

Review Paper

The role of phenotypic plasticity in driving genetic evolution

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Models of population divergence and speciation are often based on the assumption that differences between populations are due to genetic factors, and that phenotypic change is due to natural selection. It is equally plausible that some of the differences among populations are due to phenotypic plasticity. We use the metaphor of the adaptive landscape to review the role of phenotypic plasticity in driving genetic evolution. Moderate levels of phenotypic plasticity are optimal in permitting population survival in a new environment and in bringing populations into the realm of attraction of an adaptive peak. High levels of plasticity may increase the probability of population persistence but reduce the likelihood of genetic change, because the plastic response itself places the population close to a peak. Moderate levels of plasticity arise whenever multiple traits, some of which are plastic and others not, form a composite trait involved in the adaptive response. For example, altered behaviours may drive selection on morphology and physiology. Because there is likely to be a considerable element of chance in which behaviours become established, behavioural change followed by morphological and physiological evolution may be a potent force in driving evolution in novel directions. We assess the role of phenotypic plasticity in stimulating evolution by considering two examples from birds: (i) the evolution of red and yellow plumage coloration due to carotenoid consumption; and (ii) the evolution of foraging behaviours on islands. Phenotypic plasticity is widespread in nature and may speed up, slow down, or have little effect on evolutionary change. Moderate levels of plasticity may often facilitate genetic evolution but careful analyses of individual cases are needed to ascertain whether plasticity has been essential or merely incidental to population differentiation.

Keywords: carotenoid pigments; foraging behaviour; genetic assimilation; peak shift; phenotypic plasticity

1. INTRODUCTION

Many speciation events, and indeed whole adaptive radiations, result from the colonization of a new environment *via* dispersal. Entry into a new environment results in selection pressures favouring divergence from the ancestor. Different environments also directly induce changes in an individual's behaviour, morphology and physiology. Such changes are collectively termed phenotypic plasticity. They may be crucial for the colonizers to survive and reproduce above maintenance levels and, hence, for the persistence of the population (Baldwin 1896; Morgan 1896; Robinson & Dukas 1999). This plasticity is adaptive, in that individuals that show a plastic response have higher fitness than those that do not. It may have evolved as a consequence of variable conditions experienced in the ancestral environment (Levins 1968; Via & Lande 1985; Sultan & Spencer 2002). We ask how plasticity interacts with environmental conditions to produce genetic change, using a simple genetic model and a review of comparative studies.

If individuals can attain high fitness in the new environment as a consequence of a plastic response, it is not obvi-

ous why there should be directional selection at all, and there would then be no adaptive genetic differentiation from the source. Genetic differentiation is expected under two conditions. The first arises if there is a cost to plasticity *per se* (de Witt *et al.* 1998; Ancel 1999, 2000; Sultan & Spencer 2002). A cost implies that in a constant environment, where there is no selection to maintain plasticity, it will be lost. In this way, populations differentiate from each other as they exploit different environments. For example, Sol *et al.* (2002) have shown that among human-aided bird introductions those species that show more innovation in foraging techniques (i.e. show more plasticity in their foraging behaviours) are more successful. Innovative species tend to have larger brains than non-innovative species (Lefebvre *et al.* 2001), suggesting a maintenance cost. Thus, exploitation of new habitats may be associated with a loss of plasticity and the evolution of specialization. The costs of plasticity may contribute to genetic differentiation but are unlikely to be the only, or even major, cause. First, they do not result in novelty, only in the fixation of traits that were expressed in plastic form (Williams 1966, pp. 80–83). Second, limited empirical evidence suggests that absolute levels of plasticity may not change, even as some behaviours become more prevalent in populations. For example, Mettke-Hofmann *et al.*

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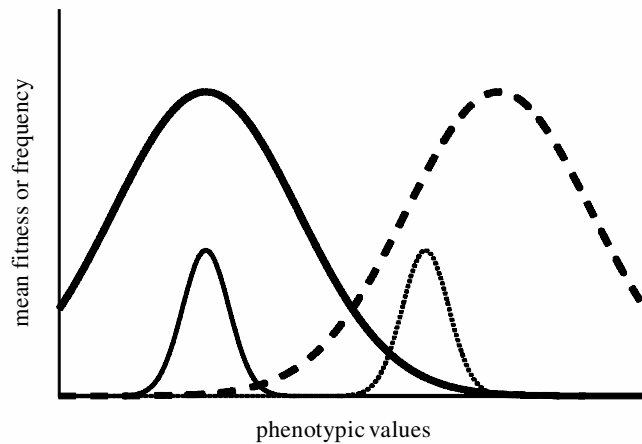


Figure 1. The contribution of plasticity to a peak shift in a changing environment (after Fear & Price 1998). Bold lines are mean fitness; the dashed line represents mean fitness in the new environment. The thin solid line represents the population distribution in the old environment and the thin dotted line represents the population distribution after a plastic response to the new environment. Note that without a plastic response the population will go extinct.

