Some Evolutionary Consequences of Niche Construction with Genotype-Environment Interaction

Kathleen Donohue

Abstract "Niche construction" is the ability of organisms to determine the environment they inhabit. Niche construction can occur through direct modification of the environment, habitat choice, or passive alterations to the environment, such as resource use or depletion. When organisms predictably alter the environment they inhabit, and when niche construction has a genetic basis, it can influence evolutionary responses to selection. Each component of evolutionary responses to selection—the strength of selection on a trait, the phenotype and phenotypic variance of a trait, and the genetic variance of a trait—can change with the environment. Examples of niche construction in plants are discussed, and a simple univariate model demonstrates that when niche construction alters phenotypic expression (via phenotypic plasticity) and the expression of genetic variation or heritability (via genotype-environment interaction), it can cause slower or faster responses to selection, less or more sustained responses to selection, or even negative responses to selection. In particular, genotype-environment interaction can counteract or augment phenotypic plasticity to the constructed environment in its effects on evolutionary responses to selection. Thus, genotype-environment interaction that results in environment-dependent genetic parameters influences evolutionary trajectories with niche construction.

Keywords Genotype-environment interaction · habitat selection · indirect genetic effects · response to selection · phenotypic plasticity

1 Introduction

One of the primary goals of empirical quantitative-genetics, especially as applied to breeding programs, is to distinguish the contributions of genetic versus environmental factors to trait variation. However, when organisms can alter the environment they are exposed to, and when this ability has a genetic basis, the environment too

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can have a genetic component, can evolve, and can influence the dynamics of trait evolution (Wolf 2003; Cheverud 2003). The ability of organisms to determine the environment they experience has been termed "niche construction" (Odling-Smee et al. 1996), which, broadly interpreted, includes what have come to be known as "indirect genetic effects" (Moore et al. 1997; Wolf et al. 1998), and more traditionally "habitat selection" (Levins 1968; Holt 1987; Rosenzweig 1987). Niche construction can occur by direct habitat modification of the environment, habitat choice, dispersal habits, or through passive processes such as resource use or depletion (Odling-Smee et al. 1996; Laland et al. 1999; Day et al. 2003; Odling-Smee et al. 2003). Animals can practice very sophisticated modes of niche construction, through the building of shelter, cultivating or hoarding of food supplies, complex behaviors of dispersal, or the creation of social (or anti-social) environments. Other organisms, such as plants, also practice niche construction through modifications of growth structure (e.g. clonal foraging and shade avoidance), environmentally regulated phenological switches such as germination or reproductive timing, or dispersal strategies (reviewed in Bazzaz 1991; Donohue 2003,2005). Many maternal effects (Falconer 1965; Kirkpatrick and Lande 1989) can also be considered to be niche construction, since the maternal parent creates the environment in which progeny develop (Wade 1998). Broadly speaking, all organisms in some sense determine the environment they inhabit and thereby are subject to the dynamics of niche construction.

Niche construction can influence evolutionary dynamics (Fig. 1). Responses to selection are determined by the strength of selection on a trait, and the proportion of the phenotypic variance of the trait that is genetically determined, or the heritability of the trait. The environment that organisms experience can influence all of these components of evolutionary responses to selection. In this manner, niche construction can influence evolutionary outcomes and trajectories.

First, the environment can influence the agents of natural selection to which an organism is exposed (Laland et al. 1999; Day et al. 2003; Donohue 2003; Schwilk 2003). Through niche construction practices that alter exposure to existing selective influences, or that create new agents of selection, organisms can alter the strength, direction, and mode of natural selection on a trait. Most investigations of the evolutionary consequences of niche construction have focused on this very important pathway, and these investigations comprise the literature on habitat selection or habitat choice. Perhaps the most interesting conclusions of these classic studies is that habitat selection can alter the dynamics of specialization. Specifically, when habitat selection increases exposure to one environment and decreases exposure to other environments, adaptation to the first environment can be faster, and adaptation can be closer to the optimum for that environment (Levins 1968; Holt 1987; Rosenzweig 1987; Brown 1990; Whitlock 1996); that is, habitat selection facilitates specialization (Via and Lande 1985; Schlichting 1986; Van Tienderen 1991; Scheiner 1993). In animal breeding programs, this aspect of niche construction can be important if traits that evolve in response to artificial selection result in new agents of natural selection, such as aggressive environment, intensity of competition, or probability of infection, for example.

