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THE BALDWIN EFFECT AND GENETIC ASSIMILATION: REVISITING TWO MECHANISMS OF EVOLUTIONARY CHANGE MEDIATED BY PHENOTYPIC PLASTICITY

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Two different, but related, evolutionary theories pertaining to phenotypic plasticity were proposed by James Mark Baldwin and Conrad Hal Waddington. Unfortunately, these theories are often confused with one another. Baldwin's notion of organic selection posits that plasticity influences whether an individual will survive in a new environment, thus dictating the course of future evolution. Heritable variations can then be selected upon to direct phenotypic evolution (i.e., "orthoplasy"). The combination of these two processes (organic selection and orthoplasy) is now commonly referred to as the "Baldwin effect." Alternately, Waddington's genetic assimilation is a process whereby an environmentally induced phenotype, or "acquired character," becomes canalized through selection acting upon the developmental system. Genetic accommodation is a modern term used to describe the process of heritable changes that occur in response to a novel induction. Genetic accommodation is a key component of the Baldwin effect, and genetic assimilation is a type of genetic accommodation. I here define both the Baldwin effect and genetic assimilation in terms of genetic accommodation, describe cases in which either should occur in nature, and propose that each could play a role in evolutionary diversification.

KEY WORDS: Adaptive divergence, adaptive radiation, canalization, developmental plasticity, evolutionary diversification, genetic accommodation, phenotypic accommodation.

Genes and environment are inextricably linked in the production of phenotype. Recent attention has been paid to the potential importance of developmental or phenotypic plasticity in promoting variability and evolutionary diversification (e.g., West-Eberhard 1989, 2003, 2005; Pigliucci and Murren 2003; Price et al. 2003; Schlichting 2004; Pigliucci et al. 2006; Ghalambor et al. 2007). The extent to which plasticity promotes evolution however, is still wrapped in contention (West-Eberhard 2003; de Jong 2005; Pigliucci et al. 2006). Furthermore, the concepts of the "Baldwin effect," proposed by James Mark Baldwin (Baldwin 1896, 1902; Simpson 1953; Robinson and Dukas 1999), and "genetic assimilation," proposed by Conrad Hal Waddington (Waddington 1953a,b,c,d, 1957, 1961) are often central to debates on plasticity and evolution; yet their definitions are often misconstrued, and they are often confused with one another (reviewed in West-Eberhard 2003). New terms, "phenotypic accommodation" and "genetic accommodation" (West-Eberhard 2003, 2005), have been introduced, but may lead to further confusion.

I here provide a detailed review of Baldwin's and Waddington's original work, and synthesize historical concepts within the framework of modern research. My main objective is to provide clear, definitive interpretations of the meanings of the Baldwin effect and genetic assimilation, so that these terms can be used to effectively convey ideas in evolutionary biology. Although

similar theories were postulated by Morgan (1896), Osborn (1896), Gause (1947), and Schmalhausen (1949), I here focus on the works of Baldwin and Waddington because these have been the most highly cited (e.g., see West-Eberhard 2003). Furthermore, I define the term "genetic accommodation" (West-Eberhard 2003) as it relates to both the Baldwin effect and genetic assimilation, discuss when each of these processes should occur in nature, and highlight instances in which plasticity should increase or decrease adaptive evolution.

Baldwin, Phenotypic Accommodation, and Genetic Accommodation

AN INTRODUCTION TO THE BALDWIN EFFECT

James Mark Baldwin (1861–1934) was an American psychologist who devised two concepts, "organic selection" and "orthoplasy," now commonly known as the "Baldwin effect" (term coined by Simpson 1953). Baldwin's theory is based on the principle that individuals are plastic and able to adapt to their environment within a generation. This plasticity dictates which individuals will survive and produce offspring and thus dictates the course of evolution. By no means, however, did he believe that acquired phenotypes could be inherited. On the contrary, he believed that natural selection acts on "variations in the direction of the plasticity. . ." (Baldwin 1902, p. 37). He thus proposed that plasticity would be a positive driving force of evolution, setting the stage for further neo-Darwinian evolution by increasing the survival of those who display a plastic response. Over time, standing genetic variation can be selected upon so that evolution can proceed in the direction of the induced plastic response. He referred to the ability of plasticity to increase survival as "organic selection," and the directional influence of organic selection on evolution as "orthoplasy" (Baldwin 1896, 1902). Because Baldwin was a psychologist, he focused heavily on the role of plasticity in behavior and learning, that is labile traits, yet also recognized that plasticity occurs in other aspects of the phenotype as well. His theories can be broadly applied, and can include developmentally plastic, nonlabile traits.

