

EVOLUTION

INTERNATIONAL JOURNAL OF ORGANIC EVOLUTION

PUBLISHED BY
THE SOCIETY FOR THE STUDY OF EVOLUTION

Vol. 57

July 2003

No. 7

Evolution, 57(7), 2003, pp. 1455–1464

PERSPECTIVE:

GENETIC ASSIMILATION AND A POSSIBLE EVOLUTIONARY PARADOX: CAN MACROEVOLUTION SOMETIMES BE SO FAST AS TO PASS US BY?

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Abstract.—The idea of genetic assimilation, that environmentally induced phenotypes may become genetically fixed and no longer require the original environmental stimulus, has had varied success through time in evolutionary biology research. Proposed by Waddington in the 1940s, it became an area of active empirical research mostly thanks to the efforts of its inventor and his collaborators. It was then attacked as of minor importance during the “hardening” of the neo-Darwinian synthesis and was relegated to a secondary role for decades. Recently, several papers have appeared, mostly independently of each other, to explore the likelihood of genetic assimilation as a biological phenomenon and its potential importance to our understanding of evolution. In this article we briefly trace the history of the concept and then discuss theoretical models that have newly employed genetic assimilation in a variety of contexts. We propose a typical scenario of evolution of genetic assimilation via an intermediate stage of phenotypic plasticity and present potential examples of the same. We also discuss a conceptual map of current and future lines of research aimed at exploring the actual relevance of genetic assimilation for evolutionary biology.

Key words.—Baldwin effect, evolutionary theory, genetic assimilation, macroevolution, phenotypic plasticity.

Received June 25, 2002. Accepted February 8, 2003.

The concept of “genetic assimilation,” or the “Baldwin effect,” has been a minor but persistent part of evolutionary thinking since Waddington’s classic experiments (Simpson 1953). The concept was first introduced by C. H. Waddington in the 1940s in the context of reconciling apparent cases of Lamarckism with the neo-Darwinian framework. In current database searches (such as the Web of Science) only 22 papers discuss genetic assimilation during the period from 1997 to 2001. However, these papers range in scope from comparative developmental biology to phenotypic plasticity, from quantitative genetic modeling to group selection, and from studies of hybrid zones to research on neural networks. The aim of this review is to briefly examine the basic idea through its historical unfolding, consider selected examples of its application to empirical and theoretical research in evolutionary biology, and assess its vitality as a scientific research program. In particular, we present a conceptual map of how to study genetic assimilation that we hope will be useful to researchers interested in pursuing the subject and, hopefully, to bring Waddington’s ideas again to the forefront of evolutionary discussions.

Historical Background: What Is Genetic Assimilation?

Organisms’ phenotypes can change across environments (phenotypic plasticity) and selection can operate both on the

expression of traits within particular environments and on the shape of the reaction norm itself (Schlichting and Pigliucci 1998). Waddington (1942; 1953) grasped both of these concepts and proposed that selection can act in such a manner as to turn an environmentally stimulated phenotype (i.e., plasticity) into a fixed response to prevalent environmental conditions (assimilation). Something similar to this apparent ‘inheritance of acquired characters’ from an evolutionary point of view was described by Baldwin (1896) as early as the end of the nineteenth century. Waddington (1961, p. 257) explicitly defined genetic assimilation as “a process by which characters which were originally ‘acquired characters’ may become converted, by a process of selection acting for several or many generations on the population concerned, into ‘inherited characters.’” Waddington’s concept of “acquired characters” is clearly equivalent to what we now consider phenotypically plastic traits. As an example of genetic assimilation he used the callosities of the ostrich (related to the crouching position in adult birds), which form while the bird is still an embryo. He wrote, “The callosities were formed as responses to external friction [what we would now call plasticity], but during the course of evolution the environmental stimulus has been superseded by an internal genetic factor” (Waddington 1942, 356).

