

## ***The Role of Phenotypic Plasticity in Diversification***

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The role of plasticity in evolutionary change had been proposed by Wright (1931, p. 147) to be primarily negative: although he praised plasticity as "perhaps the chief object of selection", he damned its role in macroevolution, by pointing out that: "It {individual adaptability} is ... of the greatest significance as a factor of evolution in damping the effects of selection". The view of plasticity as an inhibitor of evolutionary change has been reiterated over the years (Levin 1988; Schaal and Leverich 1987; Stearns 1982).

Although phenotypic plasticity has gained acceptance as an important means of adaptation to heterogeneous environments, its role in population differentiation, speciation and other macroevolutionary events is poorly understood. My goal here is to persuade: first, that phenotypic plasticity actually plays *any* part in the diversification of taxa, and further, that its role may be greatly underestimated. I will discuss several areas where the possession of plasticity may facilitate evolutionary change. Its greatest contribution appears to be in promoting the occupation of new niches by means of the production of phenotypic alternatives. This in turn may pave the way for genetic differentiation. Another key role may be to shield genetic diversity from the discerning eye of selection. Finally, plasticity may enhance the long-term survival of taxa, via species selection.

### ***Plasticity, niche expansion and peak shifts***

The geometrid moth *Nemoria arizonaria* (Greene 1989a,b) produces, via plasticity, two remarkably different larval forms. Caterpillars that hatch early feed upon the inflorescences (catkins) of oak trees and develop into catkin mimics; caterpillars hatching after catkins are gone, feed upon the newly emerging leaves, and the higher diet tannin levels shift development towards a twig morphology (note that these two morphs were initially identified as separate genera). This is an example of a class of plastic responses referred to as developmental conversion, and, reasonably, represents an evolved plastic response that minimizes predation. Schlichting and Pigliucci (1998) pointed out that developmental conversion such as seen in *Nemoria*, can produce peak shifts, strictly environmentally-induced, in the absence of genetic change (see below).

Although we traditionally look to genetic mutations as the source of new variability, it is ultimately phenotypic variation that fuels the engines of selection. It could be easily argued that the gain of distinct morphological or behavioral phenotypes via plastic responses are events of significant evolutionary importance themselves. Adding a new host plant or food type, developing facultative resistance to predators, or markedly altering physiology or morphology to optimize energy capture are all important examples of plastic responses to complex environments. Although each may represent a key adaptation, none necessarily lead to diversification in the strict sense of an increase in numbers of taxa. However, the presence of such plastic phenotypes may represent an evolutionary innovation of considerable import. One need look no farther than the development of polyphenic castes in insects as a prime example: the different castes appear to be largely due to plastic responses to cues early in ontogeny (Evans and Wheeler 2001).

The movement of populations on adaptive landscapes has been of interest since Wright first proposed the concept (Wright 1932). Although most attention has been paid to the roles of mutation and drift, the effects of selection and environmental change on the likelihood of peak shifts have been considered more recently (Price et al. 1993; Whitlock 1997). Pál and Miklos (1999) have also suggested that plasticity and subsequent genetic assimilation could produce peak shifts.

In addition to the evolution of such putative adaptive changes, the multivariate nature of correlated plastic responses may play a part in generating distinctive phenotypes. Schlichting and Pigliucci (1998, p. 321) proposed that the environmentally induced changes in correlations produced through correlated plastic responses of multiple traits “represent a prime route for either (1) moving between peaks without traversing valleys, or (2) for altering the landscape itself.” Thus, they envisioned that changes in trait correlations could produce novel combinations of characters (peak shifts or new peaks), or could alter the relationship between traits and fitness (change the heights of peaks and valleys). The likelihood that environmental change would result in coordinated changes in characters is increased by the modular architecture of organisms.

