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Development and Evolution of Character Displacement

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Abstract

Character displacement occurs when competition for either resources or successful reproduction imposes divergent selection on interacting species, causing divergence in traits associated with resource use or reproduction. Here, we describe how character displacement can be mediated either by genetically canalized changes (i.e., changes that reflect allelic or genotype frequency changes) or by phenotypic plasticity. We also discuss how these two mechanisms influence the tempo of character displacement. Specifically, we suggest that, under some conditions, character displacement mediated by phenotypic plasticity might occur more rapidly than that mediated by genetically canalized changes. Finally, we describe how these two mechanisms may act together and determine character displacement's mode, such that it proceeds through an initial phase in which trait divergence is environmentally induced to a later phase in which divergence becomes genetically canalized. This plasticity-first hypothesis predicts that character displacement should be generally mediated by ancestral plasticity and that it will arise similarly in multiple, independently evolving populations. We conclude by highlighting future directions for research that would test these predictions.

Keywords

adaptive divergence; competition; ecological developmental biology; genetic assimilation; genetics of adaptation; phenotypic plasticity

“Imagine a case in which two such species have been squeezed together in the same communities long enough for evolution to occur. When they first came into contact, they were elastic and could diverge in their habits enough to lessen competition. The differences were phenotypic, the result of environment and not genes. The compression occurred in traits that were relatively easy to change, most likely by a retreat from parts of the habitat and diet by one or both of the species. As the generations passed, genetic differences arose and hardened the distinction between the two species.” (Wilson, p. 174)¹

Introduction

Among evolutionary biology's most persistent challenges is to explain the origins of biodiversity. Darwin's² proposed solution to this problem was his “principle of divergence of character.” According to this principle, all organisms face recurring competition for scarce resources, and this competition favors individuals that are least like their competitors in resource use and associated traits. In other words, Darwin posited that competition is a

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Conflicts of interest

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primary agent of adaptive trait evolution. Moreover, Darwin argued that groups of organisms that compete should become increasingly dissimilar over time, possibly even transforming into separate species. Thus, selection deriving from competition, Darwin² held, is the primary engine of diversification.

Abundant evidence now indicates that such competition-induced selection can indeed promote adaptive—and divergent—trait evolution through an evolutionary process now known as “character displacement”^{3–6}. Moreover, researchers have come to recognize that character displacement can be driven by not only resource competition (as Darwin proposed), but also by costly reproductive interactions, such as when different species hybridize or compete for reproductive trait-space used in attracting mates^{3,4,7}. Character displacement therefore assumes two forms: “ecological character displacement”^{8,9}, which stems from resource competition, and “reproductive character displacement”¹⁰, which stems from deleterious reproductive interactions. Both ecological and reproductive character displacement derive from selection stemming from any direct or indirect interactions between organisms that reduce each individual's access to either resources or successful reproductive opportunities and is therefore deleterious [on average] to all parties^{11,12}. Because our arguments throughout this paper apply equally to either process, we do not treat ecological and reproductive character displacement separately here. Moreover, for the purposes of this paper, “competition” refers to either costly reproductive interactions or competition for resources¹³.

Because of its diversifying nature, character displacement is thought to play a vital role in the origins, maintenance, abundance, and distribution of biodiversity^{5,6,13,14}. Yet, previous research has focused primarily on when or whether character displacement occurs^{5–7}. By contrast, relatively little work has explored the proximate mechanisms of character displacement^{13,14}, although this is changing. With the advent of new technology and approaches, investigators have more opportunities than ever to identify the genetic and developmental basis of character displacement in a greater diversity of systems.

Our goal in this paper is to examine the proximate mechanisms that underpin character displacement; discuss how these mechanism influence character displacement's speed; and evaluate how these alternative mechanisms interact and affect the means by which character displacement transpires. We focus specifically on contrasting genetically canalized trait evolution versus the expression and evolution of plasticity in mediating character displacement.

Before proceeding, however, we must make an important caveat. In particular, character displacement, by its nature, is a form of adaptive evolution. The mechanisms that underpin character displacement therefore do not differ from other forms of adaptive trait evolution. We do not intend to suggest otherwise in this paper, and in that sense, character displacement is not “special.” Much of what we outline below applies to any other form of adaptive evolution and could be similarly applied to other contexts. What then is the need for understanding the proximate bases of character displacement per se?

Our answer to this question is two-fold. First, the study of character displacement provides a context in which to study both the nature of adaptive evolution and its role in diversification^{5,13,14}. Indeed, insights gained from the study of character displacement can contribute to the understanding of adaptive evolution more broadly. Yet, other types of interactions (e.g., predation¹⁵ or parasitism¹⁶) are similarly suited for studying adaptive trait evolution. Nevertheless, such interactions are not generally expected to drive diversification in the same way that character displacement does. Indeed, as Darwin² first held, competitively mediated selection acts as a “wedge” that should continually drive interacting

species apart within their respective communities. Thus, although adaptive evolution can be studied in other contexts, the study of character displacement provides powerful opportunities for relating adaptive evolution to diversification.

Second, whether or not character displacement arises can dictate a population's persistence. Unlike other forms of trait evolution where populations may persist in suboptimal states, the absence of character displacement can actually result in the local extinction of one species by another, specifically, via competitive exclusion^{17,18} or reproductive exclusion^{19,20}. In this paper, we suggest that alternative proximate mechanisms may actually be more or less likely to promote character displacement. If different mechanisms can influence the nature of character displacement, as well as whether or not it even occurs in the first place^{13,14}, identifying these proximate mechanisms becomes crucial for evaluating when and why character displacement occurs. Thus, in contrast with other considerations of adaptive trait evolution where proximate mechanisms can be treated as a "black box" (i.e., the particular form of mechanism generating a trait may be interesting in its own right, but does not necessarily affect whether or not adaptive evolution occurs), a full understanding of character displacement's role in evolutionary diversification may ultimately rest on identifying its underlying mechanisms.

Proximate Mechanisms of Character Displacement

Character displacement arises when species come to differ from one another in traits associated with resource use or reproduction^{8,21–25}. At the mechanistic level, how does such divergence arise?

Generally, the focus in character displacement has been on *genetic* differentiation between interacting species and, within each such species, between populations in sympatry and allopatry with the other species¹³. Specifically, most research presumes that divergence is mediated exclusively by genetically canalized changes (i.e., divergence that reflects allelic or genotype frequency changes). This focus on genetic differentiation has seemingly arisen, in part, for two reasons. First, models of character displacement generally assume specific genetic architectures (e.g., single locus versus multi-locus)^{8,21} and genetic processes (e.g., mutation, gene flow)^{24,26}. Empirical tests of the predictions of such alternative genetic models necessarily require investigations of the genetic basis of character displacement (see below for more detail). Second, a focus on genetic differentiation has also emerged because, among the widely accepted criteria used to define character displacement, is the requirement that putative cases of character displacement should reflect genetic differentiation of populations and species^{4,23,27–30}.

Although genetically canalized traits have largely been the focus of proximate studies of character displacement, Wilson's¹ quote at the beginning of this paper highlights an alternative mechanism that can underpin displacement: namely, phenotypic plasticity. In the context of character displacement, plasticity is manifest as competitively mediated trait expression (i.e., trait expression that varies depending on the presence of competition). Although not generally considered in the theory of character displacement (except in models involving learning and cultural transmission³¹), plasticity can effectively generate trait divergence between species and thereby serve as additional axis of variation on which selection can act to promote the evolution of traits that minimize competitive interactions between species. Indeed, because many populations exhibit heritable variation in whether and how individuals respond through environmentally induced change^{32,33}, plasticity can itself evolve^{34–36}, and plastic traits can therefore satisfy the criteria for demonstrating character displacement^{37,38}. Thus, as we describe in greater detail below, evolved environmentally induced niche shifts can constitute character displacement.

Note, however, that genetic canalization and plasticity are not mutually exclusive mechanisms of trait production. Instead, these two proximate mechanisms are best thought of as occupying different positions along a continuum of environmental influences on trait production³⁹, with strict genetic control of trait variation at one end of the continuum and with pure environmental induction of trait variation at the opposite end. In reality, most traits have both genetic and environmental components⁴⁰. Moreover, plasticity in one or more traits is often required to hold another trait constant in the face of changing environmental conditions (thereby maintaining homeostasis)⁴¹, further demonstrating the interdependence of these two influences on development.

Perhaps more importantly, these two proximate mechanisms are often evolutionarily interchangeable⁴², meaning that selection can slide trait regulation anywhere along this continuum^{39,41,42}. Specifically, when genetic variation for the degree of environmental influence is present, then selection can act on this variation to promote the evolution of either increased or decreased environmental sensitivity⁴². If selection eliminates all environmental influences (i.e., if “genetic assimilation”^{43,44} occurs), the end result is a genetically canalized trait (note that selection can also promote “epigenetic assimilation,” wherein trait expression becomes less sensitive to environmental influences due to inherited environmental effects⁴⁵, such as a maternal effect⁴⁶, cultural transmission⁴⁷, or parasite transmission⁴⁸).

With genetic assimilation, the evolution of a new trait does not require the emergence of new genes or new gene complexes; instead, selection acts on *existing* genetic architecture and epigenetic interaction^{32,49,50}. In other words, a plastic trait can be converted into a canalized trait (or, alternatively, it can be converted into a trait that shows enhanced plasticity) through evolutionary adjustments in the regulation of trait expression. Experiments have demonstrated such evolutionary shifts in the degree to which populations are sensitive to environmental influences³⁴, including the complete loss of plasticity⁴⁴, and numerous examples of evolution by natural selection might be explained by genetic assimilation^{42,51–53}.

Genetic assimilation might be a more common mechanism of character displacement than has been heretofore appreciated³⁹. We will focus on the potential role of genetic assimilation in character displacement later in this paper. Before we do so, however, we briefly review separately the evidence for genetically canalized traits in character displacement and plasticity, respectively.