Waddington was aware of a concept closely intertwined with his ideas on assimilation, Goldschmidt's (1940) notion of "phenocopy." Goldschmidt defined phenocopy as the phenotype produced by an environmental stimulus that may look like the result of a genetic mutation. In fact, Waddington (1952; 1953) followed up on Goldschmidt's phenocopy idea by using experimental data from two selected lines of *Drosophila melanogaster*. This *Drosophila* research yielded groundbreaking data in support of the idea that environmentally induced (i.e., plastic) characters can become fixed in populations (Waddington 1942). This now classic study examined how natural selection could act on phenotypes that are sensitive to an environmental stimulus, which then would result in a corresponding genetic change that canalized the phenotype. Wild-type *Drosophila melanogaster* were given a strong temperature shock (40°C) after puparium formation (21–23 h old), and this temperature shock was applied following each generation of selection. Two selection lines were formed, a crossveinless one and a "normal" line (i.e., selection for the wild-type vein pattern). Crossveinless individuals occurred in approximately 40% of flies of the original stock following temperature shock. After five generations, 60% of the flies in the crossveinless selection line had the crossveinless phenotype, and 35% of the normal selected line had the crossveinless phenotype. By generation 19 of the experiment, the crossveinless selection line had 98% crossveinless phenotype, whereas the normal selection line had only 15% crossveinless phenotype. It is especially important to note that by the 12th generation flies from the crossveinless selection line *without* the temperature treatment produced the crossveinless phenotype. Moreover, in these selected lines, the crossveinless phenotype was maintained even when raised under normal culture conditions.

Waddington (1953) noted that the genetic background had a significant impact on the penetrance of the character, suggesting that this was likely due to the polygenic nature of the crossveinless character. If the same experiment were to be repeated starting with a different population of flies, he proposed that a different genetic background could develop the same phenotype. These ideas were an early suggestion of the modern concept of genetic redundancy (Goldstein and Holsinger 1992; Pickett and Meeks-Wagner 1995; Wagner 1999). Waddington's experiments demonstrated that selection can genetically fix a phenotypic change that was initially triggered by the environment. *Genetic assimilation, then, is a process that turns a plastic response into a genetically invariant one through continued selection for stable expression of the trait under new environmental conditions.*

Baldwin's (1896) classic paper, written in arcane pre-Mendelian language, was a precursor of similar ideas, outlining the importance of development and plasticity in the evolution of phenotypes. Baldwin's (1896, p. 447) definition of his "new factor in evolution" comes close to a description of genetic assimilation: "The most plastic individuals will be preserved to do the advantageous things for which their variations show them to be the most fit, and in the next generation will show emphasis of just this direction in its variation." Waddington (1961) tried, rather unsuccessfully in our opinion, to distinguish his ideas from those of Baldwin, which later came to be termed "the Baldwin Effect."

During the modern synthesis, Huxley (1942) included genetic assimilation (in the form of the Baldwin Effect) as a subsidiary theory, but Simpson (1953) concluded that Baldwin considered "accommodation" (plasticity) to be nonhereditary and suggested that this made the Baldwin effect weak. Simpson may have misunderstood plasticity as it was being studied by some of his contemporaries (Bradshaw 1965; Marshall and Jain 1968). Bradshaw, in particular, explicitly stated that phenotypic responses to different environments may be consistent among genotypes *or* genotypes may have different plasticities (reaction norms of different slopes or changes in ranks across environments) (Sultan 1995). Arguably, Bradshaw's insights may have opened the way to the modern study of phenotypic plasticity (Pigliucci 2001), but this could not have been anticipated by Simpson when he wrote his commentary on the Baldwin effect.

We will begin our examination of the modern status of genetic assimilation by discussing the few existing theoretical investigations into the phenomenon and then describe several compelling empirical examples from the recent literature. In the final section of this paper, we will suggest a number of avenues for further research, which we consider will be fruitful paths into the study of genetic assimilation within the contemporary framework of evolutionary biology.

Theoretical Treatments and Implications of Genetic Assimilation

Williams' (1966) work is a landmark in evolutionary theory largely because of his sophisticated criticism of several ideas (such as group selection) that enjoyed common acceptance after the neo-Darwinian synthesis (Mayr and Provine 1980). Williams' criticism of genetic assimilation, however, may have been a bit premature and has probably contributed to significantly slowing down progress on the theory of genetic assimilation (it is not mentioned in one of the most recent textbooks on quantitative genetics, Roff 1997; and it is given a very brief explanation in another, Falconer and Mackay 1996). Lately, quite a few authors suggested that Williams may have discounted several other ideas too quickly, as in the case of group selection itself (Goodnight 1985; Nunney 1989; Wilson and Dugatkin 1997; Sober and Wilson 1998) or phenotypic plasticity (Schlichting 1986; Sultan 1987; West-Eberhard 1989; Scheiner 1993; Pigliucci 2001). Williams rejected a role for genetic assimilation in evolutionary theory on the grounds that plasticity preceding assimilation of the novel phenotype would have to be adaptive, and therefore the result of natural selection (Eshel and Matessi 1998). This is a rather simplistic assumption because it equates advantageous characteristics (which can be present by chance in a population) with the existence of characters that are the result of the process of adaptation. The two should remain conceptually distinct (Gould and Vrba 1982).