### ***Genetic Assimilation***

What are the mechanisms by which plasticity might promote genetic change? A process known as genetic assimilation is the prime candidate. Genetic assimilation has been something of a *phoenix*. Its basic premise is that a phenotypic change, initially strictly plastic (i.e., environmentally-induced), can over time be incorporated into the ‘normal’ (i.e., genetically

determined) phenotypic repertoire. It provides a mechanism whereby seemingly Lamarckian shifts can be explained with Darwinian principles. Originally hailed as a means of promoting evolutionary change, it was later considered to be an equally potent buffer *against* diversification. Resurrected as an evolutionary catalyst independently in Russia and England, it was subsequently dismissed as being of minor importance. Now a markedly diverse group of scientists has called for its revival again as a key factor in evolutionary change (e.g., Ho and Saunders 1979; Hinton and Nowlan 1987; Thompson 1991; Wolpert 1994; Rollo 1994; Jablonka and Szathmary 1995, Sarà 1996a,b; Chapman et al. 2000).

The theory of genetic assimilation has been the major vehicle for incorporating plasticity as an evolutionary catalyst (Waddington, 1942, 1961; Schmalhausen, 1949). There are several excellent sources for detailed historical perspectives on genetic assimilation (see e.g., Wcislo 1989; Scharloo 1991; Gottlieb 1992; Robinson and Dukas 1999), so this background to the concept will be short. First introduced as “A new factor in evolution” by Baldwin (1896), it was quickly joined by similar concepts developed by Lloyd Morgan (1896) and Osborn (1897). Their ideas never really caught on: a mechanism for microevolutionary change was of minor interest at a time when the major battle over the form of genetic control of phenotypes was brewing between “Mendelians” and “Biometricians” (Depew and Weber 1995).

### **The Heretics**

More robust conceptual versions of genetic assimilation (GA) were independently developed by C.H. Waddington (1942; 1961) and I.I. Schmalhausen (1949). For each, GA played a central role in processes of evolutionary change. Figure 1 depicts the basic scenario they envisioned. A new environmental factor induces a plastic response that has some advantage under those new conditions (for example, relative to a lack of response). This plastic response may enable the population to persist, allowing subsequent alterations in the reaction norm towards better adaptations, due to expression of formerly hidden variability, recombination or new mutations. Both Waddington and Schmalhausen suggested that, if this new environmental state persists, selection would fix the character state – that is, the plasticity would be eliminated. This step is the culmination of GA: an initially facultative phenotypic change subsequently becomes constitutively expressed by means of conventional natural selection. This could occur for several reasons: 1) there is a cost to maintaining plasticity in the absence of the original environment, 2) random mutation may disable the now hidden plastic response, and 3) there

may be selection for canalization such that the original environmental conditions would no longer elicit that phenotype.

We can create a hypothetical scenario of GA using the geometrid moth *Nemoria arizonaria*. GA could subsequently result in the fixation of either catkin or leaf 'specialists' in isolated populations whose hatch dates were cued earlier or later by temperature or photoperiod. Populations in secondary contact would perhaps be partially temporally reproductively isolated, and speciation could follow. It can be argued that many shifts from generalized to specialized feeding behavior may have followed this route.

Schmalhausen considered GA to be a common occurrence. As evidence he pointed out that in many examples of ecotypic variation in plants and animals, known genetic differences are paralleled, at least in part, by their plastic responses (e.g., Badyaev and Foresman 2000). As an example, he discusses the evolution of the plant *Camelina linicola* (pp. 87-89; see also Stebbins, 1950, pp.125-129). When grown together at low density, *Camelina linicola* differs from its progenitor *C. glabrata* (= *C. sativa*) by having less branched stems with longer internodes, fewer flowers on longer peduncles, and larger seeds. These characteristics make *C. linicola* a remarkable mimic of cultivated flax. *C. glabrata*, when grown in a dense stand of flax, responds plastically to the shading to appear almost identical to *C. linicola*. *C. linicola*, however, is not plastic. Schmalhausen interprets this as a case of GA: "modifications observed if *C. glabrata* is sown in flax fields, [have] already been stabilized and genetically "fixed"."