Genetic Mechanisms of Divergence

Competitively mediated selection is expected to target genes that influence the expression of phenotypes involved in resource use or reproduction. Although relatively few studies have thus far identified these selective targets, the studies that have been conducted to date suggest that a diversity of genetic mechanisms can mediate character displacement. Four such mechanisms are briefly reviewed below.

First, character displacement might arise via a “single-allele mechanism”^{54–56}. With such a mechanism, divergence arises between interacting species because the *same* allele in both species enhances existing differences between them. An allele that enhances sensory sensitivity to male sexual signals, for example, might render females better at identifying conspecific males and thereby promote reproductive character displacement. For instance, in fruit flies, *Drosophila pseudoobscura* and *D. persimilis*, a single allele potentially mediates species recognition⁵⁷. Females of both species from sympatric populations exhibit greater discrimination against heterospecifics compared to females from allopatric populations⁵⁸. This discrimination ability in both species appears to be at least partly mediated by a single

allele at the *Coy-2* chromosomal region that enhances discrimination against heterospecifics (likely by influencing a female's ability to detect species-specific olfactory cues)^{57,59}. Although additional mechanisms might also be involved in mate discrimination in these flies^{59,60}, this system illustrates how costly reproductive interactions between species can be reduced by a single allele shared by both species.

Single-allele mechanisms may mediate ecological character displacement as well. Enhanced sensory sensitivity might also refine pre-existing food preferences within different species and thereby reduce overlap between them in resource use. Moreover, a single allele might reduce dispersal tendencies, which could exaggerate differences between species in habitat use and thereby preclude interactions between them. Such possibilities will remain speculative, however, until more is known regarding the proximate mechanisms underlying traits that are involved in minimizing resource competition between species.

A second, slightly more complex mechanism—but one still involving a single locus—entails *alternative* alleles at a locus between the interacting species that specifies divergent resource-use or reproductive traits. Such allelic differentiation at a single locus might suffice to reduce competitive or reproductive interactions between species. For example, passion-vine butterflies (genus *Heliconius*) show great diversity in wing color patterns. This genus has undergone rapid speciation⁶¹, but many of its constituent species have also converged in wing color pattern owing to Müllerian mimicry^{62,63}. However, for species that use wing coloration in mate choice (such as *H. cydno* and *H. pachinus*, where males use wing color to discriminate conspecific from heterospecific mates⁶⁴), such convergence in wing color pattern increases the risk of hybridization⁶⁵. Thus, interacting species are expected to undergo reproductive character displacement as a means of reducing such costly interactions. Recent research suggests that a single locus might encode both mate preference and wing coloration⁶⁴. Because pigments involved in wing coloration also occur in the eye (and affect perception of wing coloration)⁶⁴, a single gene affects both coloration and perception of—and preference for—that coloration⁶⁶. Therefore, species that possess alternative alleles at a single locus can diverge in traits such as sexual signaling and mate choice that mediate character displacement. If that locus has pleiotropic effects, as in *Heliconius* butterflies, then divergence in multiple traits can arise simultaneously.

In contrast to mechanisms (such as those above) that depend either on a single allele or on alternative alleles at a single locus (with or without pleiotropy), a third mechanism arises when character displacement is under the control of multiple, divergent loci. A possible example comes from pied flycatchers (*Ficedula hypoleuca*) and collared flycatchers (*F. albicollis*), which have undergone reinforcement of female preferences and male coloration⁶⁷. Such divergence appears to be mediated by multiple, divergent loci⁶⁸. Similarly, character displacement between benthic and limnetic species of stickleback fish (*Gasterosteus aculeatus* complex²⁷) involves multiple traits that appear to be encoded by numerous loci: F₁ hybrids are intermediate in morphology between the two parent species, and these differences persist over multiple generations in a common laboratory environment^{69–72}.

At present, it is unclear how commonly character displacement arises through the different mechanisms above. Theoretical models suggest that character displacement (reproductive character displacement or reinforcement, in particular) will be more likely to arise under a one-allele mechanism than a two-allele mechanism⁵⁵. However, other models suggest that character displacement can also arise when mediated by multiple loci^{21,22}.

Given that character displacement typically entails complex suites of traits^{73,74}, one might expect that competitively mediated selection would target multiple loci. Nevertheless, a key

(theoretical and empirical) challenge has been to explain how such a mechanism would persist in the face of recombination, which would tend to break up co-adapted gene complexes that encode species differences^{75,76}. This is particularly problematic in species that exchange genes (as has been documented in species that have undergone character displacement, including sticklebacks⁷⁷, spadefoot toads⁷⁸, Darwin's finches⁷⁹, and nightingales⁸⁰), because hybridization tends to "scramble" allelic combinations at loci that isolate species. One possibility is that the loci encoding species differences might reside in areas that are protected from recombination^{75,76,81,82}, such as inside chromosomal inversions⁸¹, near the chromosome's centromere⁸², or on sex chromosomes⁶⁷.

In the three previous mechanisms (i.e., single-allele, multiple alleles at a single-locus, and multiple-loci mechanisms), the genes involved in mediating character displacement encode for proteins that produce the trait(s) that undergo divergence (e.g., cuticular hydrocarbons used in mate recognition in fruit flies; color pigments in butterflies). In other words, character displacement involves changes in the protein-encoding regions of the genome. Yet, differences in protein-coding sequences are not the only means by which species could become differentiated^{83–88}. Instead, species might diverge in *cis*-regulatory (noncoding) regions that are involved in resource use or reproduction. Thus, a fourth mechanism involves divergence in regions of the genome that regulate the expression of the genes that encode for displaced traits between species.

A putative example comes from Darwin's finches, which have undergone both ecological and reproductive character displacement in the size and shape of their beaks^{89,90} (the size and shape of an individual's beak determines not only which resources it can utilize⁸⁹, it can also affect the production of male song, which is used in territorial defense and mate attraction⁹¹).

Recent studies of how bird beaks form developmentally point to the possible genetic targets of divergent selection in this system. Beak development is influenced by several genes that encode a series of signaling molecules, including fibroblast growth factor 8 (*Fgf8*) and sonic hedgehog (*Shh*). These two gene products influence the expression of a third signaling molecule, bone morphogenetic protein 4 (*Bmp4*), which governs differences between species in beak depth^{92,93}. These two gene products also affect the expression of the gene *calmodulin* (*CaM*), which encodes for a calcium-binding protein involved in apoptosis that influences beak length⁹³.

Although any one of these four genes could conceivably serve as a target of selection during character displacement, these genes are also involved in several crucial metabolic processes (e.g., *CaM* is involved in numerous, vital cellular processes). Thus, any changes in these genes would likely have deleterious consequences. Consequently, it is unlikely that selection would favor alternative alleles at these genes in different species and populations. Instead, character displacement has likely occurred when selection brought about changes in the regulation of these genes⁸⁹.

Regulatory mutations are similarly thought to be involved in competitively induced divergence in pheromones (specifically, cuticular hydrocarbons) that are used in mate choice in the *Drosophila serrata* complex⁹⁴. Such regulatory mutations have also been implicated in mediating reproductive character displacement (specifically, floral-color divergence) in the Texas wildflower, *Phlox drummondii*⁹⁵. Although the role of regulatory mutations during adaptation remains controversial^{96,97}, such mutations are increasingly viewed as being important during adaptive population divergence⁹⁸.

In sum, studies of the genetic targets of competitively mediated selection have revealed a diversity of genetic mechanisms, which appear to differ in the degree to which they facilitate

character displacement. However, we still do not know whether one mechanism is more prevalent than the others in mediating character displacement. Although putative examples exist for each mechanism, additional work across a diversity of systems is critically needed to determine whether some genetic mechanisms are more likely to underpin character displacement than others as predicted by theory⁵⁵. In the next section, we consider an alternative mechanism that might be effective at mediating character displacement: phenotypic plasticity.

Mechanisms of Environmentally Induced Divergence

In contrast to reflecting genetically canalized differences, divergent traits might alternatively arise through phenotypic plasticity. Specifically, rather than being produced constitutively (as with the genetic mechanisms described above), divergent traits might be expressed facultatively, such that they are produced in an individual only when it experiences competition from a heterospecific (e.g., see Figure 1). Although relatively few studies have explicitly considered plasticity's role in character displacement (notwithstanding Wilson's¹ quote at the outset of this article), such environmentally contingent niche shifts may play an underappreciated role in mediating character displacement^{13,14,99}. Below, we discuss two different mechanisms by which plasticity may promote character displacement. Before we do so, we begin with a caveat.

In particular, competitively mediated plasticity may or may not constitute character displacement^{37,38}. Character displacement is defined as trait evolution that arises as an adaptive response to resource competition or deleterious reproductive interactions between species^{4,5,9,13,14,100}. Competitively mediated plasticity therefore constitutes character displacement only when it has actually evolved in direct response to competitively mediated selection and when it lessens competitive interactions between heterospecifics.

Even when environmentally induced traits have not themselves undergone character displacement, they can still be critical to the process. Specifically, lineages that respond to heterospecific competitors through facultative adjustments in their phenotype may be less likely to go extinct via competitive or reproductive exclusion. Indeed, plasticity is increasingly viewed as playing a critical role in shielding populations from extinction when confronted with changing environments^{39,101,102}. In the context of competitive interactions, plasticity might enable individuals to produce resource-use or reproductive phenotypes that are less like the phenotypes expressed by their competitors. In this way, competitively mediated plasticity might provide a mechanism for reducing the frequency and intensity of competitive interactions. Thus, even in the absence of character displacement, plasticity may promote species coexistence through resource partitioning¹⁰³ or reproductive partitioning. Such a process could serve to promote developmentally mediated species sorting, in which species express different phenotypes at contact, but not owing to pre-existing *canalized* differences^{104,105}.