Theoretical work in the area of genetic assimilation has been published over the last couple of decades, despite Williams' critique. Most theoretical treatments of genetic assimilation focus on a small number of key concepts, which we will discuss in some detail. Some models use standard quantitative genetic techniques, several rely on classic population genetic scenarios with few loci and alleles, and most make

use of a combination of analytical techniques and intensive computer simulations to tackle the complexity of the topic.

Perhaps the first modern theoretical treatment of phenotypic evolution that includes genetic assimilation as an important component is the one provided by Kirkpatrick (1982) in the context of his discussion of "quantum evolution" and punctuated equilibria. Adaptive peak shifts have traditionally been regarded as the result of environmental change, a combination of genetic drift and fitness epistasis or macromutations, and the mechanisms allowing such shifts in multidimensional adaptive landscapes are under intense investigation (Gavrilets 1997; Gavrilets et al. 1998). Kirkpatrick maintains that a peak shift can occur as a sudden event resulting from the gradual change in a parameter controlling the distribution of phenotypes in a population, without the contribution of drift. In complexity theory, such a change is referred to as a bifurcation (Kauffman 1993). This sort of modification can be triggered by an alteration in the environment (phenotypic plasticity), or by the developmental or mutational properties of the character(s) under selection. Changes in the latter properties are hypothesized to be the result of genetic assimilation and canalization. Kirkpatrick's major insight is that a fitness valley can disappear from an adaptive landscape as the result of environmental or developmental changes, if these mechanisms affect the phenotypic distribution at the population level and result in an increase of the phenotypic variance. Selection will then have to push the population toward the highest nearby fitness peak. If these ideas are correct, then we have a novel solution to Wright's (1932; 1967) problem of peak shifts. It is incidentally important to note that we consider peak shifts broadly to include any major phenotypic transition a population goes through that significantly alters the relationship between the phenotypic space occupied by the population and its average fitness. Both our conceptualization and Kirkpatrick's can be considered independent of the specific solutions required by the shifting balance theory as defined by Wright, which has been subjected recently to much critical scrutiny (Crow 1991; Wade and Goodnight 1991; Barton 1992; Phillips 1993; Price et al. 1993; Moore and Tonsor 1994; Goodnight 1995; Gavrilets 1996; Coyne et al. 1997; Peck et al. 1998).

Hinton and Nowlan (1987) reached a very similar sets of conclusions in a completely different context. These authors explored the advantage that learning (which in their model is functionally equivalent to phenotypic plasticity), can have when added to an initial process of genetic specialization (see also Schlichting and Pigliucci 1998, ch. 9). Hinton and Nowlan (1987) expressly set out to incorporate the Baldwin effect in their model and found that—as was suggested by Baldwin himself—the "evolutionary search space" can be explored much more efficiently by a combination of processes, such as selection and learning (see also: Behera 1994). Selection by itself would not be sufficient because when the population is close to the adaptive peak the favorite combination of genes can be broken up by recombination, a classical problem in evolutionary genetics. One of the interesting prerequisites for the Baldwin effect to work is that the organism must somehow "recognize" that it is approaching the adaptive peak. This means that evolution will favor the appearance of receptors of environmental signals, most es-

pecially of cues that are reliably correlated with (and possibly anticipating: Schlichting and Pigliucci 1998; Tufto 2000) the environmental factor of interest. Moreover, according to Hinton and Nowlan, the Baldwin effect works best if the adaptive landscape is complex ("rugged" in complexity theory terminology; Kauffman 1993). This is because standard evolution by genetic specialization is sufficient to locate the position of a peak in the adaptive landscape. Learning or plasticity become necessary only for the fine tuning operation of "hill climbing" in a complex landscape with many peaks of similar fitness close to each other.

Gerard et al. (1993) discuss the evolution of phenotypic plasticity and genetic assimilation as alternative outcomes in a simple two locus-two allele model. In this scenario, a wild-type and a novel phenotype are favored in distinct environments, and the bridge between the two is provided by phenotypic plasticity. In general, a directional shift in the environment ends up favoring canalization of the novel phenotype through an intermediate stage of plasticity. In contrast, environmental fluctuations tend to favor plasticity, unless there are genetic constraints limiting the range of phenotypic outcomes.