(2002) showed that parrots on islands were more explorative than their mainland counterparts, perhaps in response to a greater range of available unexploited resources or reduced predation pressures.

A second explanation for the way by which plastic traits may become genetically based lies in the process known as genetic assimilation (Waddington 1961). Here, the plastic response to the new environment is incomplete. There is, therefore, directional selection favouring extreme phenotypes in the novel environment and hence some genetic evolution of the trait. Phenotypic plasticity may increase or decrease. However, plasticity will often be reduced because gene combinations favoured under the prevailing conditions are not useful in other environments. These combinations are not selected against if those other environments are not experienced: 'The species has to use whatever genes are at its disposal to meet the demands of natural selection' (Waddington 1961, p. 285). Consider a finch population that finds itself on an island with larger seeds than previously encountered. Behavioural flexibility results in consumption of these seeds, but among the consumers, natural selection favours the larger individuals who can more easily crack large seeds. The net result is evolution of large size, and this may compromise the ability to exploit small seeds.

The process now termed genetic assimilation was first clearly outlined by Spalding (1873) who was concerned with the evolution of instincts, which he thought must have their origin in learning. He argued that selection of those individuals that were the best learners would eventually result in the appearance of the behaviour in the absence of learning. The idea that plastic traits in general could become genetically fixed was raised by Baldwin (1896), Morgan (1896) and others (reviewed by Simpson 1953; Wcislo 1989) but without the same clear statement of mechanism (selection favouring extreme phenotypes) found in Spalding (1873). Waddington (1953, 1959, 1961, 1965) experimentally demonstrated the process and made the case for its general importance in evolution.

Hinton & Nowlan (1987) and Behera & Nanjundiah (1995) used numerical simulations to show how plasticity can speed up or slow down evolution by affecting the distribution of phenotypes subject to selection. Here, we qualitatively evaluate the conditions under which genetic assimilation should occur, using the framework of peak shifts on an adaptive surface.

2. MODEL

The adaptive surface for continuously varying traits is a plot of mean fitness against mean phenotype (Lande 1976; Fear & Price 1998). Because populations are held by stabilizing selection at a peak in the adaptive surface, a central problem in evolution has been how a population can get from a lower peak to a higher peak. We distinguish two general mechanisms by which plasticity contributes to the peak shift (after Fear & Price 1998). In the first, entry into a novel environment affects both the adaptive surface and the plastic response. In the second, the novel environment affects the plastic response, but the adaptive surface is unchanged.

(a) *Environmental influences modify the adaptive surface*

Modifications of the adaptive surface are widely seen as the predominant mechanism for producing peak shifts (Whitlock 1997; Fear & Price 1998). Variation in the adaptive surface should be common, for example, because of variation in the types and abundance of available resources. The way in which plasticity contributes to peak shifts in a changing environment is illustrated in figure 1. It can be seen that in this example, without a plastic response the new environment is so harsh that the population goes extinct (mean fitness = 0). This illustrates the principle of Baldwin (1896) and Morgan (1896) that plasticity may be required for population persistence. In addition, the plastic response pushes the population into the realm of attraction of the new peak, which leads to genetic change.

Waddington (1959) demonstrated this mechanism experimentally using a selection experiment in *Drosophila melanogaster*. te Velde *et al.* (1988) showed that in high salt environments the size of the anal papillae in larvae is reduced, and this is associated with reduced salt uptake. Waddington (1959) maintained larvae in conditions of such high salt that *ca.* 60% died, and it is probable that without the plastic response in the anal papillae a larger fraction of the population would have died. Waddington found that after maintaining larvae in high salt for 21 generations (salt concentrations were modified to maintain a 60% mortality rate) the size of the area between the anal papillae had genetically increased (figure 2). At the end of the selection experiment development in 2% salt was of similar magnitude to that seen in 7% salt at the beginning; i.e. the plastic response to high salt had been genetically assimilated. In this experiment, there is a similar magnitude in the plastic response to varying salt at the beginning and end, but it is centred around a different mean value.