Many organisms appear to possess the ability to respond to heterospecifics through facultative adjustments of their phenotype^{42,103,106–109}. For example, when confronted with a heterospecific competitor, the individual members of many species of plants facultatively express traits that reduce interspecific competition, such as altering the spatial positioning of their roots^{110–113}, modifying the width of their leaves¹¹⁴, or adjusting their physiology¹¹⁵. Similarly, when faced with a heterospecific competitor, many species of fish¹⁰⁹ and amphibians^{37,38} facultatively express resource-use morphologies that differ from those expressed by the heterospecific. Some species can even facultatively adjust their mate preferences in the presence of heterospecifics, thereby reducing costly reproductive interactions between species¹¹⁶. Of such cases, several systems have illustrated that

plasticity does mediate character displacement^{37,38,99,109,117,118}, via two different mechanisms.

First, plasticity may underlie character displacement when competitively mediated selection leads to the evolution of a reaction norm that minimizes competitive interactions. Under this mechanism, selection acts on underlying heritable variation in either the tendency to respond to competitors in the first place or the manner in which individuals express these responses (or both) in propelling competitively mediated trait *evolution* (i.e., character displacement). Such evolved shifts in environmentally induced resource-use or reproductive traits can satisfy the widely accepted criteria for demonstrating character displacement³⁷. In particular, experiments have demonstrated that these induced shifts are indeed caused by the presence of a heterospecific competitor per se (Figure 1) and that they lessen costly interactions with the heterospecific that induces them^{37,116}. Furthermore, experimentally induced shifts often mirror, in magnitude and direction, the fixed phenotypic differences between naturally occurring sympatric and allopatric populations (e.g., see Figure 1). Finally, facultative shifts have been shown to evolve in sympatry versus allopatry^{38,116}.

A second general mechanism by which plasticity may promote character displacement is when it is transmitted reliably *across* generations and thereby forms the basis of an alternative inheritance system on which adaptive evolution can unfold. There are two types of transgenerational plasticity that may be important in mediating character displacement. The first are maternal effects, which occur when a female's phenotype influences her offspring's phenotype, independent of the direct effects of her coding sequences on her offspring's phenotype^{46,119–123}. Because they can be induced by interspecific competition^{117,120}, mediate adaptive phenotypic change^{46,121,122,124,125}, and be transmitted reliably across generations¹²⁶, maternal effects might play an underappreciated role in promoting character displacement¹¹⁷. A second form of transgenerational plasticity is cultural inheritance, mediated by learning. In many animals, for example, preference for conspecific mates is learned^{89,127,128}. Likewise, many animal species learn to utilize new food resources^{129,130}. Once a population acquires such learned mate or food preferences, these preferences can be transmitted across generations and even reinforce differences between species, thereby mediating ecological or reproductive character displacement^{127,130}. Such a situation is illustrated in Darwin's finches, where learning played a crucial role in mediating reproductive character displacement¹²⁷.

In sum, plasticity may play a role in mediating character displacement. However, more empirical and theoretical work is needed to determine whether and how each of the above mechanisms contributes to character displacement. For the remainder of the article, we consider some ways in which plasticity may play an important role in character displacement. In particular, we contrast plasticity and genetic canalization in terms of their effects on the rapidity with which character displacement can occur. Although the two mechanisms are by no means mutually exclusive, they differ in important ways that can ultimately affect when and how character displacement occurs.

Tempo and Mode of Character Displacement

As we highlighted in the introduction, character displacement is a form of adaptive evolution, and the above mechanisms are therefore not unique to character displacement. Nevertheless, the mechanisms mediating character displacement may determine when and how it occurs. In this section, we focus on this issue.

We first consider each mechanism's impacts on the speed of character displacement. We do so, because character displacement is a time-limited process. Populations experiencing competition are often at risk of competitive or reproductive exclusion. Therefore,

populations that do not respond sufficiently rapidly to the presence of competition risk extinction. Indeed, a meta-analysis by Schluter⁵ indicated that some taxa are more likely to undergo character displacement than others. Although such variation may reflect researcher bias in terms of the systems chosen to study character displacement, such a pattern may also indicate that character displacement is more likely to occur in some conditions versus others. One such condition that may be important is the ease and rapidity with which character displacement takes place. Thus, consideration should be given to the differential rate by which genetically canalized versus plastic traits are likely to evolve, in addition to the other conditions that appear to facilitate character displacement (e.g., strong selection, certain genetic architectures, initial trait differences)^{21,25,131,132}. Such differences in the speed of evolution for these different proximate mechanisms could therefore explain when character displacement is more—or less—likely to occur.

Why Proximate Mechanisms Likely Differ in Speed of Divergence

A key factor that determines the speed of character displacement is the amount of standing variation in the trait(s) under divergent selection^{21,131,133}. When abundant standing variation is present in a population that encounters a heterospecific competitor, competitively mediated selection could act on this variation, filtering out those variants that are the most similar to the heterospecific competitor and preserving those that are most dissimilar. This process fuels character displacement^{13,133}, and, if standing variation is abundant and selection is strong, then the rate of such an evolutionary response could be rapid in that it could transpire in a handful of generations (Darwin's finches provide a possible example of such rapid evolution¹³⁴).

When standing variation is depleted (or is initially absent), however, character displacement, like any other form of adaptive evolution, may be precluded^{5,133,135}. In particular, for character displacement to proceed, new variants must be introduced into the population. If divergence depends entirely on genetically canalized differences, new variants must be introduced into the population via mutation, recombination, or gene flow from another conspecific population or another species (through hybridization).

All three of the above means of acquiring new variants are likely to either transpire relatively slowly or, ironically, simultaneously counteract character displacement even as they provide the raw material necessary for character displacement to proceed. For example, the waiting time for favorable, new mutations to arise and spread in a population can take many generations¹³⁶. Indeed, many genetic models of character displacement predict that the pace of evolution will be slow, especially during the early phase of divergence^{21,22}. Yet, if character displacement transpires slowly, the risk of competitive or reproductive exclusion increases¹³. Moreover, although recombination and gene flow can potentially introduce favorable, new genetic variants into a population more quickly than does mutation, these processes will typically impact only a few individuals in any given generation. Perhaps more critically, theory has shown that high gene flow and recombination actually reduce a population's ability to respond to competitors, because maladaptive gene combinations over time swamp adaptive combinations²⁶. Thus, even though variants might be introduced that are favored by competitively mediated selection, the population may continue to receive an influx of gene combinations that ultimately prevent local adaptive to competition.

By contrast, divergence driven by phenotypic plasticity might promote a rapid adaptive response to competitors, for at least three reasons. First, an important consequence of environmentally induced phenotypes is that their expression can reveal cryptic genetic variation—genetic variation that is normally not visible as phenotypic variation¹³⁷. Indeed, hidden reaction norms (variation that is phenotypically expressed only after an organism experiences changes in environmental conditions) may store an evolutionarily significant

pool of cryptic genetic variation upon which selection may act³³. Therefore, with plasticity, abundant standing variation (which could fuel divergent trait evolution) can be released and exposed to selection as soon as a population experiences competition^{33,138}. Essentially, the waiting time between when a population begins to experience competitively mediated selection and when it begins to express variation on which selection can act to produce an adaptive response to competitors is negligible—the process by which new variants are produced occurs over developmental time or over the course of an individual's lifetime (as with learning) rather than over generations.

Second, environmentally induced changes typically occur in numerous individuals simultaneously⁴² (especially when a population experiences exploitative competition). This situation contrasts markedly with a mechanism in which the production of new divergent traits requires the introduction of new genetic variants, which typically arise in just a few, or even just one, individual (see above). Moreover, because individuals often differ in whether and how they respond to an environmental cue (such as the presence of a heterospecific competitor), this variation increases the likelihood and speed of an evolutionary response. Although this line of argument has been developed most prominently by West-Eberhard^{42,108,139,140} to explain rapid adaptive evolution generally, the application of these ideas to the realm of character displacement could be fruitful, because it potentially broadens the conditions under which character displacement is predicted to occur.

A third reason why plasticity might mediate rapid character displacement is that the ability to respond to competition through plasticity may already exist in a given population, having evolved as an adaptive response to *intraspecific* competition. For example, forms of plasticity present within species that may mediate character displacement between species include phytochrome-mediated shade-avoidance responses in plants¹¹⁴, resource polymorphism in many species of fish and amphibians¹⁴¹, and mating polymorphism in many invertebrate and vertebrate species^{142,143}. Note that, in some populations, intraspecific competition may also favor genetically canalized variation, such as a genetic polymorphism^{144,145}, on which competitively mediated selection can act. However, unless this constitutively expressed variation is maintained in a population by selection, it is at risk of being lost over time. By contrast, with environmental induction, unexpressed (cryptic) genetic variation can be “protected” from selection or chance loss until it is released by a change in the environment³³.

The presence of pre-existing plasticity (and underlying cryptic genetic variation) is important, because as noted in the introduction, the evolution of a new divergent trait (such as an alternative morphology or behavior that reduces competitive interactions with a heterospecific) need not require new genes or new gene complexes. Instead, selection can repurpose *existing* genetic pathways and developmental mechanisms and thereby drive rapid, divergent trait evolution. In the context of character displacement, if a population has already evolved the ability to respond to intraspecific competition via environmentally induced changes, the same mechanisms involved in such a response might be co-opted to mediate adaptive divergence between heterospecific competitors (i.e., character displacement). Indeed, empirical data exist (described below) to suggest that intraspecific variation generated by plasticity might form the basis for interspecific variation during character displacement. Essentially, pre-existing plasticity (and the underlying developmental mechanisms) might enable character displacement to evolve rapidly along lines of least resistance¹⁴⁶.

Above, we contrasted the speed with which character displacement arises via genetic canalization versus competitively mediated plasticity. However, as noted earlier, these alternative proximate mechanisms of character displacement are not mutually exclusive and

will potentially evolve in concert. Yet, because of the difference in speed with which they can occur, plasticity may initially underpin competitively mediated divergence, with genetic canalization evolving subsequently^{1,118}. We describe how this sequence of events might unfold in the next section.