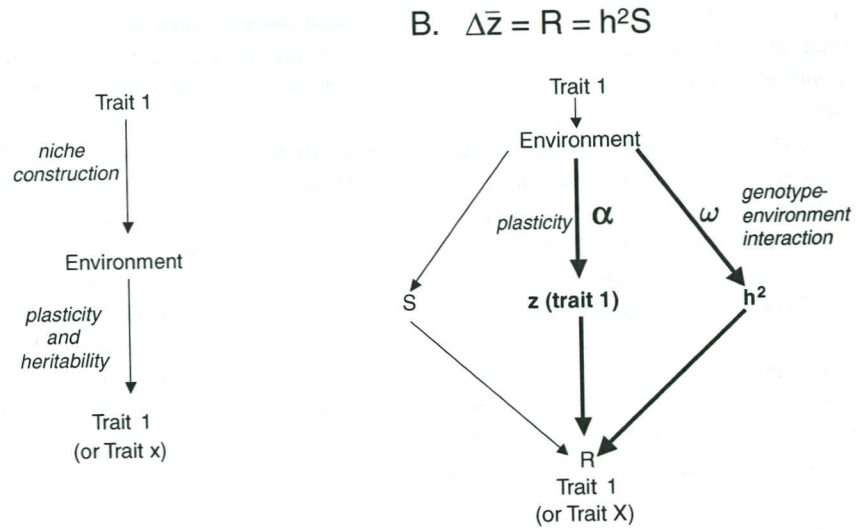

Fig. 1 (A) Niche construction. One trait influences the environment experienced by the organism, which in turn influences the evolution of that same trait or a different trait ("Trait X"). (B) The breeder's equation, showing pathways whereby effects of niche construction on phenotypic expression, α, and the heritability of the trait, ω, can influence total response to selection of the trait. z = the phenotypic value of the trait; h^2 is the heritability of the trait; S is the strength of selection on the trait; α is the factor by which the environment that is determined by the trait influences the phenotypic value of the trait, via phenotypic plasticity; ω is the factor by which the environment that is determined by the trait influences the heritability of the trait. The trait is directly associated with the environment that the organism experiences and can therefore be considered to be a metric of the environment itself. In this case, the trait influences the environment, which influences its own expression and heritability or the expression and heritabilty of a different trait ("Trait X")

Second, the environment can influence phenotypic expression via phenotypic plasticity (Schlichting and Pigliucci 1998). Phenotypic plasticity is the ability of a given genotype to alter its phenotype in response to the environment that it experiences. Phenotypic plasticity thereby can alter adaptive dynamics because it can determine how close to the optimum the phenotype of that genotype is, and plasticity can act in the same or opposite direction as that favored by selection. For example, selection for less aggressive behaviour might have the unintended consequence of depriving an animal of food, which, through plasticity in response to reduced nutrition, would result in decreased body mass. In addition, the degree of phenotypic variance via plasticity to microenvironmental factors can influence the degree of environmental variance of traits, which is an important component of trait heritabilities.

Third, the environment can influence the expression of genetic variation (Via and Lande 1987). Just as a given genotype can alter its phenotype according to the environment, when different genotypes respond to the environment differently, the differences among them can be environment-dependent. In other words, with genotype-environment interaction, the magnitude of genetic variance can depend on the environment. The effect of the environment on phenotypic and genetic variances

can cause changes in the heritability of traits and thereby alter the evolutionary potential of the trait. In the previous example, not only may body mass decline as a result of food deprivation, but heritability of body mass may also decline, making improvement of that trait more difficult.

This last path of influence—through genotype-environment interaction—has not received attention in the literature on niche construction or indirect genetic effects. Environment-dependent heritabilities and genetic variances are so widely documented that they are fundamental components of any study that attempts to predict responses to selection (Via and Lande 1987; Mazer and Wolfe 1992; Dorn et al. 2000; Schlichting and Pigliucci 1998). Usually, the environment is considered to be a fixed condition of the breeding program or ecology. In the literature on specialization and phenotypic plasticity, the environment varies between two or more states, and the organism experiences each of these states with a given frequency. When attributes of the organism itself determine the environment experienced by the organism, however, and when these attributes themselves can evolve, then the environment that the organism experiences can also evolve over time (Donohue 2003; Wolf 2003; Wolf et al. 2004). The evolution of this environment, in turn, is expected to result in predictable changes in the genetic architecture of traits—that is, changes in the genetic variances and covariances of traits—due to genotype-environment interaction.

In the simplest case, one trait can influence the environment that organisms experience, and this environment can influence the phenotypic expression and heritability of this same trait (Fig. 1). In more complex cases of interacting traits, one trait can influence the environment that organisms experience, and that environment can influence the phenotypic expression and heritability of other traits (reviewed in Donohue 2003, 2005). In multivariate cases, phenotypic and genetic covariances are also likely to be environment-dependent, contributing even more complexity to the dynamics.

In both univariate and multivariate cases, either positive or negative feedbacks can occur and are expected to influence evolutionary outcomes and trajectories. Here I discuss empirical examples of niche construction in plants and present a heuristic model of the simplest case of these dynamics: the univariate case in which one trait determines the environment that then influences its own phenotype and heritability. Even in this simplest case, niche construction via heritable (and evolvable) traits can cause faster, slower, or even reversed responses to selection, and it can constrain or sustain evolutionary responses over time. In particular, genotype-environment interaction can counteract negative or positive effects of phenotypic plasticity to the newly evolved environment, and it must therefore be considered when attempting to predict evolutionary outcomes with niche construction.

2 An Empirical Context of Niche Construction

In a volume on animal breeding, I will discuss examples of niche construction in plants, but suggest that these dynamics are frequently even more readily discernible in animals. Indeed, many historical and recent theoretical investigations

of niche construction have been within the context of social evolution and habitat selection in animals (Moore et al. 1997; Wolf et al. 1998; Wolf 2003). Examples of behaviors that can be interpreted as "niche construction" are so abundant (Odling-Smee et al. 1996) that some have complained that almost anything at all could be interpreted as "niche construction", thereby undermining the utility of the concept. While it is perhaps true that one can be fully occupied by simply characterizing diverse phenomena in terms of the omnipresent "niche construction," I would like to counter that just because something is ubiquitous does not imply that it is unimportant. Yes, social aggression; habitat contamination by waste, toxins, or disease; construction of nests, lodges, or webs; thermoregulation; these can all be interpreted as niche construction, and I leave it to the reader to decide whether consideration of the consequences of niche construction is useful for their purposes. My purpose is simply to point out that traits of organisms frequently alter the environments they experience in ways that influence the expression of those or other traits, and in ways that influence the expression of genetic variation of those traits.