The relationship between plasticity and genetic assimilation is emphasized also by Behera (1994), who correctly maintains that it is because development is inherently plastic that the whole process is possible. In this sense, as argued by one of us (Pigliucci 2001), we should think of nonplastic genotypes as the derived state, since homeostasis in the face of environmental change requires selection of particular combinations of genes. Phenotypic plasticity is inherent in the fact that biomolecules are intrinsically sensitive to at least some environmental factors, such as temperature or pH. Behera reaches his conclusions on the basis of a simple model that shows that the number of genotypic combinations required to be tested by natural selection is greatly reduced if plasticity is present (under one of the scenarios that he presents plasticity reduces the number of combinations from 2^{18} to 2^9). It is reassuring to note that these results are similar to those emerging from the study by Hinton and Nowlan (1987) mentioned above.

Behera's paper also touches on another component of the problem of evolution by genetic assimilation: the possibility of costs of plasticity (DeWitt et al. 1998; Dorn et al. 2000). This issue is explored in more detail by Mayley (1997) who suggests that costs of plasticity may lead to genetic assimilation even under moderately heterogeneous environmental conditions. Mayley carried out simulations using Kauffman's (1993) N-K models. In this kind of model N is the number of parts constituting a system, in this case the length of the genotype, and K is the number of connections among the parts, here a measure of epistasis. Mayley explored three scenarios using this modeling framework: cost-free learning (or plasticity), low cost, and high cost. In the first case the outcome leads to several genotypes equivalent in fitness because they could produce the target phenotype as a result of plasticity. Because of the absence of costs, genetic assimilation is not favored. Interestingly, plasticity performs best when the number of connections among genes (epistasis) is higher. The second scenario (low cost of plasticity) mildly selects for genotypes with low cost, which favors genetic

assimilation, but only if the correlation between genotypic and phenotypic space is high (otherwise, plasticity is still favored, despite the cost). Finally, when the cost of learning or plasticity is high, selection settles for suboptimal canalized solutions. Curiously, if the correlation between the genotypic and phenotypic landscapes is close to zero (a biologically unlikely occurrence), genetic assimilation does not occur even if the cost of plasticity is very high.

Eshel and Matessi (1998) used a quantitative genetic model that predicts the evolution of adaptively canalized systems that can break down under stress, which allows a wide array of novel phenotypes to emerge (for a similar proposal based on empirical data, see Rutherford and Lindquist 1998). The model also shows that the set of novel phenotypes is likely to include adaptive ones (in the sense of advantageous under the novel conditions), out of the variety of phenotypes accumulated by exposure to past environments and that can be generated from the release of hidden genetic variance (acquired because of canalization: (Rutherford and Lindquist 1998). In fact, the production of phenotypes is biased in favor of [pre] adapted ones in this simulation. These results are in agreement with Wright's observation (discussed in Eshel and Matessi 1998) that so-called "novel" environments are in fact never entirely novel, but are more or less correlated with the array of environments experienced by the population in the past. The authors argue that: "The phenotypes uncovered by the inactivation of the canalizing system are not expected to distribute at random. Instead, one expects a strong enrichment of their distribution in favor of phenotypes which, even before, have been advantageous at least in some rare niches of the environment in which they have evolved" (Eshel and Matessi 1998, p. 2132).

Some of the published models that examine the possibility of genetic assimilation include a role for epigenetic inheritance systems (Jablonka and Lamb 1989; Jablonka et al. 1998). This concept is similar to the suggestion made by some philosophers of science that the entire developmental system is inherited in the course of evolution, not just the genes coding for proteins (e.g., Griffiths and Gray 1997). Pal's (1998) work, for example, incorporates both plasticity and the ability of organisms to pass nongenetic information to the next generation ("memory" in the author's terminology). Pal reaches conclusions similar to those discussed above: when a character is far from a local optimum the generation of novel phenotypic variance is advantageous, but then selection will reduce phenotypic variance (causing canalization) when the population is close to the optimum. The process therefore goes from the presence of phenotypic plasticity to a genetically assimilated character state. Epigenetic inheritance systems turn out to be evolutionarily useful under fluctuating environments, because they make the genotype-phenotype mapping function (Alberch 1991) fuzzier. This fuzziness diminishes the necessity for a precise tracking of environmental changes by the genetic system, a costly and often impossible result to achieve for natural populations.

