISCUSSION

The central principle of evolutionary biology is that the environment encountered by organisms is the source of selection pressures that shape the trajectories of allele frequency change. It is equally uncontroversial that organisms influence their own environmental circumstances, degrading it with metabolic byproducts or improving it through adaptive niche construction. Although such environmental effects have long been recognized (Brodie, 2005), it is not clear whether evolutionary theory has yet fully accounted for the potential role of organisms themselves in influencing adaptive change, since standard modeling approaches take the distribution of environments as a given feature of the world rather than one that organisms alter (Lewontin, 1983). That is, in classical theory the relative frequencies of alternative temporal or spatial environments remain constant as selection proceeds. For this reason, it is the first term of the Price Equation, $\Delta W_{Natural\ Selection}$ that is emphasized, while the second term, $\Delta W_{Environment}$, is regarded as negligible and widely ignored (Queller, 2017). Here, instead of a fixed environmental distribution, we include well-established aspects of niche construction whereby individual organisms alter the distribution of the environments they experience.

We find that adaptive NC traits evolve: selection favors the capacity to preferentially choose more favorable habitat patches, to modify habitats to be more favorable, or to adjust phenotypically to more favorably match the habitat (see Kylafis and Loreau (2008) for a similar result regarding habitat construction). Ecologists have long studied these behavioral, physiological, and developmental modes of adaptive environmental mediation (reviewed by Hastings et al., 2007; Jones et al., 1997; Odling-Smee et al., 2003; Sultan, 2015; Turner, 2000; see also Bateson & Gluckman, 2011; Donohue, 2003). All of these diverse activities and behaviors share the key feature that they increase the frequency of favorable ecological environments. Animals, plants and microorganisms may *choose* favorable habitats, preferentially moving to (or sending roots into) resource-rich habitat patches; relocating among vertical layers in aquatic systems to oxygen-rich or predator-free zones; basking, huddling for warmth, or altering leaf angles to modulate body temperature; breaking dormancy only in response to cues that growth conditions are right. They may *construct* more favorable habitats by changing external environments in physiologically suitable ways, for instance by building nests, colony structures, or burrows that reduce their exposure to environmental extremes; depositing mucilage and other exudates that alter soil chemistry and biota; or manipulating phenotypes of host organisms and symbionts. Developmental *plasticity* can mitigate resource limits, predation risk, and environmental stresses such that organisms experience a poor abiotic or biotic environment as similar to a favorable one (experiential niche construction *sensu* Chiu, 2019; Sultan, 2015; see Lewontin, 1983, 2000; Walsh, 2015); in the presence of fitness "costs" or trade-offs between environments, such plasticity may evolve as a result of epistasis governing conditional expression.

In nature, variable environments include more and less favorable habitat patches, leading to well-recognized and pervasive environmental differences in fitness (Kingsolver & Diamond, 2011; Mitchell-Olds & Shaw, 1987). Our model examines how the evolution of adaptive niche construction affects the process of natural selection in a population with two such environments. We find that adaptation at the NC locus increases mean fitness by substituting the experience of the favorable environment for the other, deleterious environment, in a manner exactly analogous to the substitution of a favorable allele (one with an average positive effect on fitness) for another allele with a deleterious effect on fitness. As a result, the evolution of NCTs introduces a second, positive term into the Price Equation, $\Delta W_{Environment}$, which reflects the change in the distribution of environments. Importantly, this term is of comparable magnitude to the first term, fitness change due to selection on allele frequencies ($\Delta W_{Natural\ selection}$), and, although it too owes its direction and magnitude to natural selection, it is a term that is missing from much of classical theory. Our findings help to refute the view that the evolutionary effects of niche construction are of smaller magnitude or less importance than those captured in the first term of the Price Equation (Scott-Phillips et al., 2014). By the same token, maladaptive NCTs, such as a habitat choice in favor of a lower-fitness environment, or habitat-constructing activities that exacerbate environmental limits or stresses, will be selected against. Niche-constructing activities of particular taxa that increase the experienced frequency of a lower-fitness environment, such as higher resource depletion rates or paradoxical habitat preferences,

may indicate functional or phylogenetic constraints, hard-wired positive correlations with fitness, or initially overlooked aspects of environmental quality.