Evolution of Character Displacement: Plasticity First?

Evolutionary biologists have long hypothesized that phenotypic plasticity might precede, and even promote, the genetically canalized traits that arise during adaptive evolution^{32,39,42,44,53,139,147–153}. This “plasticity-first” hypothesis rests on the argument that, if selection acts on quantitative genetic variation regulating the expression of initially environmentally dependent traits, it can lead to the evolution of either enhanced plasticity or, alternatively, even the loss of plasticity; i.e., genetic assimilation⁴⁴. Such loss might result as an incidental by-product of selection favoring a single phenotypic trait (or an extreme phenotype in a continuum) to the exclusion of all other phenotypes^{43,44,154}. Alternatively, plasticity might be lost when the alleles that underlie it are lost through stochastic processes. In particular, when an environmental change results in one trait rarely being produced in a population (such as when, in the presence of a competitor, an individual fails to produce a resource-use or reproductive trait expressed in the competitor’s absence; Figure 1), then alleles that regulate expression of this “hidden” phenotype would not be exposed to selection and would therefore be at greater risk of chance loss^{155,156}.

That such genetic assimilation could serve as a mechanism of character displacement has not been heretofore generally appreciated^{13,14}, despite Wilson’s¹ suggestion, quoted at the outset of the paper. Consider, for example, two species that possess pre-existing plasticity in resource-use or reproductive phenotypes. Once in sympatry, each species might facultatively express a different subset of the initial phenotypes (Figure 2). Over time, each species might lose plasticity and become fixed (either through selection or chance) for a different alternative phenotype¹⁵³. Character displacement might begin with an initial phase in which each species evolves the expression of competitively induced plastic phenotypes and then subsequently transition to genetically canalized traits that minimize competitive interactions with the alternate species (Figure 2).

This plasticity-first hypothesis, of course, is not the only way in which character displacement might evolve—plasticity might not play *any* role in some species or populations, whereas in other species, plasticity might play a solitary role by mitigating against selection for any further genetically canalized divergence¹⁵⁷. However, character displacement might be first mediated by plasticity for two reasons. First, those populations that initially express plasticity might be more likely buffered from extinction while genetically canalized traits evolve that harden the distinction between species^{13,14}. When faced with a new, superior competitor, populations lacking the ability to respond through plasticity might simply undergo reproductive or competitive exclusion before canalized differences evolve¹⁵⁸. Second, even in populations where plasticity successfully minimizes competitive interactions with heterospecifics, genetic canalization of such traits might be favored if plasticity has costs^{159,160}.

This plasticity-first hypothesis has two key implications for the way in which character displacement occurs. First, character displacement may not always proceed slowly. As noted above, although most models of character displacement predict that divergence will occur slowly, observations of natural populations have revealed that character displacement can proceed rapidly (i.e., within a few generations)^{78,134,161–165}. This discrepancy between the models and the data may arise, because most models assume that divergence is based on genetically canalized differences, which (as noted above) can evolve slowly. If, instead, divergence is mediated by plasticity, then character displacement might generally proceed

rapidly. Although such rapid divergence can also be explained in terms of abundant standing genetic variation (see above), the incorporation of plasticity into models of character displacement could increase the parameter space over which character displacement can occur.

Second, character displacement that is mediated by plasticity and followed by genetic assimilation should generate repeated evolution of the same ecotype (specifically, one that minimizes interspecific competition) in multiple, independently evolving sympatric populations. Such parallel evolution of reproductive or ecological traits among independently evolving sympatric populations that are experiencing similar selective pressures from competition (“parallel character displacement”) has indeed been documented in a number of systems^{166–171}. Although the plasticity first hypothesis does not uniquely predict parallel character displacement (standing variation in genetically canalized traits could respond similarly to the same selective pressures across different populations¹⁶⁷), if stochastic processes (mutation, recombination) serve as the basis of variation in genetically canalized traits that mediate character displacement, then such parallel evolution becomes less likely: populations will be more likely to evolve *different* routes in response to competitors. By contrast, with pre-existing plasticity, the same sets of phenotypes are repeatedly revealed when individuals in different, independent populations experience similar selective pressures from competition. Thus, the environment might play a critical role in not only exerting parallel selection pressures in different populations, but also in *generating* parallel distributions of traits on which selection acts^{14,39}.

Empirical Support for the Plasticity-First Hypothesis

Several systems in which character displacement has been documented appear to conform to the plasticity-first scenario. Specifically, we highlight research on three systems, in which competitively induced plasticity appears to have preceded the evolution of canalized genetic differences.

The first case comes from stickleback fish (*Gasterosteus aculeatus* complex). Two species occur together in certain small coastal lakes in southwestern Canada: one expresses a “benthic” phenotype, whereas the other expresses a “limnetic” phenotype¹⁷². These two species are thought to have arisen following the invasion from the ocean of an ancestral limnetic ecotype into lakes that already contained an intermediate ecotype. Following this invasion, both ecological and reproductive character displacement resulted in the parallel evolution of a new benthic ecotype within each such lake, which replaced the ancestral, intermediate ecotype¹⁷².

Recall from above that the phenotypic differences between these benthic and limnetic ecotypes appear to reflect genetically canalized differences. Recent experiments have revealed, however, that diet-induced plasticity is present in marine sticklebacks¹⁷³, which are thought to represent the ancestral colonists¹⁷². More importantly, this diet-induced plasticity generates phenotypes that resemble the benthic and limnetic ecotypes found in modern-day (derived) freshwater populations¹⁷³. This recent work indicates that trait divergence between ecotypes was initially environmentally induced, but ultimately may have become genetically canalized. Such plasticity in resource-use traits has been similarly detected in dozens of species of freshwater fish^{109,174–176}, including in many that have undergone character displacement¹⁰⁹, indicating that this route to character displacement is likely not unique to sticklebacks.

A second such example comes from two species of North American spadefoot toads (*Spea multiplicata*, *S. bombifrons*), which have undergone ecological character displacement in tadpole trophic morphology^{37,38,99,164,170,177}. Where they co-occur, almost all individuals

of one species (*S. multiplicata*) develop into an omnivore morph, which feeds mostly on detritus, whereas almost all individuals of the other species (*S. bombifrons*) develop into a distinctive carnivore morph, which specializes on eating anostracan shrimp and other tadpoles (Figure 1a, b).

The proximate mechanism of character displacement appears to differ for these two species. In *S. multiplicata*, the phenotypic differences between sympatric and nearby allopatric populations appear to reflect a condition-dependent maternal effect¹¹⁷. By contrast, in *S. bombifrons*, the phenotypic differences between sympatric and allopatric populations appear to reflect genetically canalized differences¹¹⁸.

In allopatric populations (i.e., ancestral, pre-displacement populations), however, *both* species have the capacity to respond adaptively to the presence of the other species through phenotypic plasticity. Yet, despite the fact that individuals from allopatric populations of both species have the capacity to produce *both* carnivores and omnivores^{99,164}, when allopatric *S. multiplicata* are experimentally exposed to *S. bombifrons*, they facultatively produce mostly omnivores (Figure 1c), which is similar to the pattern of morph expression found among *S. multiplicata* in naturally occurring, populations in sympatry (Figure 1b). Conversely, when allopatric *S. bombifrons* are experimentally exposed to *S. multiplicata*, they facultatively produce mostly carnivores (Figure 1c), which is similar to the pattern of morph expression found among *S. bombifrons* in naturally occurring, populations in sympatry (Figure 1b). Finally, phylogenetic analyses have found that sympatry is the derived condition in this system¹⁷⁰. Thus, experimentally initiated niche shifts in ancestral (allopatric) populations mirror in direction and magnitude the canalized shifts observed in derived (sympatric) populations.

Because plasticity in allopatry is ancestral, these data suggest that character displacement in spadefoots might have evolved from an initial phase in which trait divergence was environmentally induced (as observed in modern-day allopatric populations) to one in which divergence became either developmentally canalized (as in modern-day sympatric populations of *S. multiplicata*, where character displacement is mediated by a maternal effect¹¹⁷) or genetically canalized (as in modern-day sympatric populations of *S. bombifrons*, where character displacement appears to be mediated by genetic shifts¹¹⁸). Presumably, sympatric populations of these two species differ in the mechanism of character displacement because they differ in the length of time that each has been in sympatry¹¹⁸, with sympatric populations of *S. bombifrons* having been in sympatry longer than sympatric populations of *S. multiplicata* (due to a range expansion by *S. bombifrons* into the region where character displacement occurs)¹⁷⁸.

Spadefoots therefore highlight two points: (1) they are consistent with the plasticity-first hypothesis for character displacement; and (2) they show that canalized differences need not be strictly genetic. Regarding the second point, a maternal effect in *S. multiplicata* (a form of transgenerational plasticity¹⁷⁹) has led to canalized differences between the species in morph production in sympatry. Thus, rather than being genetically canalized, the phenotypic differences in *S. multiplicata* appear to be *epigenetically* canalized. As noted above, epigenetic assimilation (such as that mediated by a maternal effect) may be an evolutionary precursor to genetic assimilation (recall from above that sympatric populations of *S. multiplicata* have only recently experienced competitively mediated selection imposed by *S. bombifrons*).

Finally, a third example consistent with the plasticity-first hypothesis for character displacement comes from *Anolis* lizards from the Greater Antilles. When different species occur together, they differ in microhabitat use^{180,181}. Specifically, on islands where they are

sympatric, different species of *Anolis* occur as different ecomorphs, which partition their habitat in different ways, residing (for example) in tree crowns (crown-giant ecomorph), on trunk/crowns (trunk-crown ecomorph), on the trunk/ground (trunk-ground ecomorph), and on twigs (twig ecomorph)^{180,181}. Different ecomorphs tend to differ in body size and limb length, and manipulative experiments have revealed that at least two species possess the ability to facultatively change limb length in response to different-diameter perches¹⁸².

