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Ecological constraints on the evolution of plasticity in plants

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Abstract. Signal detection and response are fundamental to all aspects of phenotypic plasticity. This paper proposes a novel mechanism that may act as a general limit to the evolution of plasticity, based on how selection on signal detection and response is likely to interact with gene flow in a spatially autocorrelated environment. The factors promoting the evolution of plasticity are reviewed, highlighting the crucial role of information acquisition and developmental lags, and of selection in spatially and temporally structured habitats. Classic studies of the evolution of plasticity include those on shade avoidance, on morphological plasticity in clonal plants, and on selection in spatially structured model populations. Comparative studies indicate that, among clonal plants, extensive plasticity in growth form is favored in patchy environments, as expected. However, among woody lineages from Madagascar, plasticity in photosynthetic pathway (CAM vs. C_3) appears to confer competitive success in areas of intermediate drought stress, rather than allowing individually plastic species to expand their ranges, as has often been argued. The extent of phenotypic plasticity cannot only determine species distributions, it can also affect the sign and magnitude of interactions between species. There appears to be some relationship between developmental plasticity and evolutionary lability: traits that show relatively few transitions within and among plant lineages (e.g., zygomorphy vs. actinomorphy, phyllotaxis, fleshy vs. capsular fruits) usually show no plasticity within individual plants; traits that show extensive plasticity within individuals or species (e.g., leaf size, flower number, plant height) generally also show extensive variation within and across lineages. Transaction and cybernetic costs, as well as long-lived leaves or roots, can limit the tempo of adaptive developmental responses, and create a hierarchy of responses at different temporal scales. Traits whose variation entails few transaction costs (e.g., stomatal conductance) are more likely to be shifted more frequently than those with higher costs of variation (e.g., leaf cross-sectional anatomy). The envelope of responses at the physiological and developmental time scales appears to be an important determinant of adaptive performance. However, adaptive plasticity can limit its own range of effectiveness as a consequence of energetic and competitive constraints, as seen in the allometry and zonation of emergent vs. floating aquatic plants. Plants' inherently low rate of energy capture (and, hence, developmental response and growth) and the high energetic costs of a central nervous system (CNS), may explain why they lack a brain and integrate environmental signals with a slow, hormone-based set of feedback loops rather than with a fast CNS. Finally, environmental spatial autocorrelations – especially those involving factors that determine optimal phenotype – can combine with gene flow and selection for reliance on the locally most informative signals to produce a fundamental limit on the extent of adaptive plasticity.

Key words: allometry, aquatic plant zonation, *Bicyclus*, context-specific competitive success, gene flow, lability, metapopulation genetics, optimal leaf height, signal detection, species ranges, *Sphagnum*

The adaptive domain of developmental plasticity and contiguous phenomena

An organism – or, more precisely, a genotype – exhibits *plasticity* if its phenotype varies with environmental conditions (Bradshaw, 1965, 1973; Stearns 1989; Schlichting and Pigliucci, 1998; Agrawal, 2001). Such phenotypic changes reflect shifts in the underlying pattern of developmental differentiation induced, at least in part, by differences in the external environment. Under a specific set of external conditions, development unfolds as a consequence of phenotypic differentiation among cells and tissues derived from a common genotype, based primarily on shifts in the internal environment (e.g., positional information based on hormone levels or cellular contact, or the indirect effects of other cells or tissues on abiotic factors such as oxygen tension or red/far red (R:FR) ratio). *Metamorphosis* – as seen in holometabolous insects, many marine invertebrates, and different stages of the lifecycle of chordates and higher plants – entails more or less radical changes in phenotype and developmental program at different ages/sizes (Moran, 1994; Higgins and Rankin, 1996) or more rarely under different environmental conditions, as in amphibious plants (Deschamp and Cooke, 1983; Goliber and Feldman, 1990).

Individuals within populations can possess genetic variation for invariant phenotypes, phenotypic plasticity, or metamorphosis. Each genotype can be characterized by a *norm of reaction*, or phenotype as a function of environmental conditions. Plastic norms of reaction can be complex functions of such conditions, with phenotypic change varying not only in amount and pattern, but also in rapidity of expression, reversibility, and ability to occur at different developmental stages (Schlichting and Pigliucci, 1998). Induced phenotypic differences can sometimes be manifested in the next generation, as with the maternally induced, chemical and physical defenses of the offspring of wild radish or tobacco plants exposed to herbivory (Agrawal *et al*., 1999; Van Dam and Baldwin, 2001). Rarely, plasticity can also be communicated within a generation, as with the induction of anti-herbivore defenses in tobacco in response to methyl jasmonate released by clipped sagebrush plants nearby (Karban *et al*., 2000).

Signal detection and response is central to all forms of plasticity, regardless of whether the signals are produced by the abiotic environment (e.g., daylength), by the organism itself (e.g., abscissic acid released by the leaves in response to drought), or by other organisms (e.g., decreased R:FR ratio in light filtered through or reflected from nearby plants [Smith, 1990; Dudley and Schmitt, 1996]). In this paper, I propose a new mechanism limiting the evolution of plasticity, based on how selection is likely to operate on signal detection and response in a spatially autocorrelated environment. I first review the factors promoting the evolution of phenotypic plasticity, highlighting the fundamental importance of information acquisition and developmental lags,

and of selection in spatially and temporally structured habitats. I then consider the hierarchy of plasticity at different temporal scales, emphasizing how transaction and cybernetic costs and organ longevities can limit the tempo of adaptive developmental responses, and how the envelope of responses to conditions that vary at two different time scales – the physiological and the developmental – can be an important determinant of species performance. The genetic and developmental bases for plasticity are discussed, and an argument advanced for how adaptive plasticity can limit its own range of effectiveness due to energetic and competitive constraints. The possible relationships of plasticity to niche breadth and evolutionary lability are explored. Finally, I consider how spatial autocorrelations in the environment (especially those involving the factors that determine optimal phenotype) could combine with gene flow and selection for reliance on the locally most informative signals to produce a fundamental limit on the extent of adaptive plasticity, and on its role in determining species ranges and interactions.