(b) *Phenotypic plasticity on a constant adaptive surface*

Peak shifts can also occur if phenotypic plastic responses are independent of the adaptive surface.

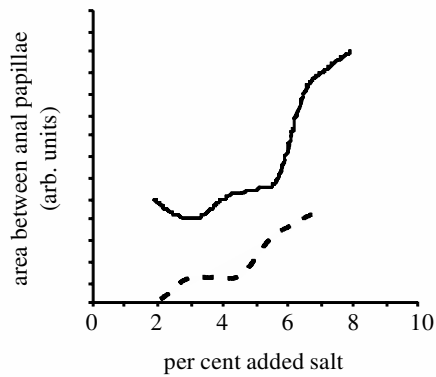


Figure 2. Results of a selection experiment for salt tolerance in *Drosophila* larvae (from Waddington 1959). The area between the anal papillae in arbitrary units is plotted against salt concentration for a lineage subject to artificial selection (solid line; maintained on high salt for 21 generations) and a control lineage (dashed line). The area between the anal papillae is strongly negatively correlated with the size of the anal papillae (te Velde *et al.* 1988). Thus, the size of the papillae probably decreased in this experiment (te Velde *et al.* 1988). Differences between the two lineages indicate a genetic response; differences within each lineage across salt concentrations reflect plasticity. Note that the plastic response to high salt in the control lineage is similar to the genetic response to low salt in the selected lineage, i.e. the plastic response has been assimilated.

Waddington (1953) experimentally demonstrated this idea by selecting for the cross-veinless phenotype in flies. The administration of heat shock to larvae resulted in the appearance of gaps in the wing-veins in a few individuals. By selectively breeding from such individuals for 14 generations, Waddington created a population in which the pattern was present in some individuals even in the absence of heat shock. Here, the environmental agent inducing plasticity is heat shock, but the adaptive surface is independent of the plastic response, being defined by the selection regime imposed by the animal breeder. Waddington (1953) bred selectively from those flies that produced the cross-veinless wing pattern even in the absence of heat shock and this led to some populations where virtually all of them displayed the new pattern. Note that in this example plasticity has been lost, i.e. heat shock in the base population created a wider range of phenotypes than in the selected population. In the parlance of the peak shift model (figure 3), when the population is centred at the lower peak, phenotypic plasticity enables limited exploitation of environments associated with both peaks. However, the higher peak lies sufficiently far to the right of the valley that when the population is centred around this peak, no individuals are able to use the environment associated with the lower peak.

(c) *Optimal plasticity for genetic assimilation*

If phenotypic plasticity is large, the plastic change in the new environment may be sufficient to shift the population directly under the alternative peak in the adaptive surface. There will be no directional selection, no genetic assimilation, and a return to the ancestral environment will result in complete reversion to the ancestral type. As Waddington (1959, p. 1655) noted for the salt tolerance experiment described in figure 2: 'adaptability [i.e.