3 Phenotypic Plasticity to the Constructed Environment: Interactions Between Flowering and Germination Time

The following example of niche construction through phenological plasticity demonstrates the significance of niche construction and plasticity to the "constructed" environment to plant life histories. Variation in the phenology of germination and flowering determines overall life-history expression in many annual plants, including *Arabidopsis thaliana*. First, the germination season determines the seasonal conditions experienced by seedlings and rosettes (Weinig 2000; Donohue 2002). It is well known in *A. thaliana* and many other species that the seasonal cues of photoperiod and temperature, and especially cold vernalization of rosettes, interact to determine flowering time (Koornneef et al. 1991; Nordborg and Bergelson 1999; Mouradov et al. 2002; Simpson and Dean 2002). In temperate climates, long days and cold vernalization usually accelerate flowering. Germination phenology determines which of these seasonal factors a rosette will be exposed to.

Likewise, the flowering season determines the seasonal conditions experienced by seeds during maturation and soon after dispersal. Seed maturation conditions strongly influence seed dormancy, and post-dispersal seasonal environments determine exposure to various dormancy-breaking factors, such as cold (reviewed in Baskin and Baskin 1998). Therefore, flowering phenology strongly determines germination phenology.

In experimental studies of *A. thaliana*, we found that the season of seed dispersal strongly influenced natural selection on germination time, phenotypic expression of germination time, and the expression of genetic variation for germination time (Donohue et al. 2005a, b, c). Seeds dispersed after a summer flowering season experienced strong stabilizing selection favoring intermediate germination timing in mid October, and seeds dispersed after an autumn flowering season experienced

weak directional selection favoring early germination. More dramatically, in the later cohort, non-dormant seeds had the highest fitness, but in the earlier cohort, non-dormant seeds had zero fitness. Phenotypic expression of germination timing also varied: seeds dispersed after a summer flowering season germinated slowly throughout the summer and autumn, while seeds dispersed after an autumn flowering season germinated very quickly that same autumn. The evolutionary potential of germination timing also strongly depended on the season of seed dispersal, with the heritability of germination timing being much higher for seeds dispersed after a summer flowering season than for seeds dispersed after an autumn flowering season.

Germination timing, in turn, influenced reproductive phenology by determining the seasonal environment experienced by young rosettes. In particular, seeds that germinated in the spring reproduced much earlier in development and at a smaller size than seeds that germinated in the autumn.

This mutual interaction between the two niche-constructing traits of flowering time and germination time leads to some interesting consequences for life-history expression. First, variation in germination timing accounts for the difference between the winter annual and spring annual life history, with winter annuals germinating in the autumn and spring annuals germinating in the spring. In addition, spring germinants responded plastically to the season of germination by accelerating their reproduction. Therefore, niche construction through germination timing, and plasticity to the constructed environment, can account for variation between spring and winter annual life histories.

Moreover, when seasonal variation in reproductive and dispersal phenology is present, a bivoltine life-history is possible. In particular, autumn flowering conditions in turn accelerated germination and enabled germination by spring, and spring germination conditions enabled spring flowering. These interactions can actually enable two generations to be completed within a single year instead of the typical one generation. This novel bivoltine life history is the outcome of one niche constructing character influencing the seasonal environment experienced by a second niche-constructing character, and vice versa. Importantly, phenotypic plasticity to the "constructed" seasonal environment plays a key role and in this example altered overall life-history expression in a manner that can influence generation time.

4 Plasticity and Environment-Dependent Heritability: Seed Dispersal as a Simple Case of One Niche-Constructing Character Influencing Itself

This example explores the consequences of niche construction on the expression of genetic variation. Seed dispersal provides an example of a simple case of one niche-constructing character influencing itself. Seeds are the most mobile life stage in most plants, excepting pollen, and their dispersal offers the opportunity to escape from adverse conditions such as pathogens, predators, or sibling competition at the maternal home site (reviewed in Howe and Smallwood 1982; Willson and Traveset 2000). Perhaps the most predictable environmental consequence of seed dispersal is reduced conspecific density experienced by efficiently dispersed seeds in open habitats, and highly competitive conditions experienced by poorly dispersed seeds (e.g. Janzen 1978; Baker and Dowd 1982; Augspurger 1983; Rees and Brown 1991; Augspurger and Kitajima 1992; Donohue 1999).

In many species, efficient seed dispersal leads to lower sibling densities after dispersal. It is therefore important to know how density in turn influences dispersal ability and the expression of genetic variation for dispersal ability. To investigate this, we conducted a quantitative genetic study using morphologically diverse inbred ecotypes of *Arabidopsis thaliana* (Wender et al. 2005; Donohue et al. 2005d). We grew plants at different densities, measured seed dispersion patterns under controlled conditions, and estimated the heritability of post-dispersal density. We found that, plants grown under high density dispersed seeds to lower sibling density than plants grown at low density. Significant genetic variation for post-dispersal density was detected, but only when the maternal plants were grown at high density, and this was due to altered environmental and genetic variances.