Darwin conceived of natural selection as analogous to the artificial selection carried out by plant and animal breeders. We note that the breeding of high-yield crops to feed the world's growing population also shows a niche-constructed environmental contribution to fitness. The "Green Revolution" grain varieties that produce very high yields do so specifically in the presence of irrigation, fertilizers, and pesticides (Rockström et al., 2007). In other words, the crop environments as well as their genotypes have been "engineered" by agricultural geneticists: enhanced output depends on "technologies required to realize the genetic potential of new crop varieties and hybrids..." including the 955% increase in N fertilizers used in world agriculture since 1961 (Pellegrini & Fernández, 2018). This "genetic potential" is realized only in human-constructed high-fitness crop environments because these varieties often show fitness genotype by environment variation, producing lower yields than traditional varieties in the absence of chemical pesticides and other inputs (Colombo et al., 2022). For natural systems, biodiversity conservation requires preservation of a species' niche-constructed environments as well as its genetic potential (Boogert et al., 2006; Fogarty & Wade, 2022).

5.1 | Evolution of niche construction via epistatically regulated plasticity

In addition to a capacity for phenotypic adjustments that mitigate poor conditions (ie, where plasticity changes the experienced environment to one with higher mean fitness, similar to habitat choice and habitat construction), we modeled plasticity evolving as an epistatic regulatory switch for conditional gene expression when alternative phenotypes are favored in different environments (i.e., crossover fitness G × E). As noted above, producing a defensive structure or compound may enhance an organism's fitness in the presence of predators but, if it is costly to produce, it may reduce its fitness when predators are absent. When alternative phenotypes entail fitness trade-offs, plasticity via a conditional-expression gene (such as a hormone that turns the defense pathway on only with a predator cue, or a gene interactant that corrects the folding of an otherwise nonfunctional protein specifically in the environment where it is beneficial) will be selectively favored even when there is no mean fitness difference between two environments. This might be the case for example in an insect population with a brown vs green crypsis polyphenism for alternative habitat patches (Heinze et al., 2022). In this scenario the NCT (i.e., conditional gene expression that affords plasticity to produce a substrate-matching color phenotype) evolves by changing the fitness consequences of the environment (since the allele that is advantageous in one environment reduces fitness in the other), rather than by changing the environmental frequencies as in the other NCTs. Like simple plasticity, however, the consequence is that an otherwise disadvantageous environment (such as a substrate mismatch) is experienced as favorable. This point underlies the approach we have taken in modeling adaptive plasticity as a developmental mode of niche construction (Chiu, 2019; Sultan, 2015; see Lewontin, 1983). We note that a large and sophisticated theoretical literature on evolution of plasticity exists outside this framework (see Baythavong, 2011; Chevin et al., 2010; Scheiner, 1993; Via et al., 1995; overview and references in Pfennig, 2021), resulting in a broad consensus that evolution of plasticity is favored by within-population (temporal and spatial) environmental variation, together with reliable cues for these alternative selective conditions and the organism's ability to accurately perceive those cues and express an appropriate phenotype (e.g., Baythavong, 2011; Berrigan & Scheiner, 2004; Moran, 1992; Sultan and Spencer 2002; Tufto, 2000 and references therein). Yet key questions about evolution of plasticity remain unresolved (Chevin et al., 2010; Engen et al., 2011) because parameters such as potential costs of plasticity and cue reliability have proved notoriously difficult to determine (Scheiner & Holt, 2012; Stinchcombe et al., 2010) and the genetic architectures of plasticity remain poorly understood (Anderson et al., 2014) due to the regulatory complexity of environmental signal perception, transduction, and downstream phenotypic expression (Moczek et al., 2011; Sultan, 2015).