PHENOTYPIC ACCOMMODATION

Baldwin often spoke of "accommodation" in reference to nonheritable phenotypic changes that occur in response to the environment and increase the survival of the organism in the particular environment in which the phenotype is induced (Baldwin 1896, 1902). The term "accommodation" is still used for similar effects today, although we now divide accommodation into genetic and phenotypic components (West-Eberhard 2003, 2005; Braendle and Flatt 2006). "Phenotypic accommodation" (West-Eberhard 2003, 2005) is the modern-day equivalent of Baldwin's "accommodation," except that the modern term incorporates responses to both genetic innovations and environmentally induced changes, whereas Baldwin used the term to only refer to the latter. "Genetic accommodation" (West-Eberhard 2003) is a similar concept related to adaptive genetic changes and will be discussed below. West-Eberhard (2005) defines phenotypic accommodation as "adaptive mutual adjustment, without genetic change, among variable aspects of the phenotype, following a novel or unusual input [genetic or environmental] during development." Another difference between Baldwin's and West-Eberhard's definitions is that Baldwin's may also include labile (i.e., nondevelopmental) plastic changes. An example of phenotypic accommodation is the "two-legged goat effect," detailed in West-Eberhard (2003, 2005). A mutant goat was born in the early 1900s, without functional forelimbs (Slijper 1942a,b; West-Eberhard 2003, 2005). We can here think of the absence of functional forelimbs as an environment to which the goat must subsequently respond. Through development, this goat was able to adopt bipedal locomotion via enlargement of the hind legs and changes to the spine and pelvis (i.e., a plastic response). Similar phenomena were observed in baboons (*Papio ursinus*; reviewed by West-Eberhard 2003) and Japanese macaques (*Macaca fuscata*; Hirasaki et al. 2004).

GENETIC ACCOMMODATION

Baldwin noted that heritable variation can occur in the same direction as the plastic response (which he termed "coincident variations"), and thus phenotypes that are originally environmentally induced can be selected upon and inherited (Baldwin 1902). Currently, this phenomenon is considered a type of "genetic accommodation" and empirical examples have been documented (see examples below). In her definition of genetic accommodation, West-Eberhard (2003, p. 142) includes "gene-frequency change due to selection on the regulation, form, or side effects of a novel trait." Although Baldwin emphasized that evolution would occur in response to an environmentally induced novel trait, "genetic accommodation" is now commonly used to refer to evolution in response to both genetically based and environmentally induced novel traits (Table 1).

Another type of genetic accommodation is "genetic compensation" (Grether 2005). Here, the environmentally induced phenotype is maladaptive, and selection favors genetic variation that occurs in a different direction than the plastic response (i.e., countergradient variation; Conover and Schultz 1995), so that a genetic change *compensates* for the plastic change. The end result may be an increase or decrease in plasticity, or no change in the level of plasticity. Because maladaptive plasticity was not considered in Baldwin's theory, I will not discuss genetic compensation in detail.

Baldwin maintained that both neo-Darwinism and neo-Lamarckism were inherently deficient in explaining evolution. His

Table 1. Mechanisms of evolutionary change mediated by phenotypic plasticity, and characteristics inherent to each mechanism.

Term	Basis for trait inducing the evolutionary response	Increase or decrease in the level of plasticity	Mean phenotypic value in the inducing environment
Baldwin effect	environmental	neither or increase[1]	changes
genetic assimilation	environmental	decrease	stays the same
genetic accommodation	environmental or genetic	neither or either	changes or stays the same[2]

[1] In certain cases, could also include a decrease in plasticity (see text).
[2] Inducing environment could include either the extrinsic environment or the genetic environment.

two criticisms of neo-Darwinism were (1) small genetic variations in the "lines of progress" were not substantial enough to adapt an organism to its environmental conditions, and (2) when structure and function of complex traits were only partially correlated, as in the early evolution of these traits, their utility would be of little advantage (Baldwin 1902). Baldwin posited that environmental induction, or accommodation, could enhance such variation or correlations. He did note, however, that neo-Darwinian natural selection is a requirement for evolution to occur after a plastic response to a novel environmental stimulus (Baldwin 1902). He criticized neo-Lamarckism more severely, stating that little or no evidence for the inheritance of acquired characters exists, and that the theory is purely speculative.

Examples of Baldwin's organic selection can be observed in nature. Yeh and Price (2004) examined dark-eyed juncos (*Junco hyemalis*) from California. In the early 1980s, individuals from an ancestral mountain population colonized and established a coastal population. The coastal environment is milder, with less seasonal variation. Yeh and Price found that the newly colonized coastal population had a breeding season (a highly plastic trait) that was nearly twice as long, and noted that this increase in breeding time was adaptive. They speculate that plasticity facilitated colonization and establishment of the new population. An example of both organic selection and orthoplasy comes from Arctic charr (*Salvelinus alpinus*) populations from a Scottish lake (Adams and Huntingford 2004). Three different morphs occur sympatrically: a benthivorous morph that feeds on macro-invertebrates, a planktivorous morph, and a piscivorous morph. The head anatomy of these morphs differs to reflect their preferred prey type. Juveniles of the benthivorous and planktivorous morphs were raised in the laboratory in a common-garden environment, and measurements of head size and shape were made on both wild-caught adults and mature laboratory-raised fish. Both genetic and environmental components of morph divergence were observed, suggesting that phenotypic plasticity may have permitted diversification of morphs into their respective niches, hence setting the stage for further genetic diversification.