This simple result has some interesting potential evolutionary consequences. Consider a population of plants growing at high density, but in which natural selection favors lower post-dispersal density, as it does in many species (e.g. Burdon and Chilvers 1975; Augspurger and Kitajima 1992; Donohue 1999). Our results predict that an evolutionary response to such selection is possible, since genetic variation for dispersal ability is expressed at high density. However, once low post-dispersal density is achieved, genetic variation for dispersal will cease to be expressed. Such a negative feedback pathway would be a constraint on the further evolution of dispersal. Note that this genetic constraint is not caused by the elimination by natural selection of inappropriate genotypes, which would reduce genetic variation. Rather, this genetic constraint is caused by plasticity alone, causing less genetic variation to be expressed, even when the genotypes are identical. The evolution of the post-dispersal density environment and the density-dependent genetic expression for dispersal are adequate to impose this constraint.

In this particular example, the dynamics operated so as to reduce the expression of genetic variation, but for other niche-constructing traits, they may increase the expression of genetic variation. In such cases, the evolution of the niche-constructing character could facilitate its further evolutionary responses to selection by enabling genetic variance to be expressed in the newly evolved environment. Thus while selection may reduce genetic variation for a trait over time, the dynamics accompanying niche construction may act to provide additional genetic variation to enable more sustained responses to selection. Therefore the ability of organisms to determine the environment they experience, and the environment-dependent genetic variation for that ability, can cause unexpected evolutionary dynamics that can either constrain or facilitate the evolution of such characters. More generally, because environment-dependent genetic expression is so commonly observed (e.g. Mazer and Wolfe 1992; Donohue et al. 2000; Munir et al. 2001; Dorn et al. 2000), niche construction has the potential to alter the evolutionary potential of many traits whose genetic variation may depend on the "constructed" environment.

5 Consequences of Environment-Dependent Heritability to Evolutionary Dynamics with Niche Construction

A heuristic model is presented that demonstrates that niche construction, combined with genotype-environment interaction that alters trait heritability, can influence evolutionary trajectories. The model follows the example of seed dispersal discussed above, and examines the simplest case of one niche-constructing trait influencing itself. It focuses exclusively on the interactions between phenotypic plasticity and environment-dependent trait heritability. Selection on the trait is assumed to be constant, as is likely to be the case in breeding programs. I assume purely directional selection in which a fixed proportion of the population sample is retained for breeding purposes, and in which the intensity of selection is measured in terms of a standardized trait (that is, the change in the mean population before and after selection is measured in standard deviation units). This assumption simplifies the model, allowing the focus to remain on the interaction between phenotypic plasticity and genotype-environment interaction. Indeed, this assumption greatly oversimplifies the dynamics if the conclusions were to be applied to natural populations, in which natural selection itself can be expected to vary with changes in trait means and variances, as discussed below.

The breeder's equation of quantitative genetics describes the evolutionary response to selection, R, as the change in phenotype from one generation to the next, as a function of the strength of selection on the trait, S, and the heritability of the trait h^2 [which is the proportion of phenotypic variance of the trait that is (additive) genetically based variance, V_A/V_P].

$$R = h^2 S \tag{1}$$

In this simplest, univariate case, let the trait of interest, z, be the niche-constructing trait, which directly predicts the environment experienced by the organism. As such, the trait, z, can itself be a measurement of the environment that the organism experiences. For example, if a heritable attribute of the organism determines the conspecific density, the quality of light, or the temperature that the organism experiences, then the environment experienced by the organism can be said to have a heritable component, and the trait of interest can be a measurement of the density, light quality, or temperature that the organism experiences. All other components of that environmental factor are assumed to be random (unpredictable), and therefore do not contribute to the directional change in the environmental factor experienced by the organism.

From this point forward, R refers to the response to selection that would be predicted without any change to the environment due to niche construction. In contrast, $\Delta\bar{z}$ refers to the change in mean phenotype from one generation to the next, $z' - z$, when niche construction also occurs.

The environment determined by the trait can influence the heritability of that trait. The new environment created by niche construction can alter the environmental

variance (V_E, where $V_A + V_E = V_P$) or the genetic variance (V_A). Changes in V_A occur because different genotypes can respond differently to the altered environment, leading to genotype-environment interactions and environment-dependent genetic variation. With changes in either V_P or V_A, the heritability itself can change. Let ω measure the effect of the new phenotype on the heritability of that phenotype, such that the heritability is altered by a factor proportional to the mean phenotype, z. When niche construction alters heritability, the response to selection is:

$$\Delta\bar{z} = (h^2 + \omega z h^2)S = R + \omega z R \tag{2}$$

The response to selection, $\Delta\bar{z}$, will be faster than R when $\omega > 0$, and it will be slower when $\omega < 0$, provided $z > 0$. The response will equal R when $\omega = 0$. There will be no evolutionary response when $\omega = -1/z$. The phenotype after selection and inheritance is:

$$z_1' = R + z(1 + \omega R) \tag{3}$$

It should be noted that the term $(h^2 + \omega z h^2)$ in equation (2) is not strictly the new heritability of the trait, but rather the predicted regression between parents and offspring. The anomaly of the revised "heritability" being able to exceed unity or even become negative can be understood in terms of genetic correlations across parent and offspring environments (Via and Lande 1985; Fry 1992; Windig 1997). Consider the extreme case when a trait is already perfectly heritable ($h^2 = 1$). If genetic variances are higher in the offspring environment than in the parental environment, the regression between parents and offspring can actually exceed one (Fig. 2), and if the genetic variances are lower in the offspring environment then the regression would become less than one. In an extreme case, if the genetic correlations across parent and offspring environments are negative, then the regression between parents and offspring can actually be negative. While this may seem unlikely, we actually have no empirical data on these dynamics. For the purposes of this model, I assume the simplest function of altered parent-offspring regressions: a linear change of heritability with a unit change in the environment that is given by the mean phenotype z. While other functions may be possible, we as yet have no empirical basis for preferring a more complex function. I will also restrict consideration to the more plausible parameter values of positive parent-offspring regressions within the range of 0 and 1.