5.2 | Evolutionary trade-offs between types of niche-constructing trait

Because all three modes of niche construction improve the environment that an organism experiences, we speculate that evolution of one type of plasticity may obviate selection for another niche-constructing trait. For instance, individuals of a population with a form of plasticity that phenotypically buffers or compensates for a stressful environment may no longer be under selection pressure to move to a better habitat patch (habitat choice) or to physically alter its environment (habitat construction). To take a classic example, a population of hares that evolve the capacity to develop larger, more highly vascularized ears in hot locations may be able to offload excess heat without either migrating to a cooler location or building a shaded burrow. Similarly, mammals and birds as well as reptiles, insects, marine

invertebrates, and plants avoid experiencing extreme hot and cold temperatures via short-term plastic adjustments to body orientation in relation to solar angle (Kemp & Krockenberger, 2004; Muñoz et al., 2005; Norris & Kunz, 2012, further references in Sultan, 2015). In contrast, many ectotherms thermoregulate by seeking suitable microclimate patches. Garter snakes, for example, have evolved habitat choice to select thicker rocks, which remain cool, under which to pass periods of high midday insolation (Huey, 1991); in consequence they need not build such burrows or alter their phenotypes to avoid experiencing intense midday heat. Similarly, many mammals and other solitary terrestrial animals choose tree holes or other natural shelters for nesting that provide relatively constant temperature and humidity conditions (Terrien, 2011), obviating the need to physically alter themselves or their environments by constructing burrows or nests.

Which type of niche-constructing trait evolves will, like other adaptations, reflect the nature and distribution of the environmental challenge or stress, the type of organism, and its phylogenetic and genetic potential. Tadpoles that hatch into temporary ponds can neither alter the water's depth nor choose a better developmental site; the only way to mediate this risky environment is to complete metamorphosis quickly while the favorable pool habitat remains (Denver, 1998, 2013). Habitat construction may be an effective means to mediate certain environmental challenges, while others may call for habitat choice or developmental or behavioral plasticity. The same broad type of NCT will also be implemented differently in different organisms: many animals may enact habitat choice by directed movement or migration, while sessile animals and plants may rely on dispersal and dormancy traits to "choose" suitable conditions for growth and function (e.g., Donohue, 2005).

On the other hand, some NC responses entail more than one type of NCT. For instance, the plastic developmental switch to a winged morph cued in aphids by predator presence or poor host-plant quality is required to implement habitat choice (dispersal to a more favorable host-plant or habitat patch) (Braendle et al., 2006 and references therein). In such cases, different NCTs act jointly rather than trading off. In other cases, the distinction between different NCTs may not be straightforward. Plants for example implement habitat choice for nutrient-rich microsite patches by means of differential proliferation of the individual's root system, a well-known form of developmental plasticity often viewed as "foraging behavior" (McNickle et al., 2009). These instances, like the trade-offs noted above, make clear the shared feature of diverse NCTs as evolved capacities for adaptive changes to the organism's experienced environment.

5.3 | How evolution of niche construction affects loci of non-niche-constructing traits

Because niche constructing traits change the distribution of environments, their evolution impacts other genetic loci whose fitness effects vary from one environment to another. For such loci, the average effect of alternative alleles on fitness is altered by the change in environmental frequencies. Moreover, the average genic fitness effects will *increase* due to NC (a) whenever there is crossover G × E fitness interaction as well as (b) in cases where more genic variance is expressed in the environment favored by NC. The latter is common in those cases where genotypic fitnesses are more similar in a stressful, disfavored environment (Charmantier & Garant, 2005; Matesanz et al., 2014). For all such traits, the evolution of niche construction changes the *first* term of the Price Equation for adaptive evolution—change in a population's mean fitness due to additive genetic variance—accelerating the process of trait adaption to the niche-constructed environmental distribution.

At the same time, as genes for niche constructing traits spread in a population, they increasingly change the distribution of alternative environments away from intermediate frequencies and toward "fixation" of the more favorable environment (i.e., toward a frequency of 1.0 in our model; Table 2). This shift in relative frequency reduces both the environmental variance ($V_{Environment}$) and the variance due to genotype by environment interaction ($V_{G\times E}$) toward 0. Since narrow-sense heritability (h_A^2) is determined by the ratio ($V_{Additive}/[V_{Additive} + V_{Environment} + V_{G\times E}]$), adaptive niche construction *always* increases the narrow-sense heritability of fitness by diminishing its denominator. Most discussions of G × E variation focus on its role in the maintenance of polymorphism (Gillespie and Turelli 1989; Hedrick, 1986, 2006; Hunter, 2005; Mitchell-Olds et al., 2007), in slowing of the rate of evolution toward an optimum joint phenotype (Via & Lande, 1987), or in providing the genetic substrate for the evolution of phenotypic plasticity (Chevin et al., 2013; Scheiner, 1993; Via & Lande, 1985; further references in Sultan, 2021). Our model reveals that niche construction creates a new role for G × E by converting it into additive genetic variation and thereby increasing heritability. However, these effects of of niche construction will pertain only to loci which show G × E fitness interaction with respect to the subset of environmental states that is altered by the organism's specific niche constructing activities (see Clark et al., 2020).