We can suppose that Baldwin's orthoplasy would be favored if plasticity is limited. In certain cases, a plastic response to a new environment may be adaptive, yet a more extreme phenotypic value would be required to attain maximum fitness. We can imagine a fitness landscape in which an environmentally induced trait pushes an individual up a new fitness peak, but does not allow it to reach the fitness maxima (figure 1 in Price et al. 2003; figure 2 in Ghalambor et al. 2007). In this scenario, positive selection would occur on heritable variation in the direction of the plastic response. Baldwin makes this apparent when he writes,

> [m]any functions may be passably performed through accommodation, supplementing congenital [heritable] characters, which would be better performed were the congenital characters strengthened [i.e., if further phenotypic change occurred]. Congenital variation would in these cases by seizing upon this additional utility [plasticity], carry evolution on farther than it had gone before [i.e., result in heritable changes in form]. . . this would give the gradual shifting of the congenital mean toward the full endowment [phenotypic optimum]. . . (Baldwin 1902, pp. 209–210)

Other such limits identified by DeWitt et al. (1998) include unreliable environmental cues and lag time between sensing an environmental cue and production of the appropriate phenotype. An empirical example of shifts in the mean phenotypic value without a change in the level of plasticity is documented in a cichlid fish (*Pseudocrenilabrus multicolor*) from low-oxygen swamps and well-oxygenated rivers and lakes in Uganda. Chapman et al. (2000, L. Chapman et al., unpubl. ms.) found that aspects of gill size were greater in swamp fish than in lake fish, and similar patterns were observed when fish were raised under high or low oxygen treatments in the laboratory. Further analyses revealed that levels of plasticity were not significantly different among populations, although populations differed in their mean gill size when raised under common conditions (L. Chapman et al., unpubl. ms.). These results indicate that since the time of population divergence, genetic accommodation has resulted in mean differences in gill size among populations, even though the ability of the gills to respond to environmental cues has remained constant. Similar results were found by Van Buskirk and Arioli (2005) in tadpoles (*Rana temporaria*) raised with or without predators. Tadpoles exhibited plasticity in predator avoidance behavior, as well

as behavioral differences among populations, yet behavioral plasticity did not differ among populations. These results suggest that phenotypic values across environments can evolve independently of the level of plasticity.

Baldwin (1902, pp. 36–37) also proposed that plasticity itself could be adaptive and selected for, thus increasing plasticity in a population. Recent interest in this theory has resulted in empirical support confirming Baldwin's intuition (e.g., Gianoli and González-Teuber 2005; Nussey et al. 2005; Richter-Boix et al. 2006). It is intuitive that plasticity should often increase under selection, if the most plastic individuals are the most capable of colonizing a novel environment or persisting in a fluctuating environment. In these instances, the individuals with the highest levels of plasticity would be under positive selection. Via (1993a,b) argues that plasticity itself would not be selected upon, but rather subject to indirect selection via selection on the most extreme trait values representing the upper limits of the plastic response. Scheiner and Lyman (1989) and Schlichting and Pigliucci (1993) on the other hand, propose that selection can occur directly on plasticity, if plasticity increases the matching between the environmental conditions and the corresponding optimal phenotype. Although these two views of selection on plasticity differ, the outcome of both cases is identical: plasticity will increase due to selection, regardless of whether selection acts on the level of plasticity or on the induced traits/trait values. Indeed, Richard Wolterek (1909), inventor of the term "reaktionsnorm" (i.e., reaction norm), proposed that the reaction norm, rather than the trait value, is inherited because nearly all traits are plastic under at least some environmental conditions (reviewed by Sarkar 2004).

Several empirical examples indicate that plasticity increases after selection under new environmental conditions. For example, Parsons and Robinson (2006) found that the body shape of pumpkinseed sunfish (*Lepomis gibbosus*) was more plastic in the derived open-water ecomorph than in the ancestral inshore ecomorph, even though the habitat of the latter is more heterogeneous. Similarly, Aubret et al. (2004) found that the head and jaw length of tiger snakes (*Notechis scutatus*) were plastic in island populations that feed on large prey, but were not plastic in mainland populations that feed on small prey. Presumably, island populations were colonized from the mainland, and plasticity allowed snakes to exploit the new prey source. Even Waddington (1959) found that plasticity can increase after episodes of directional selection (discussed below).

In summary, the Baldwin effect allows for adaptive evolution through adaptive phenotypic plasticity, which improves fitness, thus dictating the course of future neo-Darwinian evolution. West-Eberhard (2003, p. 153) states that the Baldwin effect is a type of genetic accommodation in which only changes in the regulation of a trait (i.e., decreased genetic control, or rather increased plasticity) occur. However, Baldwin clearly indicated that not only the regulation, but also the *form* of a trait could change as an evolutionary response to a new environmental stimulus (see above). Genetic assimilation is another type of genetic accommodation also relying upon environmental induction, but this mechanism relies only on evolutionary change in the *regulation* of a novel trait and on its frequency of occurrence in a population (Hall 2001; West-Eberhard 2003, 2005).