Factors promoting the evolution of phenotypic plasticity

Natural selection can favor the evolution of genetic variants that produce fixed, plastic, or radically reorganized phenotypes/developmental programs. Fitness – the average rate at which individuals of a given genotype produce viable offspring – generally is context-specific. That is, each phenotype usually has a competitive or reproductive edge in some environments but not others. No one form, physiology, or behavior can be adaptive under all conditions, given the unavoidable tradeoffs between performance under one set of conditions vs. another (e.g., see Horn, 1971; Givnish, 1979, 1995; Futuyma and Moreno, 1988; Bennington and McGraw, 1995; Rice, 1995; Dudley and Schmitt, 1996; Reboud and Bell, 1997; Rainey and Travisano, 1998; Schmitt *et al*., 1999; Kluge *et al*., 2001; Relyea, 2002). In a particular environment, selection should favor the phenotype(s) that maximize fitness relative to that of others under the same conditions.

Measurement of fitness

A major challenge to measuring the fitness associated with different patterns of plasticity is that one must, by some means, 'fool' plants into producing phenotypes they ordinarily would not (Schmitt *et al*., 1999). That is, one must somehow override a particular program of plasticity in order to evaluate its net fitness advantage relative to other programs or a fixed phenotype. In practice, this can be done by manipulating the environmental signals that cue plastic developmental responses, or by studying mutant or transgenic plants whose sensory modalities have been ablated or enhanced (Ballaré *et al*., 1991, 1994, 1997; Smith, 1992; Dudley and Schmitt, 1996).

Direct studies of selection on the phytochrome-mediated shade-avoidance response in *Impatiens* (Dudley and Schmitt, 1995, 1996; Schmitt *et al*., 1999; Dixon *et al*., 2001; Maliakial *et al*., 2001) have been paradigmatic in this regard. Many plants etiolate and produce taller canopies when exposed to light with a lower R:FR ratio; such a response should help a plant avoid current or future shade cast by neighbors whose leafy canopies have already altered the spectral quality falling on it (Smith, 1982; Casal and Smith, 1989; Schmitt and Wulff, 1993). Fitness increased with elongation under crowded conditions, as expected given the decreasing allocation to leaves with canopy height and the likelihood of a short plant being next to, and hence under, a taller competitor where coverage is dense (Givnish, 1982, 1984, 1995). Under sparse coverage, fitness decreased with elongation as expected, given the reduced likelihood of a short plant being next to a competitor. Schmitt *et al*. (1999) argued that – because fitness didn't vary with height among transplants pretreated with low or with high R:FR – the tradeoff based on increased allocation to stem tissue was inoperative, and that the main downside of elongation under uncrowded conditions might be lodging/collapse in the absence of lateral support (see also Weinig and Delph, 2001). In fact, however, R:FR pretreatment had a persistent impact on leaf vs. stem and root allocation even after the transplants accomodated themselves to crowded vs. uncrowded microsites (Maliakial *et al*., 2001). Gilbert *et al*. (2001) showed that early-successional trees exhibit far more etiolation in response to shading and low R:FR than do shade-tolerant, late-successional species; Givnish (1984) was the first to argue that precisely these differences in strategy would be adaptive, by reducing the energetic overhead associated with stem construction for a plant that can maintain positive photosynthesis in shade (and by enhancing its carbon uptake by increasing canopy area, not height, and by attempting to grow out of the shade for plants with a shade-intolerant physiology. Unfortunately, this and related papers on optimal leaf height as a function of crowding (e.g., Givnish, 1982, 1995; Tilman, 1988) have generally been ignored by those writing on phytochrome-mediated shade avoidance, even though the energetic tradeoffs discussed by these ecologists are fundamental to the questions being addressed.

Selective forces promoting plasticity vs. development of fixed phenotypes

When individuals of a given species experience the same conditions throughout their lifespan – as a result of environmental variation only at large spatial and/or temporal scales relative to the size, longevity, and movements of individuals – selection should favor the evolution of genotypes that produce invariant phenotypes (Bradshaw, 1965; Levins, 1968; Hedrick *et al*., 1976; Reboud and Bell, 1997). Examples of this phenomenon are legion, and include the evolution of heavy-metal tolerance in grasses on mine tailings (Antonovics *et al*., 1971;

Wu *et al*., 1975), of serotiny in plants of fireswept regions (Givnish, 1981; Gautier *et al*., 1996; Enright *et al*., 1998), of dark pelage in mammals on lava flows (Endler, 1986; Stangl *et al*., 1999), and of differences in diet and diet-induced jaw structure in fish exposed to closely related competitors (Schluter, 1994, 2000). No form of plasticity – even ones in which the phenotypes produced under different conditions are adaptive, and identical to the invariant forms favored locally by natural selection – is likely to yield a positive advantage under such conditions.