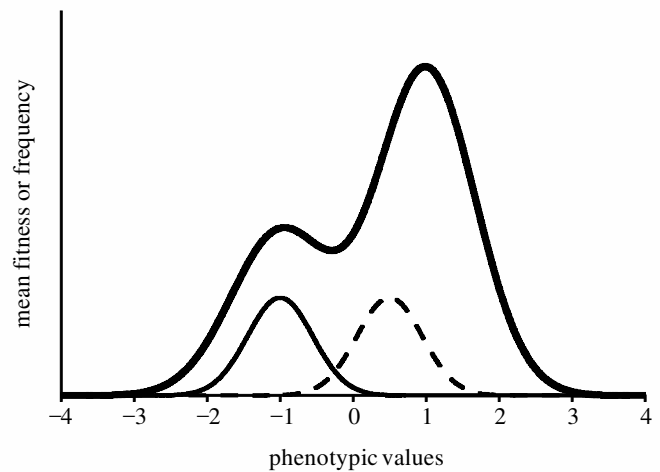


Figure 3. Peak shift produced by plasticity on an unchanging adaptive surface. The bold line shows the adaptive surface. The thin solid line shows the population distribution when there is no plasticity, whereas the thin dashed line shows the population distribution after a plastic response. The adaptive surface depicted here was used for the simulations described in figure 4 (following Kirkpatrick 1982; Whitlock 1997). It was constructed by integrating the phenotypic distribution (standard deviation = 0.44) over a mixture of two individual fitness functions, centred at -1 and $+1$. The width of each fitness function ($= 1/\sqrt{s}$ where s is defined in Whitlock (1997; eqn (1)) is equal to 0.5. The right function is twice as high as the left. Both the phenotypic distribution and fitness functions were assumed Gaussian.

plasticity] is limited and it is clearly because of this limitation that some degree of assimilation has occurred'.

By contrast, if plasticity is small there will either be extinction (figure 1) or else the population will remain trapped under a lower peak (figure 3). It appears that an intermediate level of plasticity should be optimal to produce a peak shift. We numerically demonstrate this using the adaptive surface shown in figure 3. Some results are given in figure 4, where the details of the simulation are presented. There is a threshold amount of plasticity optimal for a peak shift. At this value, an extreme environment is sufficient to push the plastic response into the realm of attraction of the higher peak. At some point during the simulation, there is a run of extreme environments. This means that for several generations the population evolves in the direction of the other peak. It then remains in the realm of attraction to that peak even when the plastic response is small, so evolution towards the peak continues. Higher levels of plasticity may speed the crossing of the adaptive valley, but they reduce the amount of genetic change towards the higher peak, and in the extreme case may prevent it altogether.

3. EMPIRICAL EVIDENCE

Phenotypic plasticity is widespread in nature. Differences between closely related species and populations often have both a genetic and plastic component (Bradshaw & Hardwick 1989; Day *et al.* 1994; Chapman *et al.* 2000). For example, Day *et al.* (1994) compared five foraging-related traits in two species of sticklebacks that they had raised from an early age on the other species'

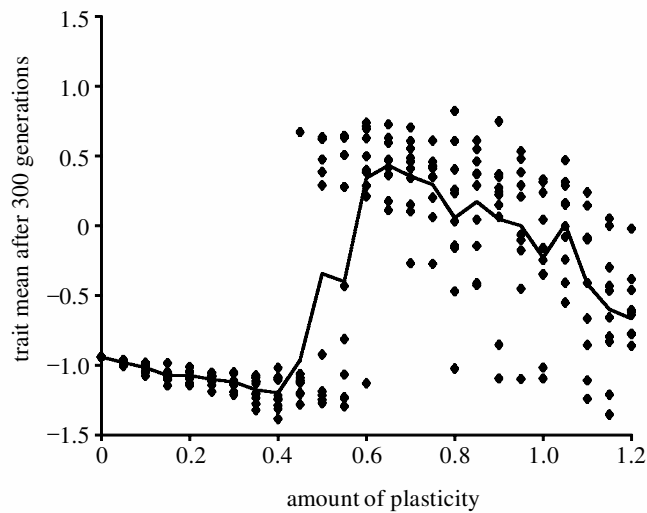


Figure 4. An intermediate level of plasticity facilitates a peak shift. Simulations were conducted by iterating evolution of a population (assumed large enough to ignore effects of drift) on the adaptive surface of figure 3, using the standard quantitative genetics model (Lande 1976) in MATLAB. To simulate plasticity in the i th generation a random quantity, P_i , was added to the mean of the population without altering the population's variance. This quantity was obtained as the absolute value of a normally distributed random variable, with mean equal to 0. High levels of plasticity result if the random variable is drawn from a distribution with a large standard deviation (as indicated on the x -axis). Selection then acts to shift the mean of the population according to the equation $r = h^2(\int p(z)w(z)zdz / \int p(z)w(z)dz - \bar{z}_P)$ (Lande 1976), where r is the change of the mean genotype within one generation, h^2 is the heritability (here $h^2 = 0.5$), $p(z)$ is the phenotypic distribution (with mean \bar{z}_P) before selection but after the plastic response and $w(z)$ is the individual fitness function. The mean in the next generation before selection is $\bar{z}_i + r + P_{i+1}$ where \bar{z}_i is the mean of the previous generation before both the plastic response and selection and P_{i+1} is the plastic response in the next generation. Points give the position of \bar{z} after 300 generations. Ten simulations were conducted at each of 25 different plasticity values for a total of 250 separate simulations. The line connects the mean for replicates at each plasticity value tested.

diet. Plasticity accounted for between 58% (head depth) and 0% (gill raker number) of the difference between the species. Kingsolver & Huey (1998) reviewed temperature acclimation experiments in several different species (maintaining individuals in a new environment and subsequently measuring their improved fitness), which also show an intermediate amount of plasticity. The widespread contribution of both plasticity and genetic differences to population differentiation implies that immediate plastic responses often influence the course of genetic evolution.