In a follow-up study, Pal and Miklos (1999) suggest that epigenetic inheritance systems (e.g., chromatin marking, as in the case of DNA methylation) can produce heritable phenotypic variation at a much higher rate than the standard genetic process based on mutation. They link this property

to the rapid production of hybrid inviability or sterility and swift phenotypic change under shifting environmental conditions. These mechanisms can cause divergence between populations prior to genetic differentiation, and then can be reinforced by classical genetic change. Similar to the work by Kirkpatrick discussed above, Pal and Miklos (1999) analyze a model with two adaptive peaks and conclude that epigenetic inheritance systems, by increasing phenotypic variance, may allow a population to approach a new peak essentially without abandoning the other one. That is, the intermediate adaptive valley is eliminated. Pal and Miklos, like Kirkpatrick (1982), put the emphasis on population-level phenomena, suggesting that epigenetic inheritance systems (or the other mechanisms that increase phenotypic variance in Kirkpatrick's model) are likely to yield novel phenotypes simultaneously in many individuals of the population, not just in rare mutants as is the case when classical mechanisms are operating. Pal and Miklos (1999) also conclude—not surprisingly—that the time to peak shift is inversely proportional to population size and the depth of the intervening valley. Interestingly, such time is dramatically reduced if epigenetic inheritance systems are allowed to evolve, regardless of population size and valley depth. Essentially, these systems function as a sort of "extra dimensional bypass" permitting the population to move from one peak to another. This idea has a counterpart in models based on classical allelic substitution in which the high dimensionality of the adaptive landscape forms natural "ridges" from one peak to the other (Gavrilets 1997; Gavrilets et al. 1998). The implications for speciation are obvious: reproductive isolation can arise as a by-product of changes in the epigenetic inheritance system.

A final work on the theory of assimilation we wish to mention briefly is the model by Sasaki and Tokoro (1999) who used neural networks to model evolution enhanced by "learning" (i.e., plasticity). The process of learning in this model is mathematically equivalent to modeling genetic assimilation, and the outcome shows that adaptation is reached very rapidly in a static environment, but that genetic assimilation does not evolve when the environment is heterogeneous, in agreement with the results discussed so far for other models.

All in all, there are some recurring themes that mark theoretical discussions of genetic assimilation. These include: (1) the association with phenotypic plasticity, which has to be present in the beginning to allow the process to start; (2) the relationship between plasticity, genetic assimilation, and the problem of adaptive peaks shifts; (3) the potential role of epigenetic inheritance systems in the process; (4) the concept of bypassing adaptive valleys; (5) the limits imposed by costs of plasticity, which may actually favor a more rapid approach to the final stage of genetic assimilation; (6) the emphasis on the rapidity of the process when compared with standard evolution by allelic substitution only; and (7) the similarity between plasticity and learning from the viewpoint of their macroevolutionary consequences. It is now time to consider some empirical studies of the potential role of genetic assimilation in evolution. As we shall see, little has been done that explicitly incorporates the transitional role of phenotypic plasticity leading to macroevolution-level biological change, but suggestive empirical data exist.

*Modern Empirical Evidence Consistent with
Genetic Assimilation*

Genetic assimilation has recently experienced something of a renaissance in the evolutionary literature. A possible example comes from a study involving selection on morphological plasticity and its implications for speciation in *Anolis* lizards from the Caribbean. Throughout the Caribbean islands *Anolis* colonize a wide range of habitats following 20 million years of evolution: from terrestrial habitats to branches, trunks, and small twigs of trees. Taxa found in habitats with broad surfaces have long limbs allowing them to maintain maximal sprint speeds, whereas those species that use narrow perches have relatively short limbs which enable them to perform delicate movements (Losos et al. 1994). This same pattern of positive correlation between perch diameter and leg length was observed for populations of *A. sagrei* and *A. carolinensis* that were established 20 years ago, after which populations had diversified in their habitat use (Losos et al. 2000). This field experiment, therefore, suggested that phenotypic plasticity leading to partial assimilation might play a role in the evolution of limb size.

To test explicitly if phenotypic plasticity and genetic assimilation or fixed genetic differentiation among populations were possible mechanisms of the evolution of limb size, Losos et al. (2000) grew *Anolis sagrei* in terraria with perches of either narrow or broad dowels to examine the influence of perch size on hind limb length. They found (for both males and females) that relative hind limb growth was significantly different between the two treatments, with greater relative hind limb growth on thicker dowels. Leg length for this particular species depended on perch size during development. The authors concluded that phenotypic plasticity may have played an important role in the evolutionary radiation of *Anolis* lizards across habitat types throughout the diverse islands of the Caribbean. If indeed phenotypic plasticity followed by genetic assimilation was the mechanism of radiation of these *Anolis* lizards, these species would be an excellent example of the role of genetic assimilation at the macroevolutionary scale.