When the environment varies only spatially, not temporally, plasticity is expected only when an individual is likely to move through more than one kind of patch in its lifetime. Kassen and Bell (1998) and Bell (unpublished ms.) argue that, when movement (or environmental change) is so slow that an individual is likely to encounter only one change in environmental conditions in its lifetime, selection is likely to favor 'ductile plasticity' – involving a one-time phenotypic shift in response to external conditions early in life. (Ductile plasticity is metamorphosis if the phenotypic shift involved is large.) A special case arises for clonal organisms that spread vegetatively through a spatially patchy environment and retain functional connections among the modules of a given clone (Hutchings and Wijesinghe, 1997). Under those circumstances, modules that are specialized for harvesting the resources that are locally abundant may yield an ecological advantage, favoring the evolution of plasticity in module form, physiology, or behavior (Friedman and Alpert, 1991; Stuefer *et al*., 1994, 1996). Clonal organisms are, almost by definition, pre-adapted for the specialization of different modules to different conditions – whenever multiple organs have the same function, some can evolve new or modified functional capabilities while others retain previous, necessary capabilities. Furthermore, the optimal pattern of plasticity for functionally integrated modules can differ dramatically from that for separate modules. The classic case involves the spread of a plant through patches that are light- or nutrient-rich (Friedman and Alpert, 1991; Hutchings and Wijesinghe, 1997). For functionally integrated clones that can share resources through intact connections, selection should favor heavy allocation to leaves in modules in light-rich patches, and heavy allocation to roots in nutrient-rich patches; exactly the opposite pattern would maximize whole-plant growth for individual, unconnected modules (Givnish, 1979, 1988; Mooney and Chiarello, 1984; Tilman, 1988; but see Dong *et al*., 2002). If the cost of transporting complementary resources is small, functionally integrated clones should outperform ensembles of unconnected modules: they require less energy allocation to roots because they root in nutrient-rich soil, and achieve higher photosynthetic rates because they bear N-rich leaves in sunny microsites. Fransen and De Kroon (2001) found, however, that selective foraging in *Holcus lanatus* can deplete resource-rich patches and thus erode the advantage of such foraging. Clearly,

repeated disturbance or resource enrichment must reset conditions for clonal plants with reversed specializations of individual modules to be favored. Plants with finer roots show increased precision of foraging for patchily distributed nutrients (Wijesinghe *et al*., 2001) and presumably would be adapted to finer-scale patterns of resource heterogeneity. However, it must be remembered that clonal connections may simply permit a plant to explore space that is temporally inhospitable to seedlings or adults; for example, energetic subsidies from well-lit ramets allows dwarf bamboo to explore densely shaded microsites in Japanese forest understories (Saitoh *et al*., 2002).

When the environment varies only through time, not space, genotypes that produce adaptive patterns of plasticity should be strongly favored, provided that the resulting variation in phenotypes can track (or, better yet, anticipate) temporal variation in the environment closely enough to yield a net advantage over a temporally invariant phenotype (Bradshaw, 1965; Novoplansky *et al*., 1990, 1994; Gabriel and Lynch, 1992; Moran, 1992; Padilla and Adolph, 1996; Reboud and Bell, 1997; van Tienderen, 1997; Tufto, 2000; Relyea, 2002). Six key questions thus emerge, each bearing heavily on whether selection should favor phenotypic plasticity in a particular situation:

(1) What are the excess energetic costs (or, ultimately, the marginal decrements to fitness) of phenotypic shifts vis-a-vis invariant phenotypes (Schlichting, 1986; van Tienderen, 1991; Scheiner, 1993; Via *et al*., 1995; Relyea, 2002)?
(2) How do such costs vary with the rate at which different phenotypes are produced?
(3) To what extent is the range of phenotypes that can be expressed limited in plastic vs. invariant genotypes (Via and Lande, 1985; Moran, 1992; DeWitt *et al*., 1998; Relyea, 2002)?
(4) What differences in relative performance (and, ultimately, in increments to fitness) would plastic vs. invariant phenotypes experience in temporally and/or spatially environments?
(5) How large need these differences be to favor the evolution of phenotypic plasticity (van Tienderen, 1991, 1997; Gabriel and Lynch, 1992; Zhivotovsky *et al*., 1996)?
(6) Which environmental factors, if sensed today, could best predict conditions in the future – and, hence, optimal behavior at that time (Ballaré *et al*., 1987, 1994; Hairston, 1987; Novoplansky *et al*., 1990, 1994; Moran, 1992; Aphalo and Ballaré, 1995; Padilla and Adolph, 1996; DeWitt *et al*., 1998; Tufto, 2000)?

In a temporally varying habitat, plasticity should be most likely to evolve when the costs and/or limits of plasticity (points 1–3 above) are low, its benefits (points 4, 5) are high, and phenotypic variation can track or anticipate environmental

change (point 6). Based on an extensive literature review, DeWitt *et al*. (1998) inferred that the costs of plasticity can include one or more of the following:

(i) *Maintenance* – The energy (or other resources) required to sustain sensory and regulatory systems needed for plasticity but not for the production of invariant phenotypes;
(ii) *Production* – The excess energy needed to produce a trait in a plastic genotype, compared with the same trait in a developmentally invariant phenotype;
(iii) *Information acquisition* – The energetic investment (or, more generally, the decrement to fitness) in sensory modalities, regulatory chains, movement, and/or exposure to potential competitors, predators, or pathogens required to acquire and integrate data on spatial and temporal variation in the nearby environment;
(iv) *Developmental instability* – Any imprecision in development under a specific set of conditions that plasticity may occasion, with consequent reduction in fitness; and
(v) *Genetic costs* – Energetic costs or decrements to fitness caused by linkage or by pleiotropic or epistatic effects associated with an otherwise favorable pattern of plasticity.