The observation that plasticity is present does not, in itself, indicate if it would enhance or slow down evolutionary progress, because this also depends on the form of the adaptive surface. Adaptive surfaces are largely unmeasured in nature (Fear & Price 1998). The strongest case for the importance of plasticity in evolutionary transitions can be made when adaptation to a new niche involves changes in both highly plastic (e.g. a behaviour) as well

as non-plastic traits (e.g. morphology). In the following sections, we investigate this by considering the genetic assimilation of two plastic traits—carotenoid pigmentation and foraging behaviours in birds.

(a) Carotenoids

Carotenoids are responsible for much of the red, orange and yellow colours of birds. Carotenoids are also found in the oil droplets of the retina of birds (Goldsmith *et al.* 1984). A carotenoid-free diet results in no carotenoids in the retina, as shown for young quail (Bowmaker *et al.* 1993). This affects the absorption spectrum, and has been shown to have small but significant influences on colour preferences (Bowmaker *et al.* 1993).

Carotenoids cannot be synthesized by birds but must be ingested. Carotenoid-free diets result in very little colour in the plumage of normally pigmented species such as the house finch, *Carpodacus mexicanus* (reviewed by Hill 1994; Hudon 1994). Quantitative differences in plumage coloration among populations have been related to the presence of specific food plants (Ryan *et al.* 1994; Slagsvold & Lifjeld 1985) and in one case demonstrated to be a result of phenotypic plasticity *via* a cross-fostering experiment (Slagsvold & Lifjeld 1985). The presence of carotenoids in the diet and their deposition in the plumage has led to subsequent genetic evolution. Thus, in many species carotenoids are modified biochemically after ingestion, including modifications that change yellow pigments to red (Brush 1990). Breeding experiments and analysis of hybrids have demonstrated genetic differences among species and populations in the quantity, colour and location of carotenoids deposited in the plumage (reviewed by Brush 1990).

In several species, the carotenoid rhodoxanthin is deposited directly into feathers without biochemical alteration. It is responsible for a dark red to purple colour (Völker 1957). Cedar waxwings, *Bombycilla cedrorum*, typically have pale yellow tips to the tail, but in the past 30 years many individuals in eastern North America have been discovered with bright orange tips as a result of ingestion of berries of an introduced honeysuckle, *Lonicera morrowii*, which contains rhodoxanthin (Hudon & Brush 1989; Witmer 1996; Mulvihill *et al.* 1996). Individuals of three other species (a sparrow, chat and warbler) in the same localities have also been found with a similar orange colour replacing yellow (Mulvihill *et al.* 1996; Craves 1999).

Hudon (1991) used a comparative study to infer how ingestion of rhodoxanthin may have triggered a genetic change in the evolution of tanagers, *Piranga* spp. The red plumage of the scarlet tanager, *P. olivacea*, of eastern North America is due to a carotenoid that requires chemical modification from precursors in the diet. The red plumage of the related western tanager, *P. ludoviciana*, is due to direct deposit of rhodoxanthin. Hudon (1991) suggested that consumption of a rhodoxanthin rich diet by the western tanager is a derived trait, and that deposition of this pigment in the feathers is advantageous because of its low metabolic costs (no chemical modification is required). Despite the difference in pigments between the two species, the colour of light reflected by the feathers is similar (Hudon 1991). At least three evolutionary changes may have occurred subsequent to the adoption of a rho-

doxanthin pigmentary system. First, because rhodoxanthin produces a dark red hue, additional yellow pigments are needed to produce the scarlet coloration (J. Hudon, personal communication). Second, feather microstructure differs between the species (the western tanager's feathers have fewer barbules) and, apparently, this compensates for the different spectral properties of the pigments. Third, species that do not normally encounter rhodoxanthin in their diet, when presented with the pigment, deposit it everywhere carotenoids are deposited (Völker 1955, 1958; J. Hudon, personal communication). The western tanager has the rhodoxanthin spatially restricted to the head region, and this spatial restriction probably reflects genetic modification. Many details remain to be determined, but Hudon's study provides one of the best comparative analyses of how plasticity may drive genetic change.