Although species differences in limb length might have evolved (at least partly) through a process of species sorting¹⁰⁵ (in which species differences might have arisen through the differential invasion into a habitat of species that happen to differ, or through the differential extinction of species that happen to be too similar to coexist), some differences also have potentially evolved through character displacement^{105,183}, and this process might have begun with environmentally induced niche shifts¹⁸². This system therefore serves as one in which the plasticity-first hypothesis could be tested more extensively.

In sum, several case studies provide evidence that is consistent with the plasticity-first hypothesis for character displacement. Nevertheless, much more work is needed to assess how general this mechanism is for explaining character displacement. As we discuss in more detail below, additional theoretical and empirical work is needed to fully consider plasticity's role in character displacement, and to determine if competitively mediated plasticity ultimately leads to the sort of genetically canalized traits described in the section above on Genetic Mechanisms of Divergence.

Conclusions and Future Directions

Despite character displacement's importance in helping to explain how new species arise, diversify, and coexist^{5,6,13,14}, relatively little is known of the proximate mechanisms that mediate character displacement. From a proximate perspective, the focus in studies of character displacement has been on understanding how—and what kinds of—genetically canalized differences evolve between populations and species⁵. Yet, increasing evidence suggests that character displacement can alternatively arise through competitively induced phenotypic plasticity^{13,14}. It is important to recognize that plasticity can mediate character displacement, because, in some populations and species, plasticity might represent the sole means by which adaptive, divergent traits arise. Perhaps more importantly, from an evolutionary perspective, character displacement might often unfold rapidly as it transitions from an initial phase in which species differences arises through phenotypic plasticity to one in which such divergence is underlain by genetically canalized differences¹¹⁸.

With recent theoretical and technical advances, now is a propitious time to both identify the mechanisms of character displacement and critically evaluate how different mechanisms interact during character displacement. Indeed, a number of key questions regarding the proximate basis of character displacement remain unanswered. Here, we highlight five specific questions that promise to provide fruitful avenues for future research.

First, what are the proximate mechanisms of character displacement in diverse taxa? At present, much of what we know comes from studies of relatively few organisms. Additional research on a greater variety of taxa is needed to clarify whether the mechanisms that have already been identified are present in diverse taxa, or whether these mechanisms are unique to the few taxa that have been studied thus far. Generally, there is insufficient data upon which to conclude whether some mechanisms are more prevalent than others.

Second, do different proximate mechanisms generate differences in the ease with which (and, hence, the likelihood that) character displacement occurs? In contrast to genetically canalized differences, shifts underlain by phenotypic plasticity might generally occur more

rapidly and affect many more individuals simultaneously⁴². Consequently, because competitively mediated plasticity buffers populations from extinction via competitive or reproductive exclusion¹⁵⁸, it might increase the likelihood that character displacement will transpire to completion¹³. Additional theoretical and empirical approaches are required to evaluate these predictions. For example, experimental or comparative studies could be used to determine whether, when confronted with a novel competitor, populations (or species) consisting of more plastic genotypes undergo character displacement more rapidly (and more readily) than those consisting of less plastic genotypes. Indeed, this question would be ideally suited for experimental evolution studies with rapidly evolving organisms, such as microbes^{184,185}.

Third, does phenotypic plasticity sometimes impede character displacement? Evolutionary biologists have long argued that phenotypic plasticity can dampen selection for diversification, for two reasons¹⁸⁶. First, plasticity allows a single genotype to produce multiple phenotypes in response to different environmental (and hence, selective) regimes, and thus genetic alternatives are not required for attaining alternative fitness optima, such as those associated with populations in sympatry with a competitor versus those in allopatry. Second, different genotypes can produce the same phenotype via plastic responses, thereby hiding genetic differences between them from selection. Future theoretical and empirical research should seek to evaluate whether there are conditions under which competitively induced plasticity impedes, rather than facilitates, character displacement. In other words, an organism might be so plastic in its responses to competitors that no further evolution transpires. In such a case, plasticity may be more likely to mediate species sorting rather than character displacement.

Fourth, can competitively induced phenotypes lose their environmental sensitivity over evolutionary time and thereby undergo genetic assimilation? Experimental studies with rapidly evolving organisms might also prove informative for addressing this question³⁴. Moreover, additional work comparing ancestral populations with derived populations that have undergone character displacement would be valuable in addressing this question.

Finally, does character displacement proceed initially by the evolution of plastic traits and subsequently transition to a phase in which genetically canalized traits evolve and mediate competitive interactions between species? If so, systems in which contact is recent should be more likely to display character displacement in plastic traits, whereas in systems where contact is older, trait differences should be mediated by genetically canalized differences. Addressing this issue is contingent, in part, on answering the fourth question above. Nevertheless, future research should aim to identify additional systems in which to evaluate the plasticity-first hypothesis for the evolution of character displacement. Only by doing so will it be possible to ultimately determine the conditions under which this route occurs and how common it is.

In addressing the above questions, equal emphasis should be placed on doing so in the contexts of both ecological character displacement and *reproductive* character displacement. Whether the relative importance of alternative mechanisms, and the nature of their interaction, is the same for these two forms of character displacement is itself an open question.

Additional research into the proximate bases of character displacement promises to have far-reaching ramifications. Indeed, because character displacement is central in the origins, abundance, and distribution of biodiversity^{5,6,13,14}, understanding its causes can help illuminate some of the most fundamental issues in evolutionary biology and ecology, including how new species arise, how they diversify, and how they coexist.

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Bibliography

1. Wilson, EO. The diversity of life. Cambridge, MA: Harvard University Press; 1992.
2. Darwin, C. The annotated origin: a facsimile of the first edition of *On the origin of species*. J. T. Costa, annotator. Cambridge, MA: The Belknap Press of Harvard University Press; 1859. (2009).
3. Brown WL, Wilson EO. Character displacement. *Systematic Zoology*. 1956; 5:49–64.
4. Grant PR. Convergent and divergent character displacement. *Biological Journal of the Linnean Society*. 1972; 4:39–68.
5. Schluter, D. The ecology of adaptive radiation. Oxford, U. K.: Oxford University Press; 2000.
6. Dayan T, Simberloff D. Ecological and community-wide character displacement: the next generation. *Ecology Letters*. 2005; 8:875–894.
7. Howard, DJ. Reinforcement: origin, dynamics, and fate of an evolutionary hypothesis. In: Harrison, RG., editor. *Hybrid zones and the evolutionary process*. New York: Oxford University Press; 1993. p. 46–69.
8. Slatkin M. Ecological character displacement. *Ecology*. 1980; 61:163–177.
9. Schluter, D. Ecological character displacement. In: Fox, CW.; Roff, DA.; Fairbairn, DJ., editors. *Evolutionary ecology: concepts and case studies*. New York: Oxford University Press; 2001. p. 265–276.
10. Crozier RH. Niche shape and genetical aspects of character displacement. *American Zoologist*. 1974; 14:1151–1157.
11. Odum, EP. *Fundamentals of ecology*. Philadelphia, PA: Saunders; 1959.
12. Dybzinski, R.; Tilman, D. Competition and coexistence in plant communities. In: Levin, SA., editor. *The Princeton guide to ecology*. Princeton, NJ: Princeton University Press; 2009. p. 186–195.
13. Pfennig KS, Pfennig DW. Character displacement: ecological and reproductive responses to a common evolutionary problem. *Quarterly Review of Biology*. 2009; 84:253–276. [PubMed: 19764283]
14. Pfennig DW, Pfennig KS. Character displacement and the origins of diversity. *American Naturalist*. 2010; 176:S26–S44.
15. Endler, JA. Interactions between predators and prey. In: Krebs, JR.; Davies, NB., editors. *Behavioural ecology: an evolutionary approach*. London: Blackwell; 1991. p. 169–196.
16. Zuk M, et al. Silent night: adaptive disappearance of a sexual signal in a parasitized population of field crickets. *Biology Letters*. 2006; 2:521–524. [PubMed: 17148278]
17. Gause, GF. *The struggle for existence*. Baltimore: Williams and Wilkins; 1934.
18. Hardin G. The competitive exclusion principle. *Science (Washington, D.C.)*. 1960; 131:1292–1297.
19. Gröning J, Hochkirch A. Reproductive interference between animal species. *Quarterly Review of Biology*. 2008; 83:257–282. [PubMed: 18792662]
20. Kishi S, et al. Reproductive interference determines persistence and exclusion in species interactions. *Journal of Animal Ecology*. 2009; 78:1043–1049. [PubMed: 19457018]
21. Doebeli M. An explicit genetic model for ecological character displacement. *Ecology*. 1996; 77:510–520.
22. Taper ML, Case TJ. Quantitative genetic models for the coevolution of character displacement. *Ecology*. 1985; 66:355–371.
23. Taper ML, Case TJ. Coevolution among competitors. *Oxford Surv. Evol. Biol*. 1992; 8:63–109.