Limits on the benefits of plasticity include:

(vi) *Information reliability* – Decrements to fitness caused by reliance on imprecise cues to assess environmental conditions, or a mismatch between organismal traits and the current environment caused by changing conditions;
(vii) *Lag times* – Decrements to fitness caused by slow developmental responses that create a mismatch between traits and the current environment; and
(viii) *Developmental range limit* – Reduction in the range (and, hence, function) of phenotypes that can be produced through plastic vs. fixed development; and
(ix) *Epiphenotype problems* – Decrements to function and fitness due to the partial ineffectiveness of 'add-on phenotypes' produced late in plastic development, compared with similar forms/physiologies/behaviors produced earlier (and possibly better integrated) in fixed development.

These costs and limits are fundamental to the evolution of plasticity: in their absence, ubiquitously adaptive plasticity should evolve (van Tienderen, 1997). Schlichting and Pigliucci (1998) argue that most of these costs and limits to benefits might be reduced by selection: the additional maintenance and production costs associated with plasticity could be reduced (i, ii), sensory

modalities fine tuned (iii), canalization favored (iv), and developmental programs altered to decrease unfavorable side effects of plasticity (v, viii, ix).[1] Schlichting and Pigliucci conclude, however, that information reliability (vi) and the potential problems caused by lags between signal detection and phenotype production in organisms with plastic development (vii) are inherently more difficult problems for natural selection to solve. The 'transaction costs' of changing phenotypes (see Mechanisms of developmental plasticity) can lead to lag times; they, together with geographic variation in the adaptive utility of different environmental signals, are at the heart of two fundamental constraints on the evolution of plasticity outlined at the conclusion of this paper (see Ecological constraints on the evolution of plasticity).

When the environment varies through both time and space, adaptive plasticity should also be favored (Zhivotovsky *et al.*, 1996; van Tienderen, 1997), again provided that phenotypic variation can track or anticipate changes in the environment closely enough to yield an advantage. Selection, operating on populations within spatial patches, should favor phenotypes that increase relative fitness within those patches. For plants, these phenotypes may often be those that capture energy at the highest rate, because such individuals will have the greatest assets with which to compete for additional resources (e.g., nitrogen, light), to invest in reproduction, and to set aside reserves for enduring harsh periods (Givnish, 1979, 1982, 1986a). Selection would then favor organisms with the highest rate of energy capture, relative to other organisms, under a specific set of conditions.

What does this say about plasticity – that is, about how that same organism's performance should vary, relative to itself, across different conditions? In essence, not very much – what is important is ecological performance relative to other species across a range of environments, not the amount of phenotypic plasticity per se. To my knowledge, a proper search for a general relationship between plasticity and niche breadth has yet to be conducted. Such a test would require (i) a clear conceptual distinction between plasticity and adaptive plasticity, and (ii) comparative, common-garden studies of the phenotypic development of species in the absence of competitors and other natural enemies across the same, wide range of environmental conditions. The first requirement produces operational measures of plasticity that do not assume the validity of the premise being tested. The second is essential for determining whether broad

[1] Indeed, Relyea (2002) recently found no support for developmental instability or decreased range of phenotypes produced by plasticity in wood-frog tadpoles adjusting their tail length and musculature, body size, and behavioral tempo to the presence of predatory dragonfly nymphs; Relyea also found some instances of an energetic benefit, rather than cost, associated with plasticity. However, Scheiner and Berrigan (1998) and DeWitt (1998) have demonstrated energetic costs associated with plasticity *per se* in predator-induced defenses of waterfleas and snails, respectively.

realized distributions cause greater amounts of observed plasticity (more or less tautologically, as a consequence of widespread species being exposed to more diverse conditions), or whether greater plasticity (assayed across species without bias introduced by their natural distributions) causes or is correlated with greater distributional breadth.

A recent study by Van Kleunen and Fischer (2001) on populations of *Ranunculus reptans* from the shoreline of Lake Constance is an important first step in this regard. Ramets from areas with substantial heterogeneity in coverage – and, hence, competition – showed much more responsiveness to heterogeneity in competition under experimental conditions than those drawn from barren areas lacking in local competitive pressures. In this species at least, phenotypic plasticity appears to confer competitive success in patchy environments, while genetic variation for the degree of plasticity appears to confer the ability to growth over a range of environments with greater and lesser amounts of local microsite heterogeneity. In a survey of $\delta\ ^{13}C$ values across lineages of Madagascar plants that exhibit CAM photosynthesis, Kluge *et al.* (2001) found that obligate CAM species were restricted to the driest areas, obligate C_3 species were restricted mainly to humid areas, and C_3-CAM cyclers with a plastic physiotype were common in areas with intermediate and varying levels of drought stress. In this case, plasticity in photosynthetic pathway appears to have conferred competitive success along just another segment of a climatic continuum, rather than expanding the ranges of individual species or lineages. The lineages with the broadest distributions were those which showed extensive *evolutionary lability*, having evolved species with all three, genetically determined photosynthetic variants, each adapted to conditions along a different portion of the climatic continuum.

Population genetic models

If we consider spatial patchiness in the environment alone, or temporal variation in the environment facing a single population, then the population genetic models for the evolution of plasticity are well understood and selection should favor invariant phenotypes and plastic phenotypes, respectively (Gabriel and Lynch, 1992; Zhivotovsky *et al.*, 1996; Reboud and Bell, 1997; van Tienderen, 1997; Kassen and Bell, 1998). But for complex patterns of spatio-temporal variation in the environment, we must develop *genetic metapopulation models* in order to understand fully the circumstances favoring fixed or plastic phenotypes. Specifically, we need to take models like those of Zhivotovsky *et al.* (1996), van Tienderen (1997), and Tufto (2000) and incorporate explicit spatial and spatio-temporal structure, so that we can analyze how rates of migration, phenotypic change, temporal variation in conditions

within patches, and spatial variation in conditions among patches may interact – constructively or destructively – to work for or against the evolution of adaptive plasticity. While temporal variation should generally favor plasticity if it occurs at rates which development can track, this may not always be true. For example, if adjacent patches tend to undergo similar shifts in environmental conditions, and the resulting 'metapatches' of such conditions are big enough and migrate across the landscape even at relatively high rates (as might happen in a dune field, or in boreal fir waves), populations may be able to migrate with the metapatches and selection may wind up favoring fixed rather than plastic phenotypes. Alternatively, if the metapatches are too small and last for shorter periods, the 'compromise' invariant phenotype favored by massive gene flow and rapid environmental fluctuations may be substantially less fit than plastic phenotypes that (imperfectly) track environmental fluctuations. Demographic subsidies from favorable patches can also maintain genotypes in many patches where they are nominally at a disadvantage (Holt and Gaines, 1992).