Waddington, Genetic Assimilation, and Canalization

AN INTRODUCTION TO WADDINGTON'S THEORY

Conrad Hal Waddington (1905–1975) proposed that development would evolve to become canalized against environmental perturbations, via selection acting on the developmental system, a process he referred to as "genetic assimilation" (1953a,b,c,d, 1957, 1961). Specifically, he defined genetic assimilation as a process "by which a phenotypic character, which initially is produced only in response to some environmental influence, becomes, through a process of selection, taken over by the genotype, so that it is formed even in the absence of the environmental influence which had at first been necessary" (Waddington 1961). Although this idea may initially seem conceptually similar to the Baldwin effect, there are fundamental differences between the two. Waddington conceptualized an "epigenetic landscape" (Waddington 1953b, 1957), where the rolling of a ball across a landscape symbolized development through time. Environmental perturbations could push the ball from one developmental pathway to another, and genetic assimilation acted as an evolutionary process to heighten the ridges of the landscape, so that, over time, increasingly greater perturbations were needed to shift the ball from one developmental trajectory to another (reviewed by Rollo 1994, West-Eberhard 2003). His theory posits that the environment induces phenotypes that are adaptive, and then selection on the developmental system acts to reduce responsiveness to the environment (i.e., reduce plasticity), so that the induced phenotype becomes "inherited" (i.e., canalized) after a number of generations of exposure to the environmental stimulus (Waddington 1942, 1953b,c, 1957, 1961). A similar idea was independently proposed by Schmalhausen (1949), who used the term "stabilizing selection" to refer to selection acting against both mutations and environmentally induced change, which push the phenotype from the local optima. A primary difference between the theories is that Waddington proposed that environmental perturbations would initially be adaptive, whereas Schmalhausen proposed that they would be predominantly (but not always) maladaptive. Schlichting and Pigliucci (1998) also note the striking similarities between Schmalhausen's and Waddington's theories, but highlight that Waddington was more radical in that he argued for revision of the Modern Synthesis (p. 43). West-Eberhard (2005) notes that

genetic assimilation is a particular case of genetic accommodation in which the frequency of a trait in the population is altered due to evolutionary change in the threshold of expression for that particular trait.

Waddington's motive for hypothesizing an environmentally triggered mechanism of evolution was similar to that of Baldwin's, in that neither the neo-Lamarckian nor the neo-Darwinian mechanisms seemed sufficient in explaining adaptive evolution. On the Lamarckian mechanism of the heritability of acquired characters, he wrote, "it has been so widely rejected by. . . the scientific world that it is hardly considered to be worthy of discussion in most of the important recent works on evolution" (Waddington 1953c). Although Waddington did not completely denounce neo-Darwinism, he highlighted the deficiencies in the theory, namely that random mutation is unlikely to produce all the variants selected upon in nature, and that the importance of environmental induction in evolution should not be ignored (Waddington 1942, 1953b,c, 1961). Waddington criticized Baldwin's theory, stating that the accumulation of genetic mutations influencing an induced trait is unlikely (Waddington 1953a, 1961). Yet, both theories rest on the assumption that natural selection acts upon favorable mutations, and are thus both fully compatible with, and in fact rely upon, neo-Darwinian evolution.

WADDINGTON'S EXPERIMENTS

Waddington noted that strains of *Drosophila* differed in their capacity to produce mutant phenotypes after environmental perturbations (Waddington 1953d). He heat-shocked the larvae of one strain and observed that although a proportion developed into adults with "cross-veinless" wings, that is disruption of the posterior cross-vein on the wing, none of the non-heat-shocked larvae developed this phenotype (Waddington 1952a, 1953b,d, 1957). He then established two artificially selected lines, selecting flies with the cross-veinless phenotype for one line, and flies with the normal wing phenotype for the other line. He continued heat-shocking larvae and performed selection for 23 generations, and examined the flies that developed from a few non-heat-shocked larvae at each generation. From generation 14 onward, he observed that the cross-veinless phenotype was present in a few non-heat-shocked individuals, indicating that the inducing environment (i.e., the heat-shock) was no longer necessary to produce the induced phenotype (i.e., cross-veinless), and the frequency of these individuals tended to increase over successive generations. He termed this the "genetic assimilation" of an "acquired" character; "acquired" because it was initially induced by the environment, and "genetic assimilation" because of increased genetic control of the phenotype (Waddington 1952b, 1953b,d).