When niche construction alters the environment that the organism experiences, the phenotype expressed by the organism may also change because of phenotypic plasticity. Let α measure the proportional change in the mean phenotype of the organism caused by phenotypic plasticity in response to the environment created by niche construction. Again, we have very little empirical data on how phenotypes change with unit changes in an evolving environment, so I assume the simplest function: a linear change of phenotype with a unit change of the environment that is given by the phenotype mean z. This is also in accordance with models of maternal effects (with "m" describing a similar proportional change in offspring

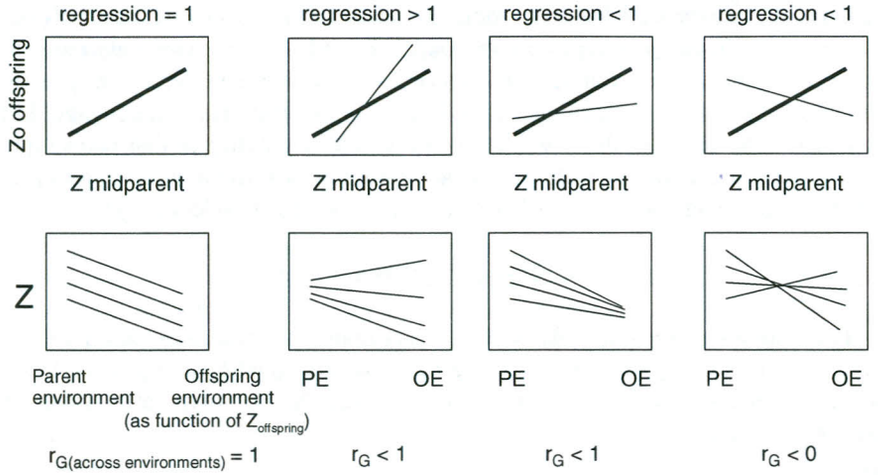

Fig. 2 Environment-dependent genetic expression. *Upper panels* show parent-offspring regressions. *Lower panels* show the reaction norms of a set of four genotypes, with the lines connecting the mean phenotype of each genotype as it is expressed in the parent (*left*: PE) and offspring (*right*: OE) environments. r_g is the genetic correlation across parent and offspring environments. The left-most panel shows an extreme case of a perfect heritability, with the parent-offspring regression equal to one. The second panel shows a case when genotype-environment interaction causes increased genetic variance in the offspring environment. The third panel shows a case in which genotype-environment interaction causes decreased genetic variance in the offspring environment. The fourth panel shows the case when genotype-environment interaction does not change genetic variance in the offspring environment, but the genetic correlation across parent and offspring environment is negative

phenotype as a function of the maternal phenotype, or more specifically the non-genetic phenotypic correlation between mothers and offspring; Falconer 1965; Kirkpatrick and Lande 1989) and indirect genetic effects (with Ψ describing the proportional plasticity of the focal trait in response to the evolving social environment; Moore et al. 1997).

Considering phenotypic plasticity to the newly evolved environment, first, selection causes an evolutionary change in the phenotype to $[R + z_1(1 + \omega R)]$, as described above. Second, because the phenotype, z_1', is also a measure of the environment, an additional change in the phenotype can result because of plasticity to the new environment. This additional change is measured as $\alpha z_1'$, which is $\alpha[R + z(1 + \omega R)]$. Thus the phenotype after selection, inheritance, and plasticity is revised to be:

$$\begin{aligned} z' &= R + z(1 + \omega R) + \alpha[R + z(1 + \omega R)] \\ &= (R + z) + \omega z R + \alpha(R + z + \omega z R) \end{aligned} \quad (4)$$

The first term, $(R+z)$, is the phenotype expected with no effect of the newly constructed environment. The second term, $\omega z R$ is the deviation from that expectation

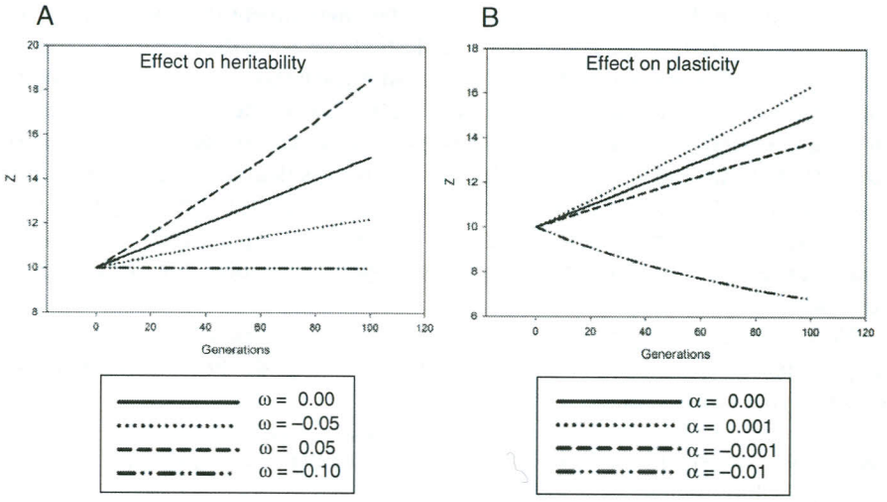

Fig. 3 Response to selection when the constructed environment increases or decreases only the trait heritability (**A**) or only the plasticity of a trait (**B**). The *y*-axis is the mean phenotype, z, of a trait that is associated with niche construction, and the *x*-axis is the number of generations of selection. Selection favors an increase in the phenotypic value, z. $h^2 = 0.5$, $s = 0.1$ in both panels. In A, $\alpha = 0$. In B, $\omega = 0$