It must be recognized that the range of microenvironments for which a given phenotype produces maximum relative fitness should decline with the number of competing phenotypes in the surrounding landscape. When the diversity of competing, adaptive phenotypes in a landscape is large, the number of microenvironments in which a given phenotype has a competitive edge may be quite small. Maximization of relative fitness under only a single set of conditions is a recipe for extinction. Thus, when a genotype becomes restricted to very few patch types, extinction might become an important selective force favoring adaptive plasticity. Plasticity might substantially reduce the chance of extinction by extending an organism's range to include several similar patch types; the selection pressures involved might be expressed at the species level, or (far more likely) at the population level within metapopulations. A key question is how important are rare events and extinction selection in promoting the rise of phenotypic plasticity.

Constraints on the evolution of adaptive plasticity can have fundamental effects on the ranges of individual species, the nature of interactions among competitors, mutualists, and predators and prey, and the composition and dynamics of communities and food chains (Agrawal, 2001). Depending on the interaction between the reaction norms of plants and pollinators, or of plants and their herbivores, these mutualistic or antagonistic relationships could either by stabilized or amplified by plasticity. For example, Kessler and Baldwin (2001) have recently shown that the release of several volatile organic compounds (e.g., methyl shikimate, *cis*-α-bergomotene, and several terpenoids) by *Nicotiana attentuata* after being damaged by herbivorous insects both deters further oviposition by such insects and elicits the arrival of several of their predators. The combination of roughly halfing the oviposition rate of herbivores and increasing predator attack rates by a factor of 4.9–7.5 resulted in

overall decrease of roughly 95% in herbivory, simply as a result of the interaction of the reaction norms of plants, herbivores, and predators. If such effects were coupled, as seems likely, to herbivore density and plant reproductive success, it seems clear that plasticity could play a predominant role in regulating the population densities and distributions of all actors in this food chain.

Types of developmental plasticity

Hierarchies of developmental plasticity

Selection within environmental patches favors the maximization of relative fitness within those patches. Selection across patches may tend to maximize the relative fitness of plastic phenotypes across several different patch types under some conditions, and in only a few under others (see above). Plasticity that is narrowly adaptive in just a few patch types should be favored by (i) diverse arrays of local competitors and natural enemies; (ii) patch sizes that are large relative to the dispersal capacity and reproductive longevity of the focal species; (iii) greater clumping of exemplars of individual patch types than of those of different but suitable patch types; (iv) a high variance in transaction costs across patch types (favoring plasticity where it is least costly); (v) a high variance in signal reliability across patch types (favoring plasticity where the most useful signals are available; and (vi) a conflict across patch types in the kinds of signals or plastic response programs that enhance fitness.

These considerations should apply to plasticity at any of its hierarchical levels: *short-term plasticity* (involving behavior or rapid physiological adjustment to fluctuating conditions, such as changes in photosynthesis in response to changes in temperature or irradiance), *intermediate-term plasticity* (involving morphological and/or physiological acclimation to different conditions [often involving cell or organ turnover], or behavioral learning), and *long-term plasticity* (involving phenotypic shifts that occur but once or a few times per lifetime, such as juvenilism or environmentally induced metamorphosis in plants). Over evolutionary time scales, populations or lineages may differ in the *lability* (tendency toward genetic variation and change) of their patterns of developmental plasticity. A key evolutionary question is whether there is a correlation between the amount of short- to long-term plasticity a population displays and the extent to which its pattern of plasticity can change through time. Do individual plant species that compete well in both sun and shade tend to have relatives that have invaded a wide range of irradiances?

Arguments can be made on either side of this question. Plasticity could reduce responsiveness to selection (resulting in a negative correlation between

plasticity and lability), or selection in different environments could favor one or another set of genes that permit the expression of alternative pheno-types under different conditions (resulting in a positive correlation). The second possibility is supported by an interesting pattern seen in herbaceous plants of temperate forests in eastern North America (Givnish, 1988). Congeners of understory species with particular leaf phenologies (e.g., spring ephemeral, early summer, late summer, winter annual, evergreen) tend to share the same seasonal pattern of foliar activity even if they occur in non-forest habitats. Relatives of some spring ephemerals (e.g., *Erythronium*) are active in the short, cool, brightly lit growing season of alpine meadows; those of some late summer species (e.g., *Aster, Desmodium*), in open habitats like prairies with a long growing season; and those of some winter annuals (e.g., *Phacelia*), in deserts following winter rains. Species with a dimorphic leaf phenology – in which two different sets of leaves, or different leaf positions, are developed at different seasons – often belong to genera that show substantial interspecific variation in leaf phenology (e.g., *Geum, Stellaria*) (Givnish, 1988).

Which plant traits are plastic?