The extent to which the original appearance of carotenoid plumage in any given bird species was driven by plasticity, i.e. the ingestion of different types and quantities of carotenoids, rather than a novel mutation remains to be assessed. There are many species that ingest carotenoids and are not colourful, so it may be argued that if colour variants are to appear in these species they must result from mutation. However, it is possible that some colour would appear in their plumage if placed on extreme diets, which would then provide the basis for subsequent assimilation. It is worth noting that many millions of canaries and budgerigars have been raised in captivity and a red mutation has never been recorded in either species, even though close relatives of both species have red colours in their plumage (Sossinka 1982).

(b) *Foraging behaviours*

There are many examples where animals respond to unexploited environments with immediate behavioural changes. They include the foraging of warblers in novel habitats on isolated small islands (Morse 1971), and expansion of altitudinal ranges on recently defaunated volcanoes (Diamond *et al.* 1989). Entry into very different environments must be accompanied by behavioural and other plastic forms of accommodation, and this will usually be followed by selection in the context of these changes. The development of tool using in the woodpecker finch, *Camarhynchus pallidus*, on the Galápagos is probably one such example. Although tool using may have arisen and spread as a result of cultural innovation, the habit now develops independently of any tutoring (Tebbich *et al.* 2001), suggesting some degree of assimilation.

Comparisons of populations of the same species on continents and islands have demonstrated differences in foraging behaviours (MacArthur & Wilson 1967; Yeaton 1974; Blondel *et al.* 1988; Prodon *et al.* 2002). Similar comparisons have also demonstrated differences in morphology (Clegg & Owens 2002). In individual cases the behavioural and morphological differences have been directly connected (e.g. Yeaton 1974; Grant 1979; Feinsinger & Swarm 1982). For example, Feinsinger & Swarm (1982) found that a population of hummingbirds on a relatively competitor-free island have longer wings than one in a more competitor-rich island, and related this to their more generalized feeding habits.

The explanation of these observations is that behavioural modifications precede the genetic change. Mayr (1963, p. 604) stated: 'A shift into a new niche or adaptive zone is almost without exception initiated by a change in behaviour. The other adaptations to the new niche, particularly the structural ones, are acquired secondarily'. MacArthur & Wilson (1967, p. 104) suggested that 'changes might be purely phenotypic at first, reflecting the species' behavioural or morphological plasticity, and be translated into genetic differences later by natural selection, perhaps involving genetic assimilation'. However, there are alternatives. In a comparison of Corsican and French mainland birds, Prodon *et al.* (2002) showed that species with expanded altitudinal ranges on Corsica have been there a long time, and many of the more recent invaders have small altitudinal ranges. Prodon *et al.* (2002) suggested that genetic adaptation is required to expand range and argued that this was brought about through natural selection in response to cycling climates in the Pleistocene. In Darwin's ground finches, measurements of selection have shown how mean beak size tracks a changing distribution of seed sizes (Grant & Grant 2002). Potentially, mean morphology could evolve to the level of species differences in response to gradually changing food resources across both time and in space, with behavioural and other forms of plastic response of minor importance.

The case that behaviour precedes and drives evolutionary change would be greatly strengthened if the inferred initial behavioural changes could be experimentally demonstrated. Several studies on tits, *Parus* spp. come closest to doing this. On the Swedish mainland the coal tit, *P. ater*, is small and forages on the outer parts of trees, whereas the willow tit, *P. montanus*, and crested tit, *P. cristatus*, are large and forage on the inner part of the tree. Experiments in both field and laboratory (Alatalo *et al.* 1985; Alatalo & Moreno 1987) show that removal of the large species results in the coal tit moving to the inner tree parts. On the island of Gotland the two larger species are absent and the coal tit forages in the inner tree parts (Gustafsson 1988). It is also larger on Gotland and this size difference is genetically based (Alatalo & Gustafsson 1988). This is the only study where an experimentally demonstrated niche shift mimics a niche shift observed in the field, with these differences associated with genetic differentiation in morphology. However, several other less complete studies on *Parus* species have identified similar patterns (Alatalo *et al.* 1986).