Reaction norm-mediated evolution via genetic assimilation may be quite common in nature (Schlichting and Pigliucci 1993; Pigliucci 2001), and the evolution of aposematic coloration in some insects is a likely candidate example. In many insect taxa, conspicuous warning coloration (aposematism) deters predators that use visual search strategies. Classic theoretical models that describe the evolution of aposematism are based on allelic substitution where genes for conspicuous coloration spread through a population that was originally cryptically colored. However, empirical evidence has accrued on the density-dependent costs of being conspicuous (Sword 1999; Rowe and Guilford 2000) and traditional models have fallen short of accounting for such costs. Investigations of *Schistocerca emarginata* and *S. gregaria* grasshoppers, a case where aposematism is mediated by insect density, have shown that warning coloration is a plastic trait (Sword 1999). Sword suggests that by being plastic the initial costs of conspicuousness may be circumvented.

Grasshoppers are also interesting because of their predator avoidance, which is independent of warning coloration.

Grasshopper palatability is mediated by host-plant chemistry. Both palatable and unpalatable populations of these grasshoppers change color depending on density (Sword 2002). Sword hypothesizes that the plastic aposematic response (via density dependence) is an intermediate, yet adaptive, stage to constitutive aposematic phenotypes. He suggests that if this were true, the palatable and unpalatable populations would have differing reaction norms, and the differences in plasticity would account for the associated costs of being conspicuous. The proportion of change to the warning coloration is strikingly higher and is a more consistent color change in the crowded unpalatable group than in the palatable group, which is in agreement with Sword's expectation. Palatable individuals that are reared at high density do not receive as high a benefit from the warning coloration, and thus are less conspicuous in their color change at high density. Sword concludes that the differential plasticity of density dependent aposematism indicates that the evolution of predator avoidance is likely to occur through genetic assimilation (Sword 2002).

A classic example of phenotypic plasticity is demonstrated in the transplant experiments of *Achillea* by Clausen et al. (1960) at different altitudes in the Sierra Nevada. Gurevitch (Gurevitch 1988, 1992; Gurevitch and Schuepp 1990) revisited their research by examining specific traits which previous ecophysiological work suggested to be influenced by altitude in *Achillea lanulosa*. In this species, leaves are larger and more highly dissected at lower altitudes and become smaller and more compact as altitude increases. Gurevitch collected genets from the Timberline (3050 m) and Mather (1400 m) populations—sites previously used by Clausen et al. (1948) and transplanted them into the greenhouse where they were subjected to two temperature regimes to examine the genetic components of phenotypic plasticity. Differences in leaf-size dimensions are not simply the result of scaling: rather, length increases more than width in warm environments compared to cold ones. Importantly for our discussion, the genetic difference between high and low altitude populations parallels the plastic response to growth in the two different temperatures and the differences observed in the field for the two altitudes remain when plants are grown in a common environment. This experiment provides a good example of how phenotypic plasticity may lead to local adaptation and then eventually genetic assimilation (which in this case would still be ongoing), thereby influencing trait evolution across populations or locally specialized ecotypes.

Generally, the examples we found in the literature follow a similar pattern that corroborates the idea of genetic assimilation via phenotypic plasticity as a mechanism of evolutionary change (Fig. 1). The pattern can be summarized as follows: when a population (A) that descends from one that was adapted to a different environment (B) is grown under conditions similar to the ancestral ones (B, and currently experienced by its close conspecifics or congeners), it expresses phenotypic plasticity in the "appropriate" direction. That is to say, the plasticity is in the direction of the adaptive phenotype expressed by the related populations or species living in the ancestral environment. For example, this is the pattern van Tienderen (1990) observed for two populations of *Plantago lanceolata*, a hayfield and a pasture population.