caused by a change in the heritability of the trait. The third term, $\alpha(R + z + \omega z R)$, is the deviation caused by plasticity of the trait to the newly constructed environment. The total evolutionary response to selection is:

$$\Delta\bar{z} = z' - z = R(1 + \alpha) + \omega z R(1 + \alpha) + \alpha z \quad (5)$$

Environmental effects on heritability alone: First consider only the effects of the newly constructed environment on the heritability of the trait (Fig. 3a). When the newly constructed environment decreases the expression of genetic variation for the trait, evolutionary responses are slower than when the environment exerts no effect on heritability. In the extreme case of $\omega = -1/z$, no response to selection occurs. Conversely, when the newly constructed environment increases the expression of genetic variation, evolutionary responses to selection are faster. Over long periods of time, the former case causes faster depletion of genetic variation, and eventually inhibits responses to selection altogether (note the non-linear, diminishing increase in z over time). The latter case, in contrast, can cause more sustained responses to selection. Thus niche construction, and the accompanying effects of the environment on trait heritabilities, can either impose constraints on long-term responses to selection, or they can enable more sustained responses to selection.

Effects of phenotypic plasticity with environment-dependent heritability: The newly constructed environment can influence not only the heritability of the trait, but it can also influence the phenotypic expression of that trait through phenotypic plasticity (Fig. 3b). Consider the case when selection favors an increase in a trait.

If an increase in the trait causes a change in the environment that causes a further increase in that trait because of phenotypic plasticity ($\alpha > 0$), then the total response to selection will be greater than the case without niche construction and plasticity to the constructed environment. Conversely, if an increase in the trait causes a change in the environment that causes a decrease in the trait due to phenotypic plasticity ($\alpha < 0$), then the total response to selection will be less than the case without niche construction and plasticity to the constructed environment. If the opposing plasticity is very strong compared to the strength of selection (see equation (11) below) the total change in the phenotype can be in the opposite direction to that favored by selection.

Now consider the effects of the newly constructed environment on both the phenotypic expression and the heritability of the trait (Fig. 4). First, no response to selection will occur when:

$$\alpha = -R(1+\omega z)/[R(1+\omega z)+z] \text{ or when } \omega = -[R(1+\alpha)+\alpha z]/zR(1+\alpha) \quad (6)$$

More significantly, no response to selection will occur when:

$$z = -Sh^2(1+\alpha)/[\omega Sh^2(1+\alpha)+\alpha] \quad (7)$$

which, when $\alpha = 0$, reduces to $z = -1/\omega$, as mentioned above. What this implies is that the phenotype, z, can reach an equilibrium, z^*, with no further change, provided that the phenotype evolves towards z^*, given in equation (7). If it evolves in the opposite direction, then the equilibrium is unstable. The direction and magnitude of evolution depends on the distance from this equilibrium value. In the simple case of $\alpha = 0$ and $z^* = -1/\omega$, it follows that whether the phenotype evolves towards or away from the equilibrium z^* depends on the signs of S and ω, since, from equation (2), $\Delta\bar{z} = h^2 S\omega(z - z^*)$. If $z > z^*$, then z evolves towards the equilibrium only when $S\omega$ is negative, and if $z < z^*$, then z evolves towards the equilibrium only when $S\omega$ is positive. Thus whether evolution proceeds in the direction of the equilibrium also depends on the starting value of z.

Next, if the newly constructed environment simultaneously increases heritability and causes a change in phenotypic expression in the same direction that selection favors (Fig. 4, dotted line), then the total response to selection is faster and more sustained. In contrast, if the newly constructed environment decreases heritability and causes a change in phenotypic expression opposite to the direction favoured by selection (Fig. 4, short-dashed line), then the total response to selection is slower.

It is also possible that the newly constructed environment influences heritability and phenotypic expression in opposite directions. For example, it may increase heritability but cause a change in the phenotype in the direction opposite to that favored by selection (Fig. 4, dash-double dot line); or it may decrease heritability but cause a change in the phenotype in the same direction favored by selection (Fig. 4, long-dashed line). As before, for a given magnitude of α ($\alpha = -0.001$ in Fig. 4), a slower response results when $\omega < 0$, and a faster response results when $\omega > 0$. Likewise for a given value of ω, the response is faster when $\alpha > 0$, and the response is slower