Waddington and colleagues experimentally demonstrated GA for a variety of traits in *Drosophila* (presence or absence of crossveins in the wings, dumpy wings, enlarged anal papillae in the larvae). For example, following induction with a high temperature treatment of the pupa, the frequency of individuals with broken crossveins (*crossveinless*) following heat shock was greater than 90% after about 15 generations, and following selection, *crossveinless* was expressed even without heat shock at 100% in some lines (Waddington 1953). However, the magnitude of change that could be incorporated in this way was demonstrated in spectacular fashion by Waddington's experimental GA of *bithorax* phenotypes (Waddington 1956). In these experiments, Waddington found that a slight enlargement of the halteres could be magnified into full expression of two pairs of wings, and eventually almost fully genetically assimilated (over 80% in the absence of the original environmental stimulus (Waddington 1956). Although the stimulus in that experiment (ether) was extreme and not likely to be a natural phenomenon, clearly Waddington believed that GA *could* produce macroevolutionary change (Waddington 1975). However, despite his persistent efforts and those of his students to promote the

importance of GA (Waddington 1961), it ultimately was perceived as playing no more than a minor role (Simpson 1953; Williams 1966).

### Revival

The early 1980s saw the beginnings of the current renaissance in several outlying camps. The descendants of Waddington continued to work on GA (Ho et al. 1983a,b; te Velde et al. 1987; te Velde and Scharloo 1988). Rachootin and Thomson (1981), recognizing the failure of the framers of the synthesis to adequately incorporate development, explicitly argued for the importance of understanding epigenetic processes. In this context, they considered that changes in development via GA, even if rare, might promote evolutionary change by opening new adaptive zones.

Matsuda (1982) constructed a scenario whereby an initially environmental response, such as the facultative neoteny of *Ambystoma tigrinum* in response to cold temperature, could be genetically assimilated by means of mutations affecting thyroid hormone secretion or sensitivity, as in the constitutive neoteny of *Ambystoma mexicanum*. Matsuda argued that GA could be easily envisioned as the basis for the evolution of neoteny in other salamanders, as well as in numerous examples of morphological change mediated by hormones (e.g., ecdysteroids and juvenile hormone in insects). Such changes, for example, have been investigated in several species of wing dimorphic insects (Fairbairn and Yadlowski 1997; Zera and Denno 1997; Zera et al. 1998a,b; Nijhout 1999).

An in depth study of the nymphalid butterfly, *Precis coenia*, examined both environmental and genetic influences on seasonal color polyphenism. Rountree and Nijhout (1995a,b) found that the shift from a beige color in the spring and summer to a dark red brown in the autumn, is mediated by ecdysteroid hormones. The effectiveness of these hormones, however, is confined to a period between 28 and 48 hours post-pupation, and produces beige butterflies. Under short day conditions, such as in autumn, ecdysteroid levels rise only after about 60 hours post-pupation, leading to production of reddish brown adults. They characterized a recessive mutant that constitutively produces reddish brown adults, due not to an alteration of the ecdysteroids, but to a change in physiology after the hormone sensitive period (Rountree and Nijhout 1995b). As these examples show, it can easily be imagined that changes in timing of production, or sensitivity of cells or tissues to morphogenetic hormones might result in manifold changes in phenotypes (Harvell 1994), some at least having macroevolutionary implications. Nijhout (1999) reviews the details of the hormonal control of polyphenism in insects.

Ancel (1999) examined the conditions that would favor the assimilation of a plastic response in changing environments, and showed that selection will initially favor broad norms of reaction that include the optimum phenotype, with subsequent selection favoring reduction of the reaction norm breadth (costs of plasticity are an assumption of the model). The likelihood of evolving narrower reaction norms is strongly decreased as the probability of an environmental transition increases.

### ***Plasticity as a Buffer and Hidden Reaction Norms***

Wright (1931) was not wrong about the buffering role of phenotypic plasticity – selection is indeed effectively blind to anything but the phenotype(s) expressed in that particular environmental condition, but with two important caveats. First, a genotype produces a single phenotype only in constant conditions (a coarse-grained environment). Fine-grained environments may elicit several phenotypes during the course of an organism's lifetime, and that individual's total fitness will reflect the summation of effects of selection on those various states. Second, an organism's fitness extends necessarily into its evolutionary future. Thus the extended fitness of a particular genotype will incorporate the fitness of its offspring as well, illuminating the importance of "demic structure" (Scheiner 1993,1998). In this context, even coarse-grained environmental variation gets averaged in, based on the relative frequencies with which offspring inhabit alternative environments, and whether genotype fitness is enhanced by a plastic reaction norm that adjusts appropriately.