It is no doubt highly significant that qualitative traits that show relatively few transitions within and among angiosperm lineages – such as petal number, radial vs. bilateral symmetry, parallel vs. pinnate venation, opposite vs. alternate leaves, and fruit type (excluding differences in seed coat thickness or dormancy) – usually show no developmental plasticity within individual plants. In contrast, quantitative traits that show extensive plasticity within species – such as leaf size, flower number, plant height, crown area, and resource allocation to leaves, stems, roots, and reproduction – also show extensive variation within and across lineages, at least some of which is genetic and, hence, evidence of a tie between evolutionary lability and developmental plasticity. It is not clear what differentiates these classes of phenotypic traits – after all, why can plants vary flower number but not petal number? I suspect that it has to do with the nature of the underlying developmental cascade: if that cascade is responsive to signals and feedbacks from the environment or the state of the plant itself, more or less continuous variation can be regulated, and differences in such regulation can evolve and become manifest in different groups. If, on the other hand, the cascade is insulated from environmental signals, quantitative adjustment of a trait cannot proceed by tuning the amount by which particular signals modulate the cascade; shifts can only occur by modifications to the cascade itself, which might proceed less rapidly due to internally generated selection pressures maintaining the status quo due to a burden of epistatic and pleiotropic effects. Even so, fixed differences in such qualitative traits

can evolve fairly rapidly in response to selection pressures. For example, in *Calochortus* and Liliaceae subfamily Lilioideae, narrow leaves, parallel venation, winged dry seeds, and bulbs have undergone concerted convergence in response to the invasion of open, seasonal habitats; their sister groups (and inferred ancestors) inhabit forest understories and bear the broad leaves, pinnate venation, fleshy fruits, and rhizomes adapted to those conditions (Patterson and Givnish, 2002).

Plasticity at two different time scales

If a plant can track environmental variation rapidly and cheaply enough, the *envelope* of its short- and intermediate-term plastic responses may sometimes be more important than the details of the responses themselves (Fig. 1). For example, when plants are grown at different levels of irradiance, they develop leaves with different photosynthetic capacities, based on differences in leaf thickness, mesophyll photosynthetic capacity, and stomatal conductance (Bjorkman *et al*., 1972; Bjorkman, 1981; Chazdon *et al*., 1996). In the short term (minutes to hours), photosynthesis per unit mass of the leaves developed under a conditioning light regime will show a response over physiological time scales, increasing with ambient irradiance at an ever decreasing rate, plateauing at high (but not damaging) irradiance. Leaves grown at higher irradiances over the intermediate term (days to weeks) tend to have higher rates of peak photosynthesis and dark respiration, and require more light to saturate photosynthesis and balance leaf respiration. Calculations for *Atriplex triangularis* (Givnish, 1988, 1995) show that – at least for the three conditioning irradiances under which plants were grown by Björkman *et al.* (1972) – steady-state photosynthesis at a given irradiance is maximized by plants grown at that irradiance (Fig. 1). Plants with higher rates of photosynthesis (and whole-plant growth) under a given light regime should have a competitive advantage under that regime. Thus, the envelope of photosynthetic light responses – that is, maximum photosynthetic rate as a function of the irradiance to which leaves are acclimated (Fig. 1) – may provide more insights into a plant's competitive ability across a light gradient than its short- or intermediate-term steady-state responses. Comparison of the response envelopes of different species would indicate the range of light regimes over which each species would have a relative edge, taking acclimation into account. Givnish and Vermeij (1976) made a similar argument for the utility of response envelopes for studies of thermal adaptation and acclimation. Response envelopes could be local (reflecting the genetic make-up and adaptations of individual populations, exposed to a particular spectrum of conditions) or global (reflecting genetic differentiation across populations and ecological conditions within a species), and thus bear

on the performance and competitive ability of a species locally or across its range.

Ultimately, response envelopes must also incorporate the dynamic response of photosynthesis to short-term fluctuations in irradiance and/or temperature. Leaves acclimated to a particular light regime, for example, still require a finite time to induce full photosynthetic capacity, so their steady-state photosynthetic responses overestimate actual carbon gain during brief sunflecks, which provide much of the useful radiation to plants in shaded understories (Chazdon and Pearcy, 1986a, b; Pearcy, 1990; Chazdon *et al.*, 1996). For such situations, the appropriate response envelope should thus be a surface relating photosynthesis (or growth) to the irradiance and average sunfleck duration to which a plant has been acclimated.