Modern research has discovered that heat-shock chaperone proteins (Hsp) act as buffers against environmental shock. When Hsp90 function is compromised in *Drosophila* and *Arabidopsis*, due to mutations or following the application of inhibitory drugs, mutant phenotypes are manifest. Following several generations of artificial selection of mutant phenotypes, they may persist in the lineage even after Hsp90 function is restored (Rutherford and Lindquist 1998; Queitsch et al. 2002; Wong and Houry 2006). As noted by Waddington (1953d), different strains tend to produce different phenotypic mutants, indicating an underlying genetic basis for the development of environmentally induced phenotypic variation, the expression of which is buffered by Hsp90 (Rutherford and Lindquist 1998; Queitsch et al. 2002). Results have indicated that epigenes are involved in the inheritance of these environmentally induced mutant phenotypes, including those involved in chromatin remodeling (*Drosophila melanogaster*, Sollars et al. 2003; *Saccharomyces cerevisiae*, Zhao et al. 2005; Wong and Houry 2006). It is thus plausible that the production and inheritance of the cross-veinless phenotype in Waddington's experiment (1953d) were due to compromised Hsp90 functioning in his stock flies, perhaps accompanied by the acquisition of heritable epigenes after heat shock. In this sense, we can imagine that the Hsp90 acts as a canalizing factor, inhibiting environmental perturbations by the environment. Positive or negative selection could act on chaperone proteins, depending on the adaptive advantage or disadvantage of underlying cryptic genetic variation. Modern research on heat-shock proteins thus provides empirical evidence for a mechanism of genetic assimilation.

In another of Waddington's experiments (Waddington 1956), he subjected fly larvae to ether vapor and noted that some produced a "bithorax-like" phenotype, in which the meta-thoracic disc gives rise to structures that normally characterize the mesothorax. He created two artificially selected lines, one in which flies showed the bithorax phenotype and one using wild-type flies. As in the heat-shock experiment above, at each generation he raised flies from the bithorax line without exposure to ether to see if the abnormal phenotype had been "assimilated." After generation 29, flies were produced with meta-thoracic bud material that is characteristic of the thorax (i.e., the bithorax phenotype). He mated flies exhibiting this phenotype to produce lines with a high frequency of the abnormal bithorax phenotype. Gibson and Hogness (1996) have since shown that variation in the bithorax phenotype (exhibited after treatment with ether vapor) is attributed to the polymorphism of the *Ultrabithorax* (*ubx*) regulatory gene, and that the loss of *ubx* expression in the third thoracic imaginal disc may result in increased ether sensitivity. In accordance with Waddington's hypothesis of genetic assimilation, selection may act on *ubx* to reduce polymorphism and thus canalize development within a population (Waddington 1953b, 1957). Modern research thus provides evidence that selection may act on regulatory networks to buffer against environmental perturbation, providing evidence for another mechanism for Waddington's genetic assimilation (reviews: Gibson and Dworkin 2004; Badyaev 2005; Flatt 2005).

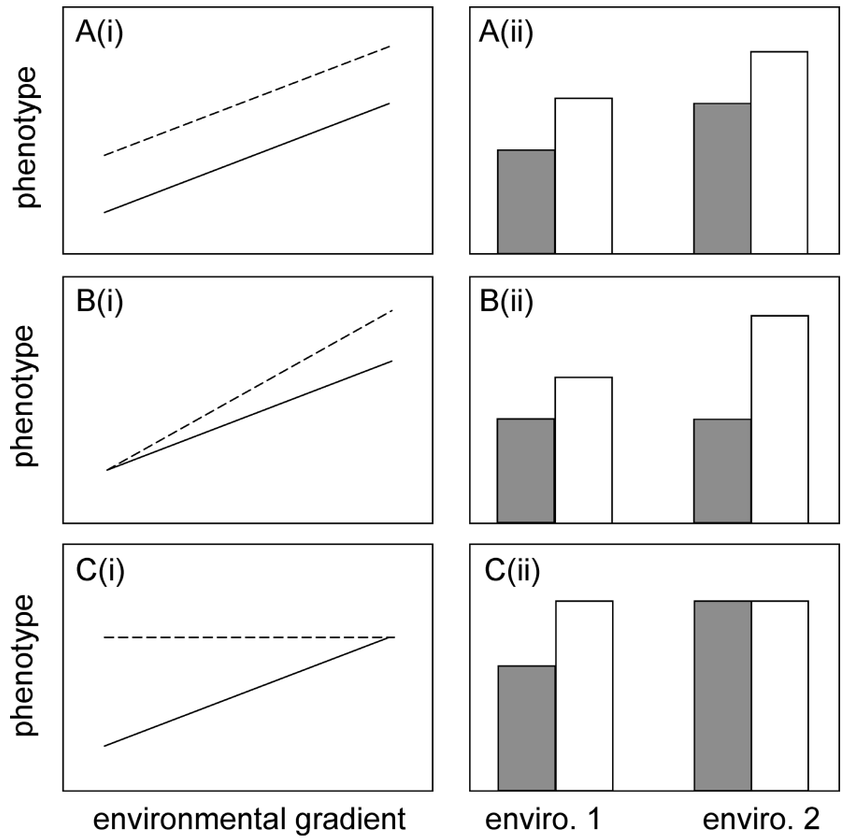

Figure 1. **Relationship between the phenotype and the environment (i.e., plasticity) before (solid lines, gray bars) and after (dashed lines, white bars) genetic accommodation. A and B represent the Baldwin effect and C represents genetic assimilation. (i) Reaction norms for continuous phenotypic variation along an environmental gradient, and (ii) polyphenisms in discrete environments (the height of the bars represent either the magnitude of the induced phenotype, or the frequency of the polyphenism in the population).**