The ability of plasticity to buffer natural selection on a trait will depend on the extent to which there is overlap between the genes that determine the trait and those that determine its plasticity. If there is considerable overlap in the genetic control of the trait and its plasticity, there will be a genetic constraint on response to selection by that trait. However, if plasticity and trait means have a degree of genetic independence, then plasticity can, at most, only slow direct trait evolution: plasticity will continue to be favored, but so will alleles that move the trait mean. A number of studies have examined the relationship between trait evolution and trait plasticity and have found evidence ranging from some to substantial independence of the two (Stearns 1983; Scheiner and Goodnight 1984; Schlichting and Levin 1986; Jinks and Pooni 1988; Scheiner and Lyman 1991; Schlichting and Pigliucci 1998; Pollard et al. 2001). Andersson (1989) compared differentiation of means and plasticities of traits in populations of the plant *Crepis tectorum*, and found, contrary to expectation, that more plastic traits appear to evolve faster.

The extent to which plasticity promotes evolutionary stasis is directly proportional to the strength of stabilizing selection favoring a specific phenotype under particular conditions. When different genotypes converge on that phenotype (in essence, canalization of the reaction norm; Schmalhausen 1949), then the other facets of their genetic differences will remain hidden (Gupta and Lewontin 1982; Rollo 1994; Sultan 1987). The evolutionary process of canalization for performance in a particular range of environmental conditions “creates” hidden reaction norms: if it selects different genotypes for their ability to converge on specific phenotypes, then “hidden” genetic and phenotypic variability will be expressed only outside of the zone of canalization (Waddington 1961) – i.e., in environments that have not imposed significant selection on the reaction norm.

A study on *Drosophila* in 1964 by Robertson demonstrates these ideas. He found that wild-type flies were well canalized relative to mutants when reared on ‘normal’ food for *Drosophila*. However, abnormal food conditions (deficiencies in various constituents such as protein, RNA, or choline) revealed substantial genetic variability within the wild-type. In some cases, similar overall changes in body size were attained by altering growth rate either in the early exponential phase or in the later time-independent phase, and it was discovered that the two growth phases were substantially genetically independent. Thus plastic responses of body weight followed different genetic pathways, but achieved similar final phenotypes.

How much variability is being suppressed by stabilizing selection for convergent reaction norms? The concept of the hidden reaction norm has received little attention, but there are several lines of evidence suggesting that the storehouse may be substantial. 1) Studies of yeast and plants exposed to different media, temperatures or pathogens have found differential and on/off gene expression in those environments (yeast: Smith et al. 1996; Wodicka et al. 1997; Jelinsky and Samson 1999; *Arabidopsis*: Ruan et al. 1998; maize: Baldwin et al. 1999). Both Oliver (1996) and Moxon and Higgins (1997) speculate that a large fraction of the putative genes of currently unknown function may be useful in different environments. 2) The production of phenocopies of known mutants by means of changing environmental conditions is also evidence for latent genetic potentials (Goldschmidt 1938; Gibson and Hogness 1996; Ren et al. 1996; Serna and Fenoll 1997).

3) The strongest evidence for hidden reaction norms comes from studies that expose organisms to multiple or to new environments. Gregor (1956) pointed out the strikingly higher variability of plant species in common gardens compared to their natural habitats. Subsequently, a large body of experiments on populations of animals and plants has confirmed that genetic variability ( $V_g$ ) does indeed change under different environmental conditions (e.g., Yampolsky

and Ebert 1994; Pigliucci et al. 1995; Bennington and McGraw 1996; Imasheva et al. 1997, 1998; Hoffmann and Schiffer 1998; Sgrò and Hoffmann 1998a; Suvanto et al. 1999; refs in Schlichting and Pigliucci 1998, Chapter 3; and Hoffmann and Merila 1999). This is an important source of G×E interaction. One detailed example is the work of Sgrò and Hoffmann (1998b) on *Drosophila melanogaster* grown at 14, 25 and 28°C. They found contrasting results for two traits: heritabilities and evolvabilities for fecundity were highest when parents were exposed to 14°C; heritabilities and evolvabilities for development time were higher at 28°C. Clausen, Keck and Hiesey (e.g., 1940, 1948) did many studies with clonal plant material transplanted to new habitats, and observed many instances of the expression of hidden variability (see example in Figure 2).