4. CONCLUSIONS

Casting the theory of genetic assimilation in terms of evolution on an adaptive surface means that many results regarding peak shifts by other evolutionary forces apply. Small environmental perturbations, or even the occasional large one, will not be sufficient to cause a permanent peak shift, but if a peak shift does occur there will be a rapid period of evolution followed by stasis. Thus this model, like models of genetic drift (Lande 1985), temporary increases in the phenotypic variance (Kirkpatrick 1982) and fluctuating selection pressures (Price *et al.* 1993) is consistent with a pattern of long periods of relative stasis followed by occasional punctuated evolution.

Recently, genetic assimilation has been modelled as evolution of the phenotypic variance (Eshel & Matessi 1998; Ancel 1999, 2000) or by considering the possibility of non-genetic forms of inheritance (Jablonka & Lamb 1995; Pál 1998) or non-additive genetic determination (Gerard *et al.* 1993). Here, we have emphasized a simple additive genetic model that captures most of the features observed in nature and in experiments. Extreme phenotypes are extreme because of their genes as well as the environment in which they have developed, so selection favouring these phenotypes will result in genetic changes that mimic the plastic response. The amount of phenotypic plasticity may evolve as a correlated response to the change in the mean. Levels of phenotypic plasticity will also be modified in response to selection pressures peculiar to the new environment. Such changes in the phenotypic variance, however, can be logically separated from genetic assimilation.

The central theme of this review is that as a result of plasticity the environment directly influences which phenotypes are exposed to selection (Waddington 1965; West-Eberhard 1989, p. 252). The principles are similar whether single or multiple traits are considered, but in the case of multiple traits, plasticity affects not only the probability of evolutionary change, but also directions of evolution. Phenotypic plasticity affects directions of evolution by determining which peaks on the adaptive surface form the realm of attraction. For behaviour, in particular, there is likely to be a large element of chance. Many bird species show a realm of exploratory foraging behaviours, occasionally resulting in quite innovative foraging techniques (Lefebvre 2000; Lefebvre *et al.* 2001). Some of these may spread through cultural means, and become dominant in a population (Lefebvre 2000). In this way, behaviour can drive genetic diversification along unusual lines. The recent demonstration that feeding innovations are correlated with species numbers in parvorders of birds (Nicolakakis *et al.* 2003) may partly reflect this diversifying role of plasticity. However, the correlation may also be attributable to the greater abilities of plastic species to colonize and persist in new areas, as shown for human-aided introductions by Sol *et al.* (2002).

Throughout this paper, we have assumed that the phenotypically plastic response is adaptive, enabling increased chances of survival and reproduction in the new environment. This may be generally true if the new environment does not differ too greatly from the old one, but entry into a very different environment may produce plastic responses that are maladaptive. Such maladaptive changes will reduce the chances of population persistence and for this reason are less likely to be involved in the creation of novel lineages. However, if the population does persist despite the maladaptation, strong and novel selection pressures could lead evolution along particularly unusual lines.

Entry into a new environment normally involves changes in many traits that interact with each other. We considered two examples. First, carotenoids must be obtained from the environment. Different carotenoids in the diet result in different colours in the plumage, placing selection on other pigments and feather structure. In one example, the interaction of different carotenoid pigments and feather structure actually resulted in very similar

phenotypes (Hudon 1991). Nevertheless, this should be regarded as an example of assimilation, because placing the birds on similar diets would result in differences between them, as a result of genetic differences that have evolved in response to ingestion of different carotenoids. Second, selection on morphology and physiology in the context of changed behaviours results in morphological and physiological evolution. Even if the behaviour itself remains plastic its average characteristics will change from one generation to the next as the underlying morphological and physiological traits evolve, and in this way, the behaviour becomes genetically assimilated.

In this review, we have argued that moderate amounts of (adaptive) plasticity are optimal for evolution in novel environments. Moderate levels of plasticity enhance population persistence and place populations under directional selection towards new peaks. However, the term moderate is defined with respect to the particular environmental conditions. Plasticity may be essential, merely facilitate, or even retard adaptation to the new environment. Future research is needed to ascertain the likelihood of each of these scenarios in nature.

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