24. Kirkpatrick M. Reinforcement and divergence under assortative mating. *ProcRSoc. Lond. Ser. B-Biol. Sci.* 2000; 267:1649–1655.
25. Liou LW, Price TD. Speciation by reinforcement of premating isolation. *Evolution.* 1994; 48:1451–1459.
26. Case TJ, Taper ML. Interspecific competition, environmental gradients, gene flow, and the coevolution of species' borders. *American Naturalist.* 2000; 155:583–605.
27. Schluter D, McPhail JD. Ecological character displacement and speciation in sticklebacks. *American Naturalist.* 1992; 140:85–108.
28. Losos JB. Ecological character displacement and the study of adaptation. *Proceedings of the National Academy of Sciences, U.S.A.* 2000; 97:5693–5695.
29. Arthur W. The evolutionary consequences of interspecific competition. *Adv. Ecol. Res.* 1982; 12:127–187.
30. Connell JH. Diversity and the coevolution of competitors, or the ghost of competition past. *Oikos.* 1980; 35:131–138.
31. Servedio MR, et al. Reinforcement and learning. *Evolutionary Ecology.* 2009; 23:109–123.
32. Schlichting, CD.; Pigliucci, M. Phenotypic evolution: a reaction norm perspective. Sunderland, MA: Sinauer; 1998.
33. Schlichting CD. Hidden reaction norms, cryptic genetic variation, and evolvability. *Annals of the New York Academy of Science.* 2008; 1133:187–203.
34. Suzuki Y, Nijhout HF. Evolution of a polyphenism by genetic accommodation. *Science.* 2006; 311:650–652. [PubMed: 16456077]
35. Aubret F, Shine R. Genetic assimilation and the postcolonization erosion of phenotypic plasticity in island Tiger Snakes. *Current Biology.* 2009; 19:1932–1936. [PubMed: 19879141]
36. De Meester L. Evolutionary potential and local genetic differentiation in a phenotypically plastic trait of a cyclical parthenogen. *Daphnia magna.* *Evolution.* 1996; 50:1293–1298.
37. Pfennig DW, Murphy PJ. Character displacement in polyphenic tadpoles. *Evolution.* 2000; 54:1738–1749. [PubMed: 11108600]
38. Pfennig DW, Murphy PJ. How fluctuating competition and phenotypic plasticity mediate species divergence. *Evolution.* 2002; 56:1217–1228. [PubMed: 12144021]
39. Pfennig DW, et al. Phenotypic plasticity's impacts on diversification and speciation. *Trends in Ecology and Evolution.* 2010; 25:459–467. [PubMed: 20557976]
40. Gilbert, SF.; Epel, D. Ecological developmental biology: integrating epigenetics, medicine, and evolution. Sunderland, MA: Sinauer Associates; 2009.
41. Whitman, DW.; Agrawal, AA. What is phenotypic plasticity and why is it important?. In: Whitman, DW.; Ananthakrishnan, TN., editors. *Phenotypic plasticity of insects.* Enfield, NH: Science Publishers; 2009. p. 1-63.
42. West-Eberhard, MJ. Developmental plasticity and evolution. New York: Oxford University Press; 2003.
43. Waddington CH. Selection of the genetic basis for an acquired character. *Nature.* 1952; 169:278. [PubMed: 14910749]
44. Waddington CH. Genetic assimilation of an acquired character. *Evolution.* 1953; 7:118–126.
45. Rossiter MC. Incidence and consequences of inherited environmental effects. *Annu. Rev. Ecol. Syst.* 1996; 27:451–476.
46. Mousseau, TA.; Fox, CW. Maternal effects as adaptations. New York: Oxford University Press; 1998.
47. Payne RB, et al. Imprinting and the origin of parasite-host associations in brood-parasitic indigobirds. *Vidua chalybeata. Anim. Behav.* 2000; 59:69–81.
48. Engelstadter J, Hurst GDD. The ecology and evolution of microbes that manipulate host reproduction. *Annu. Rev. Ecol. Evol. Syst.* 2009; 40:127–149.
49. Emlen DJ, et al. On the origin and evolutionary diversification of beetle horns. *Proceedings of the National Academy of Sciences of the United States of America.* 2007; 104:8661–8668. [PubMed: 17494751]

50. Ledón-Rettig CC, et al. Ancestral variation and the potential for genetic accommodation in larval amphibians: implications for the evolution of novel feeding strategies. *Evolution and Development*. 2008; 10:316–325. [PubMed: 18460093]
51. Matsuda, R. Animal evolution in changing environments with special reference to abnormal metamorphosis. New York: John Wiley and Sons; 1987.
52. Hall, BK. Evolutionary developmental biology. Dordrecht: Kluwer; 1999.
53. Moczek AP, et al. The role of developmental plasticity in evolutionary innovation. *Proceedings of The Royal Society, Series B*. 2011
54. Felsenstein J. Skepticism toward Santa Rosalia, or why are there so few kinds of animals? *Evolution*. 1981; 35:124–138.
55. Servedio MR, Noor MAF. The role of reinforcement in speciation: Theory and data. *Annu. Rev. Ecol. Syst.* 2003; 34:339–364.
56. Coyne, JA.; Orr, HA. Speciation. Sunderland, MA: Sinauer; 2004.
57. Ortiz-Barrientos D, Noor MAF. Evidence for a one-allele assortative mating locus. *Science* (Washington, D.C.). 2005; 310:1467–1467.
58. Noor MA. Speciation driven by natural selection in *Drosophila*. *Nature*. 1995; 375:674–675. [PubMed: 7791899]
59. Ortiz-Barrientos D, et al. The genetics of speciation by reinforcement. *Plos Biology*. 2004; 2:2256–2263.
60. Barnwell CV, Noor MAF. Failure to replicate two mate preference QTLs across multiple strains of *Drosophilapseudoobscura*. *Journal of Heredity*. 2008; 99:653–656. [PubMed: 18728083]
61. Brower AVZ. Parallel race formation and the evolution of mimicry in *Heliconius* butterflies: a phylogenetic hypothesis from mitochondrial DNA sequences. *Evolution*. 1996; 50:195–221.
62. Turner JRG. Adaptation and evolution in *Heliconius*—a defense of neodarwinism. *Annu. Rev. Ecol. Syst.* 1981; 12:99–121.
63. Mallet J, Gilbert LE. Why are there so many mimicry rings—correlations between habitat, behavior and mimicry in *Heliconius* butterflies. *Biological Journal of the Linnean Society*. 1995; 55:159–180.
64. Kronforst MR, et al. Linkage of butterfly mate preference and wing color preference cue at the genomic location of wingless. *Proceedings Of The National Academy Of Sciences Of The United States Of America*. 2006; 103:6575–6580. [PubMed: 16611733]
65. Estrada C, Jiggins CD. Interspecific sexual attraction because of convergence in warning colouration: is there a conflict between natural and sexual selection in mimetic species? *Journal of Evolutionary Biology*. 2008; 21:749–760. [PubMed: 18312559]
66. Chamberlain NL, et al. Polymorphic butterfly reveals the missing link in ecological speciation. *Science*. 2009; 326:847–850. [PubMed: 19892982]
67. Saetre GP, et al. A sexually selected character displacement in flycatchers reinforces premating isolation. *Nature*. 1997; 387:589–592.
68. Backstrom N, et al. A high-density scan of the Z chromosome in *Ficedula* flycatchers reveals candidate loci for diversifying selection. *Evolution*. 2010; 64:3461–3475. [PubMed: 20629730]
69. McPhail JD. Ecology and evolution of sympatric sticklebacks (*Gasterosteus*): morphological and genetic evidence for a species pair in Enos Lake, British Columbia. *CanJZool.-Rev. Can. Zool.* 1984; 62:1402–1408.
70. McPhail JD. Ecology and evolution of sympatric sticklebacks (*Gasterosteus*): evidence for a species pair in Paxton Lake, Texada Island, British Columbia. *CanJZool.-Rev. Can. Zool.* 1992; 70:361–369.
71. Hatfield T. Genetic divergence in adaptive characters between sympatric species of sticklebacks. *American Naturalist*. 1997; 149:1009–1029.
72. Hatfield T, Schluter D. Ecological speciation in sticklebacks: environment-dependent hybrid fitness. *Evolution*. 1999; 53:866–873.
73. Smith RA, Rausher MD. Experimental evidence that selection favors character displacement in the ivyleaf morning glory. *American Naturalist*. 2008; 171:1–9.

74. Martin RA, Pfennig DW. Evaluating the targets of selection during character displacement. *Evolution*. 2011 **in press**.
75. Butlin RK. Recombination and speciation. *Molecular Ecology*. 2005; 14:2621–2635. [PubMed: 16029465]
76. Hoffmann AA, Rieseberg LH. Revisiting the impact of inversions in evolution: from population genetic markers to drivers of adaptive shifts and speciation? *Annu. Rev. Ecol. Evol. Syst.* 2008; 39:21–42. [PubMed: 20419035]
77. Behm JE, et al. Breakdown in postmating isolation and the collapse of a species pair through hybridization. *American Naturalist*. 2010; 175:11–26.
78. Pfennig KS. A test of alternative hypotheses for the evolution of reproductive isolation between spadefoot toads: support for the reinforcement hypothesis. *Evolution*. 2003; 57:2842–2851. [PubMed: 14761062]
79. Grant BR, Grant PR. Fission and fusion of Darwin's finches populations. *Philosophical Transactions of the Royal Society B-Biological Sciences*. 2008; 363:2821–2829.
80. Reifova R, et al. Ecological character displacement in the face of gene flow: evidence from two species of nightingales. *BMC Evol Biol*. 2011; 11:138. [PubMed: 21609448]
81. Noor MAF, et al. Chromosomal inversions and the reproductive isolation of species. *Proceedings of the National Academy of Sciences, U.S.A.* 2001; 98:12084–12088.
82. Noor MAF, Bennett SM. Islands of speciation or mirages in the desert? Examining the role of restricted recombination in maintaining species. *Heredity*. 2009; 103:439–444. [PubMed: 19920849]
83. King MC, Wilson AC. Evolution at two levels in humans and chimpanzees. *Science*. 1975; 188:107–116. [PubMed: 1090005]
84. Raff, RA.; Kauffman, TC. *Genes, embryos, and evolution*. New York: MacMillan; 1983.
85. Carroll, SB., et al. *From DNA to diversity: molecular genetics and the evolution of animal design*. Malden, MA: Blackwell; 2001.
86. Wilkins, AS. *The evolution of developmental pathways*. Sunderland, MA: Sinauer Associates; 2002.
87. Wray GA. The evolutionary significance of *cis*-regulatory mutations. *Nature Reviews Genetics*. 2007; 8:206–216.
88. Wray, GA. Embryos and evolution: 150 years of reciprocal illumination. In: Bell, MA.; Futuyma, DJ.; Eanes, WF.; Levinton, JS., editors. *Evolution since Darwin: the first 150 years*. Sunderland, MA: Sinauer Associates, Inc.; 2010. p. 215–239.
89. Grant, PR.; Grant, BR. *How and why species multiply: the radiation of Darwin's finches*. Princeton, NJ: Princeton University Press; 2008.
90. Lack, D. *Darwin's finches*. Cambridge: Cambridge University Press; 1947.
91. Podos J. Correlated evolution of morphology and vocal signal structure in Darwin's finches. *Nature*. 2001; 409:185–188. [PubMed: 11196640]
92. Abzhanov A, et al. *Bmp* and morphological variation of beaks in Darwin's finches. *Science*. 2004; 305:1462–1465. [PubMed: 15353802]
93. Abzhanov A, et al. The calmodulin pathway and evolution of elongate beak morphology in Darwin's finches. *Nature*. 2006; 442:563–567. [PubMed: 16885984]
94. McGraw EA, et al. High-dimensional variance partitioning reveals the modular genetic basis of adaptive divergence in gene expression during reproductive character displacement. *Evolution*. 2011 **in press**.
95. Hopkins R, Rausher MD. Identification of two genes causing reinforcement in the Texas wildflower. *Phlox drummondii*. *Nature*. 2011:411–414.
96. Hoekstra HE, Coyne JA. The locus of evolution: evo devo and the genetics of adaptation. *Evolution*. 2007; 61:995–1016. [PubMed: 17492956]
97. Razeto-Barry P, Maldonado K. Adaptive *cis*-regulatory changes may involve few mutations. *Evolution*. 2011 **in press**.
98. Pavey SA, et al. The role of gene expression in ecological speciation. *Annals of the New York Academy of Science: The Year in Evolutionary Biology*. 2010; 1206:110–129.