Other studies have focused on examining environments that are novel for the organism. Several of these have found that  $V_g$  is higher in novel conditions (Service and Rose 1985; Guntrip et al. 1997; Holloway et al. 1997; but see Kawecki 1995). Pigliucci et al. (1995) proposed that the changes in genetic variability can be directly linked to previous selection history and the differential expression of genes in environments that have seldom been encountered. Hoffmann and Merila (1999) provide a review of this and other hypotheses for why genetic variability changes with environment.

The intriguing finding of Rutherford and Lindquist (1998) that Hsp90, a chaperonin that assists in proper protein folding, is capable of 'hiding' a wide variety of structural mutations of proteins, reveals another facet of the hidden reaction norm. Mutations in Hsp90, or environmental stress that reduces the available pool of the chaperonins, increases the expression of the mutant phenotypes. Rutherford and Lindquist's results indicate that the expression of some of these mutants can be readily stabilized, again raising the possibility of trait evolution via genetic assimilation (McLaren 1999).

These studies suggest that there are several routes to the production of new phenotypic variation. 1) Canalization/buffering mechanisms may be depressed via mutation, as in the *hsp90* example, resulting in the production of aberrant phenotypes. 2) Canalization mechanisms may be ineffective if environmental stimuli exceed the limits within which canalization was selected. 3) Evolved systems of response (i.e., adaptive plasticity) to environmental variation may directly activate alternate genetic pathways. In some cases these pathways may represent atavisms - ancestral plastic responses to currently uncommon stimuli.

The standard route to increased phenotypic variation is mutation. Plasticity may play a role even here - some studies have shown that mutation or recombination rates are enhanced under novel or stressful environmental conditions (Gorodetskii et al. 1991; Bridges 1997; Lamb

et al. 1998; Marini et al. 1999; Radman 1999; Goho and Bell 2000; but see Sniegowski et al. 2000; Tenaillon et al. 2000).

Schmalhausen (1949) and especially Waddington (1961) believed that GA was a direct consequence of canalization - the variability hidden by canalization would be selectable when revealed in novel environments (see also Scharloo 1991). Hypothetically, though, GA can occur even if genetic variation is initially absent for that trait in the population (Rollo 1994; Schlichting and Pigliucci 1998) - completion of the process begun solely by plasticity will depend exclusively upon the addition of new mutational variation.

We are taught (or teach!) that phenotypic evolution proceeds via the process of allelic substitution - a change in selective factors favors an allele that just happens to be hanging around the gene pool at some low frequency. Alternative modes are never mentioned. We have suggested (Schlichting and Pigliucci 1998), from first principles, that genetic assimilation is likely to also play a major role in evolutionary change. We note the following conditions: 1) the low probability of having appropriate mutations available to deal with any particular environmental change (low mutation rate  $\times$  low probability of advantageous mutation  $\times$  probability of particular environmental change), 2) the overwhelming likelihood of some form of environmental change, and 3) the pervasiveness of plastic responses (high likelihood of plasticity  $\times$  low to moderate likelihood that plasticity is in appropriate direction – up vs. down). Combined, these conditions actually favor GA over allelic substitution.

If significant hidden phenotypic/genetic variability is uncovered, then allelic substitution can proceed given the initial push in the 'right' direction by phenotypic plasticity. If the new environmental conditions alternate with the former ones, then plasticity itself will be selectively favored. If the new conditions become predominant, then GA can continue until the new phenotype is 'perfected'.

### ***Plasticity and Speciation***

West-Eberhard provided detailed arguments for the importance of polyphenism or behavioral shifts as precursors of evolutionary change (West-Eberhard 1986, 1989, 1992). Although not explicit, the descriptions in these papers make it clear that GA is the primary route of evolutionary change. Here again the spotlight is on plasticity via developmental conversion: discrete phenotypes are produced by a single genotype in response to environmental alternatives, such as in many polyphenisms in insects. West-Eberhard argues that such polyphenism in fact facilitates speciation. For example, a species with two environmentally

induced states could be easily converted to separate forms via *loss* of plasticity in either of the two environments.