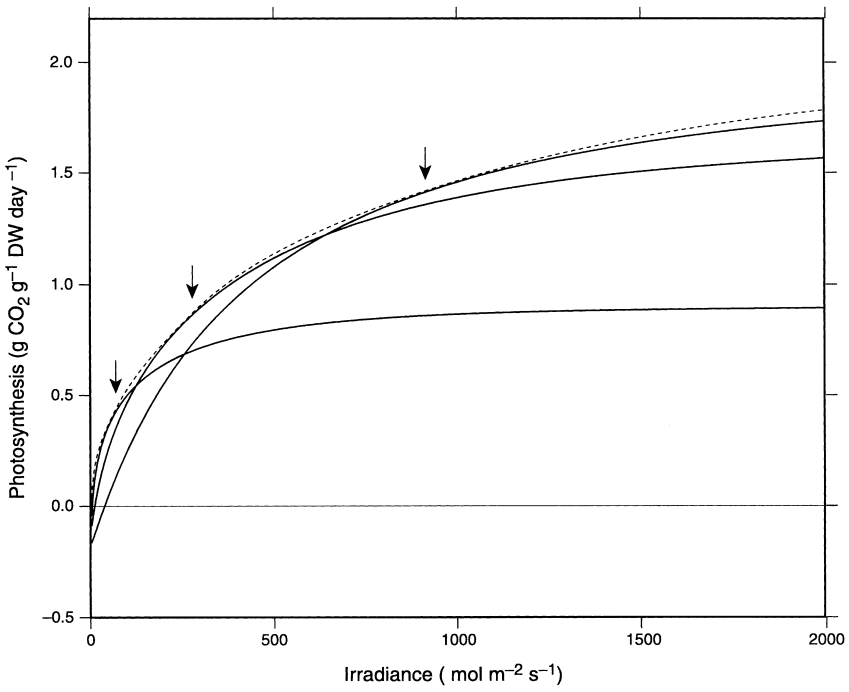

Figure 1. Photosynthesis as a function of irradiance in *A. triangularis*, expressed as daily carbon balance per unit leaf mass (Givnish 1988, based on data of Björkman *et al.*, 1972). Arrows indicate the high, intermediate, and low irradiances at which plants were grown and to which leaves were acclimated. The three solid curves shown represent the short-term photosynthetic response to irradiance by leaves acclimated to high, intermediate, and low conditioning irradiances. Leaves acclimated to a given irradiance have the greatest carbon gain at that irradiance, but only if photosynthesis is expressed per unit investment (i.e., mass or soluble protein), not per unit area as presented classically (see Givnish, 1988). The dashed curve represents the hypothetical 'envelope' (local maximum) of such responses, showing steady-state photosynthesis at a particular irradiance for leaves acclimated to that irradiance.

Mechanisms of developmental plasticity

At the genetic level, phenotypic plasticity can reflect *allelic sensitivity* or *regulatory control* (Schlichting and Pigliucci, 1993, 1998). Allelic sensitivity entails shifts in the amount or activity of transcripts from a gene caused directly by a change in environmental conditions, such as temperature; regulatory control involves the epistatic modulation of development by one or more regulatory genes whose action is sensitive to specific environmental cues. In regulatory control, a specific environmental signal (e.g., a low R:FR ratio) is thought to affect a receptor, whose change of state is transmitted by the products (often hormones) of a transducer gene, which triggers the production of transcriptional regulators by regulatory genes, which affect target promoter regions (*cis*-regulatory elements) upstream of other regulatory or (ultimately) structural genes, the amount and activity of whose products, finally, help generate an environmentally dependent phenotype (Smith 1990, 1995, 2000; Rollo, 1994; Arnone and Davidson, 1997; Callahan *et al*., 1997; Doebley and Lukens, 1998; Purugganan, 2000). Schlichting and Pigliucci (1995) and Pigliucci (1996) argue that the complex and closely integrated mechanism of regulatory control is most likely to be adaptive, allowing the precise modulation of the products of structural genes regardless of their own biochemical sensitivity to conditions, and permitting anticipatory responses to environmental change via reactions to environmental signals (e.g., photoperiod, low R:FR ratio) that are closely correlated with future conditions (e.g., temperature or rainfall, density of competitors). Classic studies of regulatory control of plasticity in plants include the detection and transduction of irradiance at different wavelengths by members of five phytochrome families (Smith, 1990, 2000), and of ethylene – produced by plants in response to wounding, pathogenic attack, flooding, or fruit ripening – by receptors encoded by ETR1 and four other genes related to bacterial histidine kinases (Bleecker *et al*., 1998; Johnson and Ecker, 1998; Woeste and Kieber, 1998; Hall *et al*., 1999; Bleecker and Kende, 2000).

The conservation of such signal detection mechanisms across higher plants, but their apparently divergent effects on development, is one of several lines of evidence suggesting that changes in transcriptional regulators and the downstream promoter regions they target may be a central means by which development and phenotypic plasticity evolve (Doebley and Lukens, 1998; Purugganan, 2000; Shepard and Purugganan, 2002). That is, phytochrome and ethylene sensitivities are universal, but different plant species have diverged strikingly in their morphogenetic responses to the same stimuli. Individual transcriptional regulators are enmeshed in far fewer developmental cascades than genes involved in signal detection and transduction, and so – less constrained by pleiotropy – should be free to evolve more rapidly

(Doebley and Lukens, 1998). Many structural and signaling genes also appear to be under rigorous selection to maintain the production of functional products, and vary little among distantly related organisms (Halder *et al*., 1995; Endo *et al*., 1996). Transcriptional regulators (and the promoters they target) might thus evolve rapidly, permitting rapid shifts in the spatial or temporal domains of target gene expression, and be capable to produce functional but novel phenotypes (Goodrich *et al*., 1992; Hanson *et al*., 1996). Recent research on MADS box-containing transcriptional regulators in plants (Meyerowitz, 1994; Münster *et al*., 1997; Goto *et al*., 2001) and homeodomain-containing transcriptional regulators in animals (Panganiban *et al*., 1995; Averof and Patel, 1997) has generated many important insights into developmental regulation, as well as the large-scale evolution of developmental pathways and body plans. Since the Precambrian, animal phyla have conserved a remarkable concordance between the genetic order of transcriptional regulators and the physical order of the body segments whose development they regulate (Carroll, 1995; Valentine *et al*., 1996; Erwin *et al*., 1997). Duplication of key regulatory genes appears to have been an important intermediate step in the evolution of increased developmental complexity and precision over time (Smith, 1995; Averof *et al*., 1996; Cooke *et al*., 1997; Grenier *et al*., 1997). Mutations to the ABC family of duplicated MADS-box genes control development of the four floral whorls across angiosperms and homologues occur in all other vascular plants (Meyerowitz, 1994; Munster *et al*., 1997), and a search has now begun to determine how these homeotic genes and control pathways have evolved throughout the history of the angiosperms (Goto *et al*., 2001; Soltis *et al*., 2002). Detailed molecular analyses of regulators have finally made it possible to identify such enigmatic organs as the lodicules and the palea/lemma of grasses as being homologous to the petals and possibly the sepals of other flowering plants (Ambrose *et al*., 2000; Goto *et al*., 2001).