99. Pfennig DW, et al. Ecological opportunity and phenotypic plasticity interact to promote character displacement and species coexistence. *Ecology*. 2006; 87:769–779. [PubMed: 16602305]
100. Schluter, D. Character displacement. In: Pagel, M., editor. *Encyclopedia of evolution*. Oxford, UK: Oxford University Press; 2002. p. 149–150.
101. Pfennig DW, McGee M. Resource polyphenism increases species richness: a test of the hypothesis. *Phil. Trans. Roy. Soc. Lond, Ser. B*. 2010; 365:577–591. [PubMed: 20083634]
102. Chevin LM, et al. Adaptation, plasticity, and extinction in a changing environment: towards a predictive theory. *Plos Biology*. 2010; 8 e1000357.
103. Schoener TW. Resource partitioning in ecological communities. *Science*. 1974; 185:27–39. [PubMed: 17779277]
104. Case TJ, Sidell R. Pattern and chance in the structure of model and natural communities. *Evolution*. 1983; 37:832–849.
105. Losos JB. A phylogenetic analysis of character displacement. *Evolution*. 1990; 44:558–569.
106. Agrawal AA. Phenotypic plasticity in the interactions and evolution of species. *Science* (Washington, D. C.). 2001; 294:321–326.
107. Fordyce JA. The evolutionary consequences of ecological interactions mediated through phenotypic plasticity. *Journal of Experimental Biology*. 2006; 209:2377–2383. [PubMed: 16731814]
108. West-Eberhard, MJ. Behavior and evolution. In: Grant, PR.; Horn, HS., editors. *Molds, molecules, and metazoa: growing points in evolutionary biology*. Princeton, NJ: Princeton University Press; 1992. p. 57–75.
109. Robinson BW, Wilson DS. Character release and displacement in fish: a neglected literature. *American Naturalist*. 1994; 144:596–627.
110. Nobel PS. Root distribution and seasonal production in the northwestern Sonoran desert for a C3 subshrub, a C4 bunchgrass, and a CAM leaf succulent. *American Journal of Botany*. 1997; 84:949–955. [PubMed: 21708649]
111. Cahill JF, et al. Plants integrate information about nutrients and neighbors. *Science*. 2010; 328:1657. [PubMed: 20576883]
112. Mahall BE, Callaway RM. Root communication among desert shrubs. *Proceedings of the National Academy of Sciences*. 1991; 88:874–876.
113. Gersani M, et al. Tragedy of the commons as a result of root competition. *Journal of Ecology*. 2001; 89:660–669.
114. Schmitt J, et al. Manipulative approaches to testing adaptive plasticity: phytochrome-mediated shade-avoidance responses in plants. *American Naturalist*. 1999; 154:S43–S54.
115. Rhee G-Y. Effects of N : P atomic ratios and nitrate limitation on algal growth, cell composition, and nitrate uptake. *Limnol. Oceanogr*. 1978; 23:10–25.
116. Pfennig KS. Facultative mate choice drives adaptive hybridization. *Science* (Washington, D. C.). 2007; 318:965–967.
117. Pfennig DW, Martin RA. A maternal effect mediates rapid population divergence and character displacement in spadefoot toads. *Evolution*. 2009; 63:898–909. [PubMed: 19154374]
118. Pfennig DW, Martin RA. Evolution of character displacement in spadefoot toads: different proximate mechanisms in different species. *Evolution*. 2010; 64:2331–2341. [PubMed: 20394671]
119. Donohue, K.; Schmitt, J. Maternal environmental effects in plants: adaptive plasticity?. In: Mousseau, TA.; Fox, CW., editors. *Maternal Effects as Adaptations*. New York: Oxford University Press; 1998. p. 137–158.
120. Allen RM, et al. Offspring size plasticity in response to intraspecific competition: an adaptive maternal effect across life-history stages. *American Naturalist*. 2008; 171:225–237.
121. Badyaev AV, et al. Sex-biased hatching order and adaptive population divergence in a passerine bird. *Science* (Washington, D. C.). 2002; 295:316–318.
122. Badyaev AV. Maternal effects as generators of evolutionary change: a reassessment. *Annals of the New York Academy of Science*. 2008; 1133:151–161.

123. Räsänen K, Kruuk LEB. Maternal effects and evolution at ecological time-scales. *Functional Ecology*. 2007; 21:408–421.
124. Galloway LF, Etterson JR. Transgenerational plasticity is adaptive in the wild. *Science* (Washington, D. C.). 2007; 318:1134–1136.
125. Uller T. Developmental plasticity and the evolution of parental effects. *Trends In Ecology & Evolution*. 2008; 23:432–438. [PubMed: 18586350]
126. Plaistow SJ, et al. Context-dependent intergenerational effects: The interaction between past and present environments and its effect on population dynamics. *American Naturalist*. 2006; 167:206–215.
127. Grant BR, Grant PR. Songs of Darwin's finches diverge when a new species enters the community. *Proceedings of the National Academy of Sciences of the United States of America*. 2010; 107:20156–20163. [PubMed: 21048082]
128. Kozak GM, et al. Sexual imprinting on ecologically divergent traits leads to sexual isolation in sticklebacks. *Proceedings of the Royal Society, Series B*. 2011
129. Papaj DR, Prokopy RJ. Ecological and evolutionary aspects of learning in phytophagous insects. *Annual Review of Entomology*. 2011; 34:315–350.
130. Price, T. Speciation in birds. Greenwood Village, CO: Roberts and Company Publishers; 2008.
131. Milligan BG. Evolutionary divergence and character displacement in two phenotypically-variable competing species. *Evolution*. 1985; 39:1207–1222.
132. Martin RA, Pfennig DW. Evaluating the targets of selection during character displacement. *Evolution*. 2011
133. Rice AM, Pfennig DW. Character displacement: in situ evolution of novel phenotypes or sorting of pre-existing variation? *Journal of Evolutionary Biology*. 2007; 20:448–459. [PubMed: 17305810]
134. Grant PR, Grant BR. Evolution of character displacement in Darwin's finches. *Science* (Washington, D. C.). 2006; 313:224–226.
135. Barrett RDH, Schluter D. Adaptation from standing genetic variation. *Trends in Ecology and Evolution*. 2008; 23:38–44. [PubMed: 18006185]
136. Phillips PC. Waiting for a compensatory mutation: phase zero of the shifting-balance process. *Genetical Research*. 1996; 67:271–283. [PubMed: 8690275]
137. Gibson G, Dworkin I. Uncovering cryptic genetic variation. *Nature Reviews Genetics*. 2004; 5:1199–1212.
138. Ledón-Rettig CC, et al. Diet and hormone manipulations reveal cryptic genetic variation: implications for the evolution of novel feeding strategies. *Proceedings of the Royal Society of London, Series B*. 2010; 277:3569–3578. [PubMed: 20573627]
139. West-Eberhard MJ. Phenotypic plasticity and the origins of diversity. *Annu. Rev. Ecol. Syst.* 1989; 20:249–278.
140. West-Eberhard MJ. Developmental plasticity and the origin of species differences. *Proceedings of the National Academy of Sciences, U.S.A.* 2005; 102:6543–6549.
141. Smith TB, Skúlason S. Evolutionary significance of resource polymorphisms in fishes, amphibians, and birds. *Annu. Rev. Ecol. Syst.* 1996; 27:111–133.
142. Emlen DJ. The evolution of animal weapons. *Annual Review of Ecology, Systematics, and Evolution*. 2008; 39:387–413.
143. Gross MR. Alternative reproductive strategies and tactics: diversity within sexes. *Trends in Ecology and Evolution*. 1996; 11:92–98. [PubMed: 21237769]
144. Levene H. Genetic equilibrium when more than one ecological niche is available. *American Naturalist*. 1953; 87:331–333.
145. Clarke BC. The contribution of ecological genetics to evolutionary theory: detecting the direct effects of natural selection on particularly polymorphic loci. *Genetics (Supplement)*. 1975; 79:101–113.
146. Schluter D. Adaptive radiation along genetic lines of least resistance. *Evolution*. 1996; 50:1766–1774.
147. Baldwin JM. A new factor in evolution. *American Naturalist*. 1896; 30:441–451.