In her 1989 review, West-Eberhard also promotes the case for a macroevolutionary (speciation and higher) role for behavioral plasticity. First, she notes that behavior is more labile than morphology. Adaptive behavioral plasticity is more likely to evolve because of availability of cues to stimulate different behaviors (and probably the direct feedback between behavior and reward). Finally, changes in behavior are known in some cases to result in suites of correlated changes in morphology (e.g., Smits et al. 1996b; Thompson 1999). This line of thinking is echoed by Gottlieb (1992), who explicitly invokes GA in the process of speciation.

Wimberger (1994) has also focused on the consequences of behavioral plasticity for diversification in fishes. His focus is on trophic polymorphisms and he posits the following sequence of events: 1) Behavioral plasticity leads to foraging specialization; 2) the morphological differences arising from plasticity tend to reinforce this specialization by altering foraging efficiencies; 3) this promotes partial reproductive isolation, 4) which can be solidified by philopatry (offspring remain in natal territory); 5) leading ultimately to speciation. Plasticity has been especially prominent in discussions of diversification of cichlid fishes, being invoked, either implicitly or explicitly, numerous times (in addition to Wimberger 1991, 1992, see Rachootin and Thomson 1981; Meyer 1987; Witte et al. 1990; Sturmbauer and Dallinger 1992; Rollo 1994; Galis and Drucker 1996; Smits et al. 1996a; Galis and Metz 1998).

Plasticity has also been proposed to have a role in lineage (species) selection. Species selection occurs by means of either differential survival or differential extinction of taxonomic lineages. Factors that promote diversification or survival, such as limited or widespread dispersal, respectively, have been the foci of most studies (e.g., Stanley 1975; Jablonski 1987; Norris 1991; Grantham 1995). Here I present a few instances where phenotypic plasticity might be seen to be an object of lineage selection.

Bürger and Lynch (1995) developed a model to examine the likelihood of extinction under different conditions. He discovered that for a population under directional selection, the mean phenotype will parallel, but lag behind, the optimum favored by environmental change. The magnitude of this lag is directly related to the susceptibility to extinction. Additionally, he found that in finite populations, stochastic variation in genetic variance can contribute to increased likelihood of extinction (see also Lande and Shannon 1996). Plastic responses could clearly ameliorate both problems. A plastic response in the direction of the optimum would reduce the strength of selection on the population, and also perhaps reduce the lag between environmental change and attainment of a fully adapted phenotype. This would appear to

provide ideal conditions for species selection favoring plasticity as a safeguard against extinction. In the case of stochastic variation in  $V_G$ , the higher the magnitude of Wright's "damping the effects of selection" that plasticity has, the lower the probability of extinction via random genetic drift.

Eshel and Matessi (1998) have proposed that a breakdown in canalization *per se* can have similar repercussions. They present a model that indicates that a canalization system (i.e., those mechanisms enforcing canalization) that is inactivated when exposed to extreme environments is more advantageous than one that is resistant. This result is achieved in their model because the inactivated system generates enough random phenotypic plasticity to produce some individuals that are better adapted to the new environment than the canalized phenotype. Additionally, they argue that a system where the breakdown of canalization is 'predictable' can "postadapt" a population to subsequent environmental changes resembling historical environmental extremes – i.e., the genotype contains hidden genetic variability for response to such environments.

Björklund (1994) examined selection on the genetic variance/covariance structure at the population level, and found that a higher degree of integration can respond to selection faster: species selection favoring higher integration. Because adaptive plastic responses often require a genetic framework for integrating the responses of a variety of traits (Schlichting and Pigliucci 1998), there could be a component of higher level selection operating here as well. The most common problem associated with detecting a signal that species selection has operated, is that the signal will be obscured if selection at the individual level is operating in the same direction. Several of the examples above share this difficulty, in that the plastic responses could easily be envisioned as adaptive for the individual first.