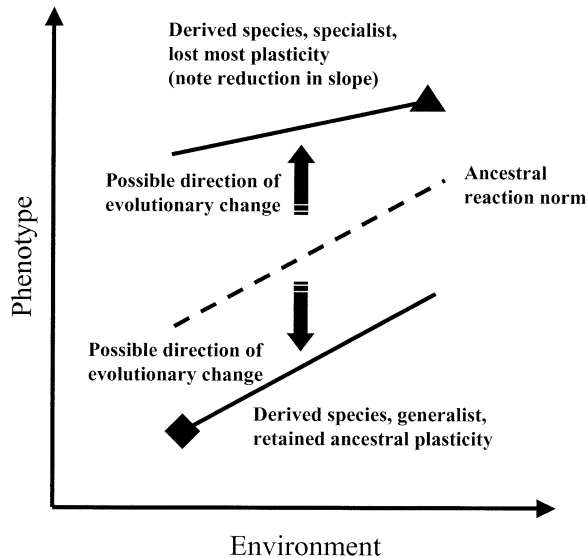


FIG. 1. A scenariogram illustrating a common pattern of differentiation of phenotypic plasticity that may be caused by ongoing genetic assimilation: the lines represent population or species reaction norms for an ecologically important character expressed in response to the variability of a given environmental factor. The ancestral reaction norm is depicted as a broken line. After speciation (or separation of the original population) the two taxa evolve an increasing degree of adaptation to their particular environment, which at least in some cases (upper reaction norm) is accompanied by a reduction of the degree of plasticity. See text for a discussion of some actual examples following this general pattern.

In their home environment, hayfield populations have an erect growth habit, whereas pasture plants normally have a prostrate growth habit. This is consistent with the characteristics of the habitat, because in the hayfield there is competition among plants for light, few rosettes are formed, and they grow erect to compete for light and to reproduce before the field is mowed. However, in pastures plants have more rosettes, an appropriate phenotype in an environment where trampling and grazing are a common selective pressure. When grown in the opposite environmental conditions (high vs. low vegetation associated with hayfield and pasture, respectively), the plants tend towards the phenotype (erect or prostrate) typically expressed in that environment. van Tien-deren (1990) suggests that these results imply limits to adaptive plasticity, probably because of costs or trade-offs, leading to specialization through adaptation to local conditions. However, this pattern is also consistent with the mechanism of differentiation via genetic assimilation (through a progressive reduction of plasticity followed by selection for a specific phenotype in the focal environment), which represents a nonmutually exclusive hypothesis in respect to van Tien-deren's conclusions. The appropriate phenotype is present in the home environment, yet a suboptimal phenotype is also expressed in the opposite environment, consistent with the fact that these specific genotypes have probably never experienced the alternative environmental conditions.

This kind of appropriate response across environments has also been observed for closely related species. Day et al. (1994) raised two species of three-spine stickleback (unnamed species of *Gasterosteus* called "benthic" and "lim-

netic") on the diets typical of the other species under natural conditions. The authors found (as for the ecotypes of *Plantago*) that the natural morphological gap between the two species was narrowed considerably (although not quite bridged) by phenotypic plasticity, depending upon the trait of interest. The species with the more variable diet also showed the greater magnitude of plasticity. Because Day et al. (1994) detected plasticity in these species, their work may be an example of genetic assimilation still in progress.

These empirical studies are similar in the types of experiments that were used, and lend indirect support to the hypothesis of genetic assimilation (through reduction of adaptive plasticity) as a common mode of evolution. The ancestral reaction norm may have been intermediate, or may have been closer to one of the currently existing descendant populations (Fig. 1). This is why it is important to study the evolution of reaction norms using an informed phylogeny (Doughty 1995). Several additional studies on adaptive phenotypic plasticity across multiple populations or species uncover patterns similar to the one described here, yet authors rarely discuss genetic assimilation as the mechanism of differentiation (Pigliucci 2001). In the final section of this paper, we describe several lines of investigation that we think may get us closer to including genetic assimilation into the mainstream of evolutionary research.

Toward a Research Program that Incorporates Genetic Assimilation

Philosopher of science Imre Lakatos (1977) has pointed out that one of the best ways to decide if a scientific theory or idea is a good one is not by directly evaluating its truth-value (which is far from a straightforward problem to tackle), but to see if it leads to a proliferating research program. Even ideas that ultimately turn out to be incorrect, such as the theory of a stationary universe, are useful in science if they spur scientific research which yields fruits, sometimes independently of the accuracy of the original theory (e.g., consider the Big Bang theory which emerged from critiques of the stationary model). In biology, for example, we will probably have a permanently hung jury on the relevance of gradualism versus punctuated equilibria (Eldredge and Gould 1972; von-Vaupel Klein 1995), but the original idea has spurred fruitful research in paleontology and evolutionary biology over the past three decades.