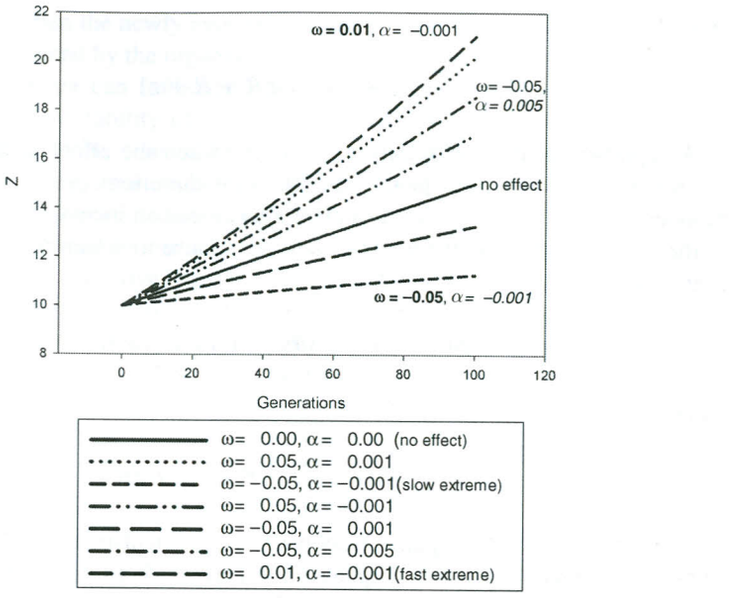

Fig. 4 Response to selection when the constructed environment influences both the heritability and the phenotypic expression of the trait. The y-axis is the mean phenotype, z, of a trait that is associated with niche construction, and the x-axis is the number of generations of selection. Selection favors an increase in the phenotypic value, z. $h^2 = 0.5$, $s = 0.1$ "no effect" refers to the case in which the newly constructed environment created by the trait has no effect on the phenotypic expression or the heritability of the trait (when $\alpha = 0$ and $\omega = 0$). Direct comparisons of the effects of different magnitudes of ω are shown in bold. Direct comparisons of effects of different magnitudes of α are shown in italics.

when $\alpha < 0$. Moreover, the effect of a change in magnitude of ω (from -0.05 to $+0.05$) depends on the value of α. For example in Fig. 4, the phenotype after 100 generations changed by 6.9 units when $\alpha = 0.001$, but with the same change in ω, the phenotype changed by 5.7 units when $\alpha = -0.001$. Likewise, when α changed from -0.001 to $+0.001$, the change in phenotype after 100 generations was 3.2 when $\omega = 0.05$, but it was 2.0 when $\omega = -0.05$. Thus, one value of ω enhanced the effect of changed α, while another value masked that effect, and vice versa. Whether niche construction causes a faster or slower response to selection therefore depends on the relative magnitudes of α and ω. When the two effects oppose one another, the effect on heritability can exactly balance the effect of phenotypic plasticity when, from equation (6):

$$R(1+\alpha)+\omega z R(1+\alpha)+\alpha z = R \quad (8)$$

which occurs when:

$$\omega = [-\alpha(R+z)]/[zR(1+\alpha)] \quad (9)$$

or when:

$$\alpha = -\omega z R/(\omega z R + R + z) \tag{10}$$

A negative response to selection can occur when the effect of the constructed environment causes a large plastic change in the direction opposite to that favored by selection, even when the environment also causes an increase in the heritability of the trait (Fig. 5). Specifically, a negative response to selection can result when, from equation (5):

$$R(1+\alpha) + \omega z R(1+\alpha) + \alpha z < 0 \tag{11}$$

which occurs when:

$$\alpha < -R(1+\omega z)/(R + z + \omega z R) \tag{12}$$

The magnitude of the negative response to selection also depends on whether the environment alters heritability (Fig. 5a). If it increases heritability, then the negative response is slower, whereas if it decreases heritability, the negative response is faster than when the environment exerts no influence on heritability.

The magnitude of the negative response also depends on the strength of selection on the trait (Fig. 5b). Specifically, selection on the trait will override plasticity in the opposite direction when:

$$S > -\alpha z/\left\{h^2[(1+\alpha) + \omega z(1+\alpha)]\right\} \tag{13}$$

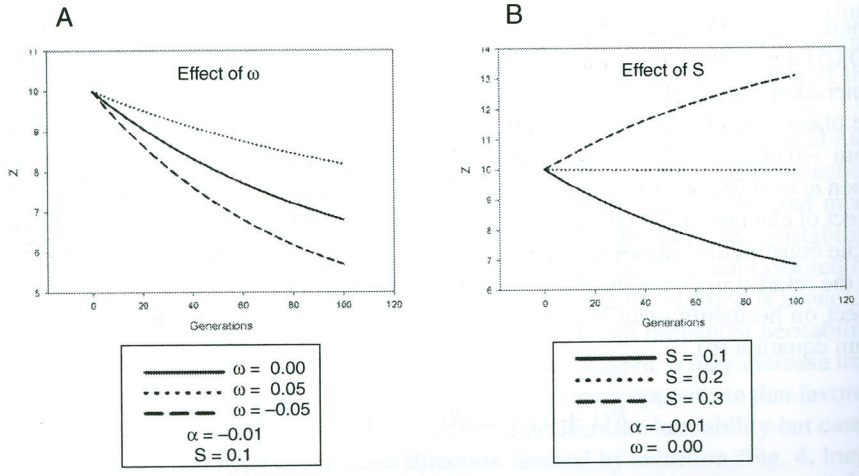

Fig. 5 Cases in which niche construction causes a negative response to selection. *Panel A* shows effects of different magnitudes of ω. $\alpha = -0.01$, $S = 0.1$, $h^2 = 0.5$. *Panel B* shows effects of different strengths of selection (S) favoring an increase in z. $\alpha = -0.01$, $\omega = 0$, $h^2 = 0.5$