Robinson and Dukas (1999) point out that the hypothesis of plasticity as a hindrance or stimulus for evolutionary change can be tested by 1) comparing the diversity of taxa in more or less plastic lineages, or 2) comparing the evolutionary divergences among species using the genetic vs. the total phenotypic trait variances within species (*caveat emptor*: there are many assumptions in such comparisons). Schluter (1996) presents such a comparison: although he did not distinguish between genetic and total phenotypic trait variances, his data suggest that at least environmental variation was not an impediment. Stearns (1983) proposed another approach – comparison of the evolutionary rates of taxa that are more or less plastic.

## **Summary**

I have presented a number of lines of evidence implicating phenotypic plasticity in the process of evolutionary change. A primary role of phenotypic plasticity may be “to just buy time”: the appropriate mutational variation does not have to be lying concealed in the gene pool for a population to persist through an environmental challenge. Additionally, environmental change may release a pool of unexpressed variability, formerly hidden by stabilizing selection and canalization of development. These are the essential parts that plasticity plays in the process of genetic assimilation, suggested as a primary mode for the evolution of phenotypes (Schlichting and Pigliucci 1998).

Genetic assimilation has been of special interest to workers in behavioral evolution (e.g., Wilson and Dugatkin 1998), and can easily be envisioned to be a major mode of change (for example, in changes in host range of phytophagous insects). Another area where GA can clearly be important is in the evolution of the effects of hormones on morphogenesis. Both the timing of hormone production and the sensitivity of tissues are known to be environmentally altered, and both have been seen to have significant effects on morphology and behavior. Genetic assimilation provides a prime route by which initially environmentally induced phenotypic alternatives can be incorporated as constitutive components.

West-Eberhard in particular has championed the role of plastic changes through developmental conversion as an important springboard for evolutionary change. She has argued that the existence of separate adaptive, and well integrated, developmental pathways is the perfect prologue to divergence. These developmental pathways have themselves been molded as a result of selection for divergent phenotypes under different environmental conditions, and they are expressed as plastic responses (condition-dependent polymorphism or polyphenism).

All told there appears to be a substantial amount of variation in the genomes of most organisms in the form of hidden reaction norms (Schlichting and Pigliucci 1998). The buffering role of plasticity and the phenotypic convergence of the reaction norms of different genotypes on the same phenotype (canalization) leads to hidden developmental reaction norms and the possibility of a subsequent ‘explosion’ of phenotypic diversity when the range of ‘normal’ environments is exceeded.

Several authors have independently proposed that the process of development is itself a form of reaction norm to internal (and external) environmental conditions (Wolpert 1994; Sarà 1996b; Schlichting and Pigliucci 1998). Viewed in this light, they have postulated a strong role for genetic assimilation in the evolution of development itself, whereby it results in the

substitution of a constitutive expression of a phenotype for a previously facultative phenotypic response to the internal environment.

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## Figure Legend

Figure 1. A model of the evolutionary progression of genetic assimilation. The development of genotypes is shown at four points in evolutionary time. The optimal phenotype in Environment 1 is 'C'; in Environment 2 it is 'E'. In Environment 1, development proceeds from phenotype 'A', through phenotype 'B' to phenotype 'C'. Although it is only necessary that there be one genotype with the potential for 'D', I have represented two genotypes, the non-plastic **Q** and the plastic **R**, that both produce phenotype 'C'; only **R** has an unexpressed capacity to produce 'D'. In the second time period, the environment fluctuates between conditions 1 and 2. Because Environment 2 is equally frequent, the ability of genotype **R** to express 'D' is favored because it is closer to the optimum, phenotype 'E'. In time period three, Environment 2 is constant, and mutations arise producing a genotype **S** that expresses phenotype 'E'. Note that **S** still retains the capacity to produce 'C' in Environment 1. A final stage (period 4) may be attained, when a non-plastic genotype **T** replaces the plastic genotype **S**.