Disentangling the Baldwin Effect and Genetic Assimilation

In modern studies, how can we disentangle the Baldwin effect from genetic assimilation? In both cases, traits would initially be plastic, and neo-Darwinian selection would act upon the reaction norm. The end result of selection however, differs between the two processes. In the Baldwin effect, selection may act to change mean trait values without changing the level of plasticity in the population (Fig. 1A; Table 1), or alternately, selection acting on the phenotype can result in increases in the level of plasticity (because the most plastic individuals possess the most extreme phenotype and are thus positively selected; Fig. 1B; Table 1). The key to the Baldwin effect is that the initial plasticity allows for the survival in a novel environment, and neo-Darwinian evolution can proceed from there. Genetic assimilation, conversely, should act to decrease plasticity (i.e., increase canalization) in a population within a given range of environmental conditions (Fig. 1C; Table 1), if an increase in canalization is adaptive. Both the Baldwin effect and genetic assimilation may occur simultaneously for a trait within a population (Fig. 2).

Another of Waddington's experiments on "genetic assimilation of acquired characters" (Waddington 1959) illustrates why the Baldwin effect may easily be confused with genetic assimilation. Waddington raised larvae of three strains of *D. melanogaster* in a salt medium that resulted in at least 60% mortality. He raised flies for 21 generations and increased the salt concentration each generation to maintain a constant mortality rate. After 21 generations, he raised larvae from the selected lines and from the original stock flies on media with differing levels of salt. He noted that although the unselected stock tended to have higher survival at lower salt concentrations, the selected lines had higher survival at higher salt concentrations. In all cases, the survivors in the selected lines had greater areas of the anal papillae (involved in osmoregulation), and anal papillae area increased with increasing salt concentration. He speculated that the increased area of the anal papillae in the selected lines is evidence for genetic assimilation, but this is clearly a contradiction of his own definition. He defined genetic

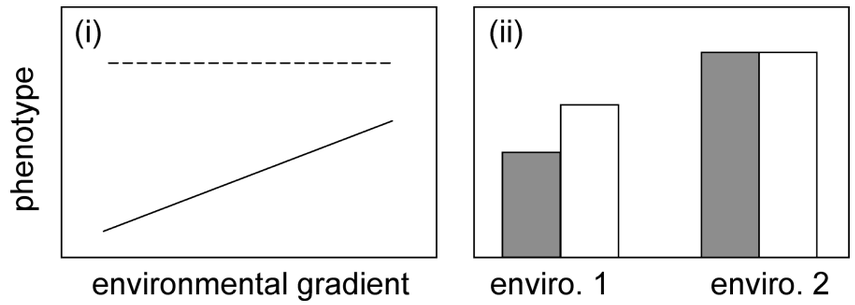

Figure 2. **Both the Baldwin effect and genetic assimilation can occur simultaneously for a trait in a population. See Figure 1 for legend.**

assimilation as a process leading to "canalization," or rather increased genetic control of a phenotype. In this case, however, the phenotype that was manifest in higher salt concentrations is clearly not canalized in the selected lines, as evidenced by the reaction norm. Indeed, it appears that the salt-selected larvae had an even steeper slope of the reaction norm, indicating that plasticity had *increased* (Waddington 1959, 1961). Could the individuals with the largest anal papillae have been the ones to survive, thus providing variation for large papillae in subsequent generations? Perhaps the most plastic individuals were the ones capable of survival, and thus Waddington inadvertently selected for plasticity. Waddington's study (1959) therefore does not conclusively demonstrate genetic assimilation, but rather appears to demonstrate the Baldwin effect. Considering these conclusions, it is understandable that modern-day confusion occurs surrounding the terms "genetic assimilation" and the "Baldwin effect." I propose that the best way to deal with this issue is to assume that Waddington misinterpreted his results, and to adhere to his strict definition of genetic assimilation, that is, increased genetic control of a trait that was once environmentally induced.

EVOLUTIONARY CHANGE IN PLASTICITY

Although Baldwin's theory revolves primarily around the evolution of trait means (i.e., the *elevation* of reaction norms), Waddington's theory is based on the assumption that plasticity itself (i.e., the *slopes* of reaction norms) will evolve. To document evolvability of a slope of a reaction norm, a genotype-by-environment (G × E) interaction must be present (Via and Lande 1985; Scheiner and Lyman 1989; Scheiner 1993). Several empirical studies have documented G × E interactions, indicating the potential for plasticity to evolve (e.g., papers reviewed by Windig et al. 2004; Danielson-François et al. 2006; Gutteling et al. 2007). Using a different approach, Nussey et al. (2005) documented significant heritability in the slope of the reaction norm for breeding date in relation to spring temperature in great tits (*Parus major*), indicating that plasticity in breeding date can evolve in these birds.