Something similar may hold in the case of genetic assimilation. The first time around, when genetic assimilation was proposed and passionately pursued by Waddington, few people followed up on his research program. The clearly unnatural environmental treatments of most of Waddington's experiments made many people question the importance of genetic assimilation in natural populations. Yet, the idea remains in the literature, infrequently cited but appearing as a plausible alternative hypothesis across the decades. A valiant attempt was made by M. J. West-Eberhard (1989), when she hypothesized that a shift in the threshold of expression of environmentally induced changes in morphology could generate distinct populations very rapidly and result in speciation. For some reason that should be of interest to historians and sociologists of science, the concept of genetic assimilation

ulating more natural situations—both in the laboratory, and whenever possible, in the field—will be more likely to lead to informative data on the timescale and environmental conditions required for genetic assimilation to occur. These lines of research would show convincingly that genetic assimilation is, in fact, a viable evolutionary mechanism under realistic conditions, and would provide important insights into the dynamics that might be involved in its occurrence in natural populations.

As for molecular and developmental biology, comparative studies (again, phylogenetically informed) of the evolution of known environmental sensors (e.g., the phytochrome molecules sensing different aspects of light in plants: Schmitt et al. 1999; Smith 2000; Simmons et al. 2001) in very closely related taxa may uncover situations in which, through time, plasticity has resulted in a canalized phenotype via assimilation. In such cases, one should be able to demonstrate the redundancy of the once-useful environmental receptors, or even find pseudogenes that once coded for active receptors of environmental information. Similarly, comparative studies of changes in methylation patterns and other alterations of gene regulation—put in an ecological perspective—might help in revealing historical (indirect) traces of past genetic assimilation. However, the success of this line of attack will depend crucially on the ability of molecularly inclined evolutionary biologists to make as precise predictions concerning the expected patterns as possible.

A third area of inquiry within molecular biology should focus on the role of hormones as mediators between the external and internal environment, a role much underappreciated in the literature on phenotypic plasticity (Pigliucci 2001, ch. 5). A splendid example comes from the work of Emery et al. (1994) on two ecotypes of *Stellaria longipes*. They compared plants from two environments: an alpine form with low plasticity for stem elongation and a prairie form with high plasticity. They found that these plants responded in opposite directions to the presence of ethylene, a hormone known to be involved in wind-stimulated elongation responses. It is intriguing that the same molecular machinery, and in particular the same hormone, can produce radical alterations of the reaction norms as an adaptation to contrasting ecological situations *within the same species*. This is one clue that genetic assimilation and adaptive evolution via phenotypic plasticity may play important roles in macroevolutionary processes, yet also suggest that in some cases cladeogenesis may be a much faster phenomenon than most researchers interested in the micro-/macroevolution gap usually considered.

There is also a host of related questions that have the potential of shedding light on the issue at hand, while at the same time being valuable fields of research of their own. Some of these further enrich our conceptual map: costs of plasticity (DeWitt et al. 1998; Van Buskirk 2000; van Kleunen et al. 2001), developmental plasticity and heterochrony (Callahan et al. 1997; Gilbert 2001; Weinig and Delph 2001), phylogenetic studies of the evolution of plasticity (Doughty 1995; Qualls and Shine 1996; Pigliucci et al. 1999), epigenetic inheritance systems (Jablonka and Lamb 1989, 1998), the genetic basis of homeostasis (Mitton and Grant 1984; King et al. 1995; Fenster and Galloway 1997), simulated or

artificial adaptive evolution (Wagner 1995; Mayley 1997), and theoretical studies of the relationship between phenotypic variance and adaptive peak shifting (Price et al. 1993; Whitlock 1995; Whitlock 1997). There is no space in this review to examine any of these even briefly, but they are all vigorous areas of research that are mature for a synthesis, and that synthesis may gravitate around the concept of genetic assimilation.

We feel that recent advances in quantitative genetics, molecular biology, and theoretical systematics pave the way for a more complete understanding of genetic assimilation. Although there has been skepticism as to whether we will be able to “catch it in the act,” we assert that the areas of research that we have mentioned above will give us a more complete understanding of genetic assimilation in nature.

ACKNOWLEDGMENTS

We would like to thank C. Boake, J. Dole, and C. Schlichting for commenting on previous drafts of this manuscript and for discussions on genetic assimilation. This work was partially supported by National Science Foundation grant DEB-0089493.

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Corresponding Editor: J. Mitton