Figure 2. An example of hidden reaction norms. Graphed are the mean date of flowering of transplants of four types of the species *Potentilla drummondii* at three stations along the transect from the coast to the Sierra Nevada. These subspecies/varieties all occur at elevations above about 1500m and up to 3650m. Note that, although they are generally indistinguishable at the elevations they normally inhabit, there is significant differentiation under "novel" environmental conditions. (Redrawn from Clausen, Keck and Hiesey 1940)

# Genetic Assimilation

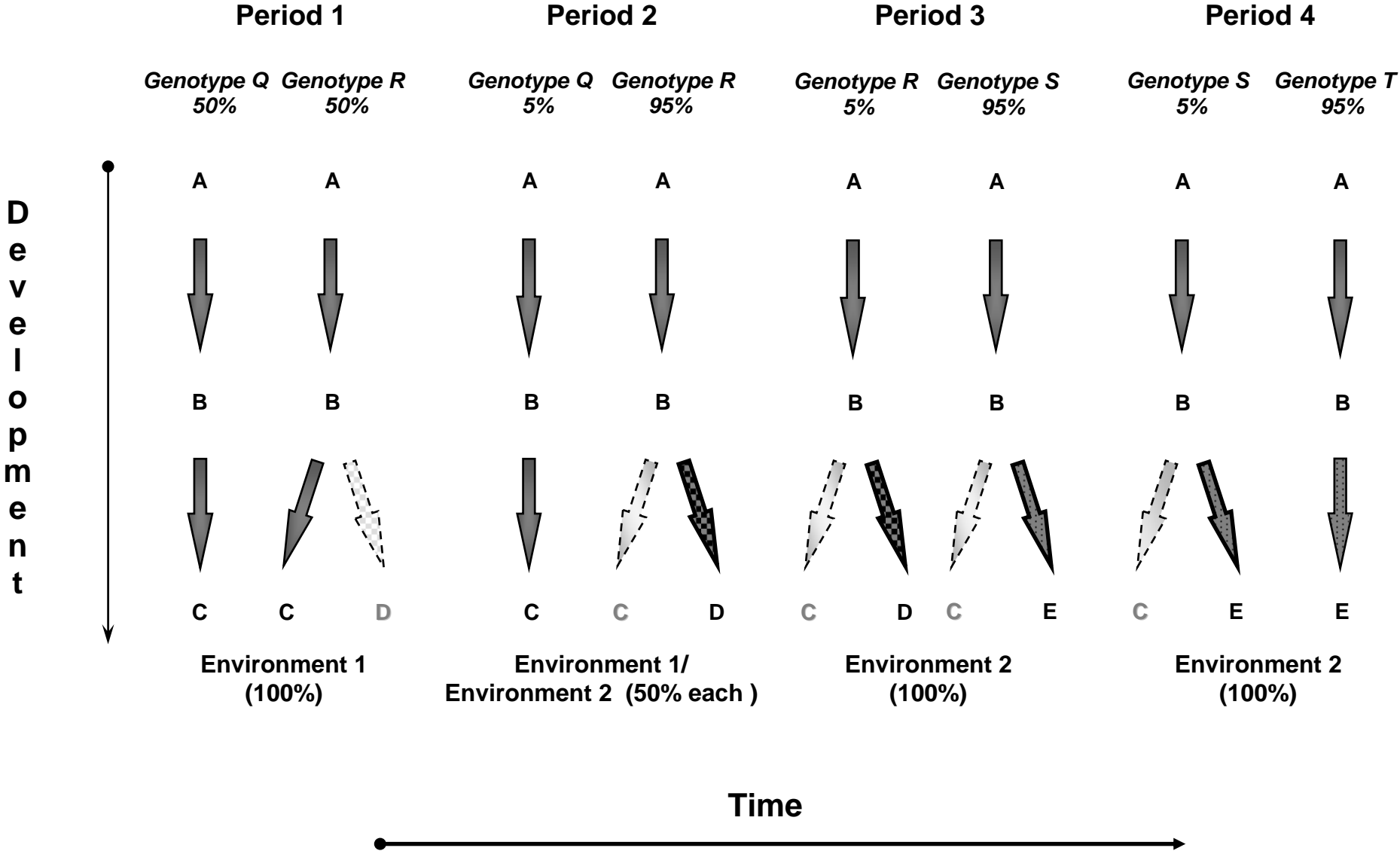


Figure 2.