148. Schmalhausen, II. Factors of evolution: the theory of stabilizing selection. Chicago: University of Chicago Press; 1949. [1986].
149. Pigliucci M, Murren CJ. Genetic assimilation and a possible evolutionary paradox: Can macroevolution sometimes be so fast as to pass us by? *Evolution*. 2003; 57:1455–1464. [PubMed: 12940351]
150. Pál C, Miklos I. Epigenetic inheritance, genetic assimilation and speciation. *Journal Of Theoretical Biology*. 1999; 200:19–37. [PubMed: 10479537]
151. Price TD, et al. The role of phenotypic plasticity in driving genetic evolution. *Proceedings of the Royal Society of London, Series B*. 2003; 270:1433–1440. [PubMed: 12965006]
152. Moczek AP. On the origins of novelty in development and evolution. *Bioessays*. 2008; 30:432–447. [PubMed: 18404691]
153. Schwander T, Leimar O. Genes as leaders and followers in evolution. *Trends In Ecology & Evolution*. 2011; 26:143–151. [PubMed: 21257223]
154. Lande R. Adaptation to an extraordinary environment by evolution of phenotypic plasticity and genetic assimilation. *Journal of Evolutionary Biology*. 2009; 22:1435–1446. [PubMed: 19467134]
155. Masel J, et al. The loss of adaptive plasticity during long periods of environmental stasis. *American Naturalist*. 2007; 169:38–46.
156. Lahti DC, et al. Relaxed selection in the wild. *Trends In Ecology & Evolution*. 2009; 24:487–496. [PubMed: 19500875]
157. Schlichting, CD. The role of phenotypic plasticity in diversification. In: DeWitt, TJ.; Scheiner, SM., editors. *Phenotypic plasticity: functional and conceptual approaches*. New York: Oxford University Press; 2004. p. 191–200.
158. Pijanowska J, et al. Phenotypic plasticity within *Daphnia longispina* complex: differences between parental and hybrid clones. *Polish Journal of Ecology*. 2007; 55:761–769.
159. Relyea RA. Costs of phenotypic plasticity. *American Naturalist*. 2002; 159:272–282.
160. Auld JR, et al. Re-evaluating the costs and limits of adaptive phenotypic plasticity. *Proceedings of the Royal Society B-Biological Sciences*. 2010; 277:503–511.
161. Fenchel T. Character displacement and coexistence in mud snails (Hydrobiidae). *Oecologia*. 1975; 20:19–32.
162. Diamond J, et al. Rapid evolution of character displacement in Myzomelid honeyeaters. *American Naturalist*. 1989; 134:675–708.
163. Yom-Tov Y, et al. Competition, coexistence, and adaptation amongst rodent invaders to Pacific and New Zealand islands. *Journal of Biogeography*. 1999; 26:947–958.
164. Pfennig DW, Murphy PJ. A test of alternative hypotheses for character divergence between coexisting species. *Ecology*. 2003; 84:1288–1297.
165. Hoskin CJ, et al. Reinforcement drives rapid allopatric speciation. *Nature*. 2005; 437:1353–1356. [PubMed: 16251964]
166. Hansen TF, et al. Comparative analysis of character displacement and spatial adaptations as illustrated by the evolution of *Dalechampia* blossoms. *American Naturalist*. 2000; 156:S17–S34.
167. Schluter D, Nagel LM. Parallel speciation by natural selection. *American Naturalist*. 1995; 146:292–301.
168. Marko PB. An intraspecific comparative analysis of character divergence between sympatric species. *Evolution*. 2005; 59:554–564. [PubMed: 15856698]
169. Matocq MD, Murphy PJ. Fine-scale phenotypic change across a species transition zone in the genus *Neotoma*: disentangling independent evolution from phylogenetic history. *Evolution*. 2007; 61:2544–2557. [PubMed: 17927780]
170. Rice AM, et al. Parallel evolution and ecological selection: replicated character displacement in spadefoot toads. *Proceedings of the Royal Society, Series B*. 2009; 276:4189–4196.
171. Adams DC. Parallel evolution of character displacement driven by competitive selection in terrestrial salamanders. *BMC Evol Biol*. 2010; 10:1–10. [PubMed: 20044934]

172. Rundle, HD.; Schluter, D. Natural selection and ecological speciation in sticklebacks. In: Dieckmann, U.; Doebeli, M.; Metz, JAJ.; Tautz, D., editors. Adaptive speciation. Cambridge, UK: Cambridge University Press; 2004. p. 192-209.
173. Wund MA, et al. A test of the "flexible stem" model of evolution: ancestral plasticity, genetic accommodation, and morphological divergence in the threespine stickleback radiation. *American Naturalist*. 2008; 172:449–462.
174. Skúlason, S., et al. Sympatric morphs, populations and speciation in freshwater fish with emphasis on arctic charr. In: Magurran, AE.; May, RM., editors. Evolution of biological diversity. Oxford, UK: Oxford University Press; 1999. p. 70-92.
175. Meyer A. Phenotypic plasticity and heterochrony in *Ciclasoma managuense* (Pisces, Cichlidae) and their implications for speciation in cichlid fishes. *Evolution*. 1987; 41:1357–1369.
176. Robinson BW, Wilson DS. Genetic variation and phenotypic plasticity in a trophically polymorphic population of pumpkinseed sunfish (*Lepomis gibbosus*). *Evolutionary Ecology*. 1996; 10:631–652.
177. Pfennig DW, et al. Field and experimental evidence for competition's role in phenotypic divergence. *Evolution*. 2007; 61:257–271. [PubMed: 17348937]
178. Rice AM, Pfennig DW. Analysis of range expansion in two species undergoing character displacement: why might invaders generally "win" during character displacement? *Journal of Evolutionary Biology*. 2008; 21:696–704. [PubMed: 18341542]
179. Fox, CW.; Mousseau, TA. Maternal effects as adaptations for transgenerational phenotypic plasticity in insects. In: Mousseau, TA.; Fox, CW., editors. Maternal effects as adaptations. New York: Oxford University Press; 1998. p. 159-177.
180. Losos, JB. Lizards in an evolutionary tree: ecology and adaptive radiation of Anoles. Berkeley, CA: University of California Press; 2009.
181. Williams EE. The origin of faunas. Evolution of lizard congeners in a complex island fauna: a trial analysis. *Evolutionary Biology*. 1972; 6:47–89.
182. Losos JB, et al. Evolutionary implications of phenotypic plasticity in the hindlimb of the lizard *Anolis sagrei*. *Evolution*. 2000; 54:301–305. [PubMed: 10937208]
183. Schoener TW. Size patterns in West Indian *Anolis* lizards. 2. Correlations with the size of particular sympatric species—displacement and convergence. *American Naturalist*. 1970; 104:155–174.
184. Tyerman JG, et al. Experimental demonstration of ecological character displacement. *BMC Evol Biol*. 2008; 8:34. [PubMed: 18234105]
185. Kassen R. Toward a general theory of adaptive radiation: insights from microbial experimental evolution. *The Year in Evolutionary Biology 2009: Annals of the New York Academy of Sciences*. 2009; 1168:3–22.
186. Schlichting, CD. The role of phenotypic plasticity in diversification. In: DeWitt, TJ.; Scheiner, SM., editors. Phenotypic plasticity: functional and conceptual approaches. New York: Oxford University Press; 2004.
187. Pfennig, DW.; Pfennig, KS. Evolution's wedge: competition and the origins of diversity. Berkeley, CA: University of California Press; forthcoming.

Figure 1.

An experimental demonstration of how phenotypic plasticity might mediate ecological character displacement. Spadefoot toad tadpoles (*Spea multiplicata* and *S. bombifrons*) typically occur as (a) an omnivore morph, (b) which specializes on plant material and detritus, and (c) a morphologically distinctive carnivore morph, (d) which specializes on, and is induced by, anostracan fairy shrimp. (e) In allopatric populations, each species produces similar, intermediate frequencies of both morphs. However, in sympatry, *S. multiplicata* shift to producing mostly omnivores, whereas *S. bombifrons* shift to producing mostly carnivores; i.e., these two species have undergone character displacement in morph production. (f) Similar niche shifts can be experimentally recreated in the lab. When

allopatric individuals are reared with a single conspecific and fed both shrimp and detritus, they produced similar proportions of both morphs (as in natural allopatric populations; see panel e). By contrast, when the two species are experimentally combined, *S. multiplicata* shift to producing mostly omnivores, whereas *S. bombifrons* shift to producing mostly carnivores (as in natural sympatric populations). These facultative niche shifts appear to reflect differences between species in ability to capture and consume shrimp, an environmental cue that induces carnivores.

Figure 2.

How character displacement might evolve from an initial phase in which trait divergence is environmentally induced to one in which divergence is based on genetically canalized differences and thereby becomes expressed constitutively. (a) Initially, two interacting species may express plasticity in resource-use or reproductive phenotypes. In this case, two species of plants facultatively produce different-sized leaves as an adaptive response to different light levels. (b) When they come into sympatry and compete (e.g., for photons), each species may begin to facultatively express a different subset of the initial phenotypes. Here, Species 1 overtops Species 2, thereby gaining increased access to photons, which triggers the facultative production in Species 1 of small leaves only. By contrast, because it is shaded by Species 1, Species 2 facultatively produces larger leaves only (in each case, the expressed phenotype—and underlying genes—are shown in black, whereas the unexpressed ones are shown in gray). If individuals within the same population harbor genetic variation in the degree to which they respond to environmental cues, and if these reaction norm evolve in response to competitively mediated selection and thereby minimize competition between species, then such evolved environmentally induced shifts would constitute character displacement. (c) Over time, both species may lose this pre-existing plasticity and become fixed for a different alternative phenotype, possibly because of the loss (through selection or chance) of alleles or gene combinations underlying the non-expressed phenotype. Thus, character displacement might proceed through an initial phase in which

trait divergence is environmentally induced to a later phase in which divergence becomes genetically canalized. Essentially, during character displacement, each species may evolve from expressing a wide range of phenotypes to becoming genetically canalized for a narrower range of phenotypes (in this case, as each species evolves from being polymorphic for leaf shape to being monomorphic).