Both the Baldwin effect and genetic assimilation could apply to different traits within a lineage, or even to a specific trait in different lineages. Indeed, Suzuki and Nijhout (2006) found evidence for genetic accommodation in two selected lineages of tobacco hornworms (*Manduca sexta*), one in which the plastic response decreased (genetic assimilation), and the other in which the plastic response increased after selection. *M. sexta* consist of two color morphs exhibiting either green (wild-type) or black (mutant) larvae. Heat-shocking the mutant larvae results in the production of a variety of different color morphs, ranging from pure green to pure black. Suzuki and Nijhout created two selected lines from the heat-shocked black mutant strain, one line selected for green and the other for black larval coloration. They also established an unselected control line, in which larvae were heat-shocked but were not subjected to artificial selection. Reaction norms of larval coloration were observed after 13 generations of selection, using developmental temperatures ranging from 20°C to 42°C. The reaction norms of the control and green-selected lines followed sigmoidal curves (see figure in Suzuki and Nijhout 2006). The control line consisted of mostly black phenotypes, but the green coloration increased at an inflection point around 32.7°C. Whereas the green-selected line was mostly black at low temperatures, at high temperatures, it was completely green, with an inflection point around 28.5°C; that is, both the slope and elevation of the reaction norm *increased* relative to the control line (figure 2B in Suzuki and Nijhout 2006). The black-selected line remained black at all temperatures; that is, both the slope and elevation of the reaction norm *decreased* relative to the control line (figure 2B in Suzuki and Nijhout 2006). What do these results tell us in terms of the Baldwin effect and genetic assimilation? First, the environmental switch point, the temperature at which green phenotypes are manifest, is lower, and the elevation of the reaction norm is higher in the green-selected line than in the control line. This is evidence for the Baldwin effect (i.e., plasticity allows for survival via artificial selection, followed by heritable change in the reaction norm). Second, in the black-selected line, green coloration could no longer be induced after 13 generations, even with temperatures as high as 42°C. This is evidence for genetic assimilation, that is, canalization of development of larval coloration. Suzuki and Nijhout (2006) thus successfully

demonstrated that both the Baldwin effect and genetic assimilation can occur for the same trait in different lineages.

Extricating the Baldwin effect from genetic assimilation may prove difficult, perhaps one reason why their definitions are easily confused. One example that demonstrates this difficulty is the monophyletic group of four genera of spadefoot toads and parsley frogs (Gomez-Mestre and Buchholz 2006). The tadpoles of Old World genera, *Pelobates* and *Pelodytes*, live in long-lasting or permanent ponds, whereas tadpoles of North American genera, *Spea* and *Scaphiopus*, live in shorter-lived ephemeral ponds. The former have longer larval periods than the latter. Reaction norms for larval period in relation to the duration of the larval environment showed no overlap in larval period between Old World and North American genera. In addition, the North American genera had shallower slopes, indicating a lesser degree of plasticity. Phylogenetic analysis revealed that the longer and more plastic larval period is in the ancestral state, thus shorter and less plastic larval periods must have evolved after colonization of North American ponds. This indicates that plasticity in larval period may have allowed for colonization of North American ponds (i.e., Baldwin's "organic selection"), followed by genetic changes that shifted the elevation of the reaction norms (i.e., Baldwin's "orthoplasy"). At first glance, it would appear that genetic assimilation might be occurring because the slope of the reaction norm has decreased in the most recent lineages relative to the ancestral lineages. However, it remains possible that decreased plasticity in larval period is not adaptive, but rather occurs due to constraints imposed by a minimum larval period required for normal development.

CONDITIONS FAVORING THE BALDWIN EFFECT AND GENETIC ASSIMILATION

Under which natural conditions would each process, the Baldwin effect and genetic assimilation, be favored? The Baldwin effect should be favored if plasticity is beneficial, and in situations in which maintenance of the ability to respond to environmental cues is not costly (see DeWitt et al. 1998). Plasticity may be beneficial to organisms in heterogeneous environments (e.g., Day et al. 1994; Gianoli and González-Teuber 2005; Richter-Boix et al. 2006), or it may be beneficial in lineages with high gene flow among populations in divergent environments (e.g., Sultan and Spencer 2002). An increase in plasticity, or shift in trait means without changes in plasticity, would be favored if limits to plasticity occurred following an adaptive plastic response to a novel environmental condition. That is, if an environmental change induces a new peak on a fitness landscape, the plastic response may push the population up the new peak, but to reach the fitness maxima further phenotypic changes, via genetic accommodation, would be necessary (Price et al. 2003; Ghalambor et al. 2007).