At the same time niche construction may relax selection on traits that are functionally related to the aspects of environmental experience mitigated by the NC activity. For instance, selective pressure to evolve

physiological heat tolerance may be slowed in a population of niche constructing individuals who choose cooler habitat patches, build burrows to avoid midday heat, or plastically modify the morphology or orientation of their thermal-regulatory structures (references in Sultan, 2015), because the population less frequently experiences an extreme-heat environment. Furthermore, if traits related to thermal tolerance show crossover $G \times E$ for fitness (for instance, if they bear a fitness cost in a cool environment), the fitness trade-off and hence the selection pressure will be reduced for adaptation to the lower-fitness heat-stress environment.

5.4 | Limitations of our approach

Selection at two loci simultaneously creates negative linkage disequilibrium, known as selective interference, which slows the rate of evolution of both genes. In our model, this effect is small, suggesting that such interference will not substantially influence the interplay between NCT loci and other trait loci. However, there is another source of positive LD if we permit positive epistatic interactions for fitness between the NC C Locus and the A locus (i.e., $s_{AC} > 0$). Such an effect would lead to positive LD between the two loci, reciprocally accelerating each other's evolution. Including such fitness effects into our model is left to future work as is a full investigation of its two-locus dynamics (Fogarty and Wade, in prep.).

It is important to note that we modeled a highly simplified set of genetic and environmental factors affecting fitness: alternative alleles at two haploid loci (A and a; C and c) in two alternative environments (E_1 and E_2). There are many interesting and salient dimensions of niche construction that this simple model does not address, including environmental inheritance (Odling-Smee et al., 2003; with Price Equation, Uller & Helanterä, 2017) and other transgenerational effects (e.g., González-Forero, 2023) as well as effects on evolution of symbionts and other co-occurring species (e.g., Clark et al., 2020). Further studies of different, and more complicated, scenarios are needed to test the robustness of our findings.

6 | CONCLUSIONS

The Price Equation lets us examine two ways to alter mean fitness, change in additive genetic variance V_A (due to selection on alternative alleles) and change in the environment. In standard quantitative genetics models, the environmental distribution is assumed to be fixed ($E' = E$); the second, environmental change term is consequently set at zero. Indeed, without niche constructing traits—that is, as long as the distribution of environments remains unchanged as selection proceeds—there will be no second term, even with $G \times E$ for fitness. Our model focuses on the crucial fact that adaptive niche constructing activities change environmental frequencies. It shows how, through these diverse activities, organisms increase population mean fitness so as to provide a positive (and potentially substantial) second term in the Price Equation while at the same time increasing the V_A that fuels adaptation. Both effects accelerate adaptive evolution; neither effect occurs without these organism-mediated environmental changes. These findings are consistent with the concept of niche construction as framed by Richard Lewontin(1983, 2000), who emphasized the roles played by organisms in functionally defining as well as shaping their own selective environments.

ACKNOWLEDGMENTS

Funding support for this collaboration was provided by grant 61369 from the John Templeton Foundation to S. E. S., a Visiting Fellowship awarded to S. E. S. by the Konrad Lorenz Institute for Evolution and Cognition (KLI), and by NIFA/USDA award 2019-33522-30064 to M. J. W. The opinions, interpretations, conclusions and recommendations are those of the authors and are not necessarily endorsed by the John Templeton Foundation. We are grateful to two anonymous reviewers for valuable comments and suggestions.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

Data sharing not applicable—no new data generated.

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ENDNOTE

[1] There is a considerable body of theoretical work examining the conditions for the maintenance of polymorphism within populations given a specified environmental distribution of genotypic fitnesses and a pattern of migration among localities (e.g., Levene 1953; reviewed in Felsenstein, 1976).

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How to cite this article: Wade, M. J., & Sultan, S. E. (2023). Niche construction and the environmental term of the price equation: How natural selection changes when organisms alter their environments. *Evolution & Development*, 1–19. https://doi.org/10.1111/ede.12452