In summary, when the newly evolved trait is associated with a change in the environment experienced by the organism—that is, when niche construction occurs—that new environment can influence both phenotypic expression, via phenotypic plasticity, and the heritability of the trait under selection. Whether these dynamics accompanying niche construction accelerate, retard, or reverse evolutionary responses to selection depends on the relative magnitudes and directions of the effect of the newly constructed environment on plasticity and the expression of genetic variation. Importantly, environment-dependent heritability can actually override the effects of "negative plasticity", or plasticity in the opposite direction from that favored by selection. Thus genotype-environment interaction, which results in environment-dependence of heritability and other genetic parameters, is important to consider when trait evolution causes a change in the environment.

6 Discussion

This univariate model shows that the dynamics of niche construction can influence evolutionary responses to selection. They can accelerate, retard, or reverse responses to selection. They can also impose genetic constraints on responses to selection, or they can enable more sustained responses to selection.

Accelerated, retarded, or reversed responses to selection can occur through the effects of the environment on phenotypic expression alone. These dynamics have been demonstrated in previous literature on maternal effects, in which the magnitude of the maternal effect measures the degree of correlation between maternal and offspring phenotype that is not due to genetic inheritance but rather due to phenotypic plasticity (Kirkpatrick and Lande 1989; Wade 1998). Similar dynamics have also been reported more generally in models of "indirect genetic effects", in which the social environment (genetic composition of groups of interacting individuals) evolves over time and also influences plastic social behaviour (Moore et al. 1997; Wolf 2003; Wolf et al. 1998, 1999, 2004). The basic dynamic occurs when selection causes a response in a trait mean, and when the trait causes a plastic response in that same trait or a different trait. The results presented here recapitulate those dynamics.

Also included in the model presented here are effects of the environment on the expression of trait heritabilities. In the case of environmental effects on trait heritabilities alone, the environment alters rates of evolutionary change. What is particularly interesting is that environmental effects on trait heritability can impose a severe constraint on evolutionary responses if an evolutionary change in the mean trait causes an opposite effect on the heritability of the trait, as for example, when an adaptive increase in the trait causes lower heritability of the trait (Donohue 2005; Donohue et al. 2005d). It should be emphasized that this reduction in heritability is due not to the selective elimination of maladaptive genotypes causing a reduction in genetic variance. Instead, it is caused by genotype-environment interaction whereby

even the exact same sample of genotypes can be less phenotypically distinct (exhibit less genetic variance) in the new environment.

Conversely, an evolutionary increase in the population mean trait may increase heritability of that trait. In this case, more sustained responses to selection may be possible, even when maladaptive genotypes are being eliminated from the population and the number of distinct genotypes actually decreases. If the phenotypic differences among the remaining genotypes become more pronounced in the new environment, then the depletion of genetic variation due to the selective elimination of genotypes can be counteracted.

The model presented above excludes some components that would be important especially for predicting evolutionary responses under natural, as opposed to artificial, selection. First, with changes in the phenotype and phenotypic variances, natural selection would also be expected to change as a direct consequence. When the intensity of selection is measured in units of the raw trait as opposed the standardized trait (as may be more relevant for cases of hard selection), a scalar change in phenotype (by magnitude α) would result in a scalar change in directional selection (literally, the difference in the mean of the unstandardized trait before and after selection). The departures are even more apparent when natural selection is stabilizing as opposed to purely directional, since increases or decreases in trait variance due to plasticity (with phenotypic variance scaling as α^2) would directly affect the intensity of stabilizing selection. These effects on selection would be further complicated by additional changes to genetic variances caused by genotype-environment interaction (ω). Greater resolution of the dynamics might also be possible by considering environmental effects on genetic versus environmental variances of traits separately, and this would be especially important for a multivariate analysis, in which phenotypic correlations influence patterns of multivariate selection. Finally, the model here considers only the univariate case. Most examples of niche construction probably occur with multiple interacting traits, as in the example of germination and flowering phenology discussed above. In such cases, environment-dependent covariances among traits are likely to alter the dynamics appreciably. In the case of maternal effects, interactions among correlated traits can cause "cycles" of feedbacks that lead to oscillating responses to selection (Kirkpatrick and Lande 1989), which illustrates the importance of considering trait evolution in multivariate terms. Future investigations of genotype-environment interactions with niche construction will need to incorporate their consequences to natural selection and will need to consider these dynamics within a multivariate context.

When the constructed environment influences both trait expression and trait heritability, the evolutionary trajectories and outcomes depend on the relative magnitudes of the effect of the environment on these two factors. In particular, environmental effects on heritability can counteract or augment effects of phenotypic plasticity. To predict these dynamics requires knowledge of how environments influence both trait expression and the heritability traits, the traits with which organisms influence their exposure to particular environmental factors, and the evolutionary potential of these niche-constructing traits.

Experimental demonstrations of these dynamics are lacking, but departures from expectation in empirical evolutionary studies, including breeding programs, may be due in part to unmeasured changes in the environment accompanying evolutionary change. More explicit studies of the dynamics of niche construction would be useful for predicting responses to selection in both artificial and natural populations. Acknowledging the manner in which all organisms alter their exposure to environmental factors, moreover, can increase the precision of evolutionary predictions and contribute to our understanding of evolutionary dynamics more generally.

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