Genetic assimilation, on the other hand, would tend to occur when plasticity is costly to maintain or if it is maladaptive. Several costs to plasticity have been identified, including, but not limited to (1) energetic costs associated with the maintenance of sensory and regulatory mechanisms, (2) costs associated with the production of the phenotype, for example trade-offs, (3) developmental instability, and (4) genetic costs, for example linkage with disadvantageous genes, or disadvantageous pleiotropy or epistasis (DeWitt et al. 1998). Relyea (2002) examined potential fitness costs of morphological plasticity in wood frog (*Rana sylvatica*) tadpoles in response to predatory insects. In the presence of the predator, increased plasticity in several aspects of body shape had negative effects on growth and development, but survival increased with increasing plasticity in body depth. In the absence of the predator, however, survival tended to *decrease* with increasing plasticity in muscle depth and width. Thus, in stable environments, selection may act to canalize development of body and muscle shape in tadpoles. Steinger et al. (2003) showed that in the plant *Sinapis arvensis*, plants that had greater plasticity in leaf area in response to light levels produced fewer seeds. In the snail *Physa heterostropha* (DeWitt 1998), growth rate was negatively correlated with plasticity in shell shape in response to fish predation. Thus fitness costs to plasticity do occur in nature, and genetic assimilation could be favored if plasticity is not beneficial. For example, Buckley et al. (2007) examined growth in western fence lizards (*Sceloporus occidentalis*), the eggs of which had been incubated under two thermal treatments (24°C and 30°C). Hatchlings were then maintained at common temperatures, and aspects of growth were monitored for seven weeks posthatching. At hatching, hind-limb length, inter-limb length, and body mass differed between the treatments, but by week seven no significant differences were observed between the groups. This may be an example of genetic assimilation that has occurred to canalize development following environmentally induced changes that occurred during the incubation period.

THE BALDWIN EFFECT, GENETIC ASSIMILATION, AND EVOLUTIONARY DIVERSIFICATION

Recently, attention has been paid to whether the Baldwin effect, genetic assimilation, and phenotypic plasticity in general can promote evolutionary diversification in nature. Price et al. (2003) modeled how phenotypic plasticity can allow for diversification by allowing for individuals to move among peaks on a fitness landscape. Pigliucci and Murren (2003) and Pigliucci et al. (2006) propose that plasticity could allow expansion into novel environments (i.e., Baldwin's "organic selection"), and that genetic assimilation could then result in adaptive genetic divergence among populations (see Fig. 2); although Baldwin's orthoplasy could ultimately have a similar effect. de Jong's (2005) quantitative genetic model showed no evidence that phenotypic plasticity and genetic assimilation could provide a means for ecotype divergence. Yet Pigliucci et al. (2006) criticize de Jong on several accounts: (1)

she fails to consider that plasticity can facilitate survival in novel environments, (2) her model is limited because it focuses only on adaptive plasticity, cannot infer causal mechanisms or predict future outcomes, and does not take into account limits to plasticity that would warrant further evolution, (3) she ignores evidence for genetic assimilation and the ability of plasticity to positively influence diversification in empirical studies (see examples above), (4) she views the hypothesis that plasticity influences evolutionary diversification as a threat to the Modern Synthesis, but Pigliucci et al. (2006) note that this hypothesis is compatible with the Synthesis (note above, that neo-Darwinian evolution is a key component to the Baldwin effect and genetic assimilation). Genetic assimilation could also allow for the accumulation of cryptic genetic variation, which could later become expressed after removal of the canalizing factor, or "de-canalization," thus promoting evolvability (Rutherford and Lindquist 1998, Gibson and Dworkin 2004; Flatt 2005). Theoretical predictions and empirical evidence thus point toward the possibility that the Baldwin effect and genetic assimilation could play important roles in the evolution of diversification.

Summary

The Baldwin effect increases the survival of individuals in environments in which they would otherwise be maladapted or have reduced fitness. Through the neo-Darwinian process of genetic accommodation, populations can adapt genetically to these environments, leading to new evolutionary trajectories. The Baldwin effect couples phenotypic accommodation with genetic accommodation to result in adaptive evolution. Genetic assimilation, on the other hand, can either prevent induction of a maladaptive phenotype, or eliminate costs associated with the sensory and regulatory mechanisms needed to elicit a plastic response. The Baldwin effect may tend to increase plasticity or have no effect on the level of plasticity, whereas genetic assimilation decreases plasticity within a range of environmental variables. Whereas the Baldwin effect may promote evolutionary diversification, genetic assimilation has a stabilizing effect in populations, or enhances evolvability by allowing for the accumulation of mutations. It is unclear which mechanism occurs more commonly in nature, and it is conceivable that both mechanisms may act simultaneously in a population, each on different traits, or even on the same trait.

Although the term "genetic assimilation" has been used to refer to both a loss of plasticity (i.e., canalization; Waddington 1953a,b,c,d, 1961) and shifts in the phenotype without decreases in plasticity (Waddington 1959; Chapman et al. 2000; Price et al. 2003), I propose that we restrict the use of the term to refer to adaptive decreases in plasticity (i.e., Waddington's original interpretation of genetic assimilation). A more appropriate alternate term, that is genetic accommodation (as used by West-Eberhard 2003 and Braendle and Flatt 2006), should be used to refer to any type of shift in the reaction norm (shift in the elevation or slope) after exposure to a novel environmental stimulus. Through the refinement of these definitions, we will provide ourselves with more effective tools to convey ideas about the implications of phenotypic plasticity in evolutionary biology.

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