At the developmental and ecological levels, plastic changes in phenotype require energetic investment (see point [1] above); if such changes occur more than once during the lifetime of an organ (or, where appropriate, an individual), then plasticity *must* have an excess cost over the production of similar fixed phenotypes. Furthermore, plastic changes in phenotypes with lower energetic costs should occur more frequently and over shorter time scales (see point [2]; Mooney and Chiarello, 1984; Givnish, 1986b). For example, in plants, stomatal aperture and conductance can be varied at very low costs, while changes in leaf thickness require the irrevocable commitment of cellulose and other materials in building a new leaf, a far greater cost. It is thus not surprising that plants adjust stomatal conductance over periods of seconds to minutes, while leaf thickness is adjusted – via the production of new leaves – only over periods of weeks to years. Small differences in performance (on the

order of 1 or 2%) separate adaptively optimal patterns of plasticity in stomatal conductance in relation to humidity, photosynthesis in relationship to irradiance, and leaf nitrogen in relationship to position within the canopy (Field, 1981; Cowan, 1986; Givnish, 1986a, b, c). Thus, not only can very small differences in performance have profound evolutionary consequences, the exact amount of the 'transaction costs' of the shifting of phenotype are crucial for understanding the evolution of plasticity. The modulation of traits at different frequencies has important implications for the integration of these traits at different temporal scales; traits that vary at high frequency (e.g., stomatal conductance) should be co-adapted to and track those that vary at low frequency (e.g., leaf phenology, root/shoot allocation) (Mooney and Chiarello, 1984; Givnish, 1986b).

In plants, low photosynthetic rates per unit leaf mass are associated with long leaf lifetimes (Reich *et al*., 1997) – if plants fix carbon at a low rate, old leaves are only slowly shaded by a plant's own new leaves, putting off the day when it is profitable to withdraw the remaining leaf N and put it into new leaves atop the canopy (Reich *et al*., 1992). Other things being equal, factors that tend to decrease photosynthesis and whole-plant carbon gain – such as soil infertility, or deep shade – should increase leaf lifetime and decrease plasticity in traits that can only be adjusted by building new foliage (e.g., leaf thickness, maximum mesophyll photosynthetic capacity) (see Kitajima, 1996).

Unlike higher animals, plants must develop plasticity without the benefit of a central nervous system. Possession of a CNS confers the ability to sense and respond rapidly to environmental changes. This ability comes at a substantial cost, however. Among vertebrates, brain mass increases as the 0.75 power of body mass (Jerison, 1973; Allman, 1999). At a given body mass, carnivores and frugivores tend to have a greater brain mass than herbivores, and primates (and mammals generally) tend to have a greater brain mass than birds, and birds a greater brain mass than reptiles or fish. Metabolic rate per unit body weight is positively correlated with relative allocation to brain tissue at a given body size (Allman, 1999). Although the biomass allocation to the CNS may be small in absolute terms, its metabolic rate per gram is extremely high, much higher than muscle and far higher than bone or fat.

Plants may simply have too low a rate of energy capture to make it profitable to invest in a costly neural computer with which to integrate environmental factors and muster a rapid response. Moreover, the low rate of energy capture in plants might so slow the rate at which plasticity could be expressed through the growth of new tissue that it would largely eliminate any advantage that might be gained through the rapid integration of environmental stimuli and calculation of adaptive phenotypic responses. Perhaps if plants had

evolved on an alternative, non-greenhouse Venus and had been able to harness a much stronger flux of solar energy, they would have evolved brains to position their leaves to counter moves by competitors! Or perhaps they might have done so on Earth, if the efficiencies of light capture by chlorophyll and CO_2 capture by Rubisco had been a few orders of magnitude higher. As it is, plants integrate and respond to environmental stimuli partly by using slow-acting feedback loops driven by antagonistic hormones (e.g., abscissic acid released by leaves during drought tends to decrease stomatal conductance and to increase leaf shedding and root proliferation; cytokinins released by the roots tend to enhance leaf production [Cowan, 1986]).

Consequences of developmental plasticity for higher organizational levels

Plasticity and ecological distribution

There is no necessary relationship between the extent of phenotypic plasticity – in form, physiology, or behavior – and the range of environments over which a species occurs. Plasticity, per se, does not limit species distributions; the extent to which plastic responses depart from the locally adaptive strategies does. To paraphrase Darwin (1872), natural selection favors neither phenotypic plasticity nor phenotypic invariance as a general rule. Plasticity in phenotype may have nothing to do with niche breadth, or be negatively correlated with it.

A species' pattern of plasticity may limit its distribution proximally, by determining where that species is better adapted to local environments than its competitors. In this respect, plasticity differs little from other aspects of phenotype, except that it contributes an advantage mainly by tracking environmental variation and enhancing average performance across such variation. A nice illustration of this principle is the relationship between plasticity in growth rate in different species of *Sphagnum* mosses, and the breadth and position of their distributions along small-scale environmental gradients from hummock top to hollows in peat bogs (Fig. 2). As shown by Clymo and Reddaway (1972), the species that grow fastest relative to competitors in particular microsites are those that dominate those microsites. Among the taxa studied, there does not appear to be any relationship between the extent of plasticity in growth rate and the breadth of a species' distribution; most species dominate in only one of three microtopographic microsites from hummock top to pool, regardless of the plasticity in growth rate they exhibit (Fig. 2). There is also no relationship between the microsites where a species reaches its maximum growth rate (its physiological optimum) and those where it reaches its maximum growth relative to competitors (its ecological optimum). Species niche breadth is, however, closely related to the range of microsites over which its

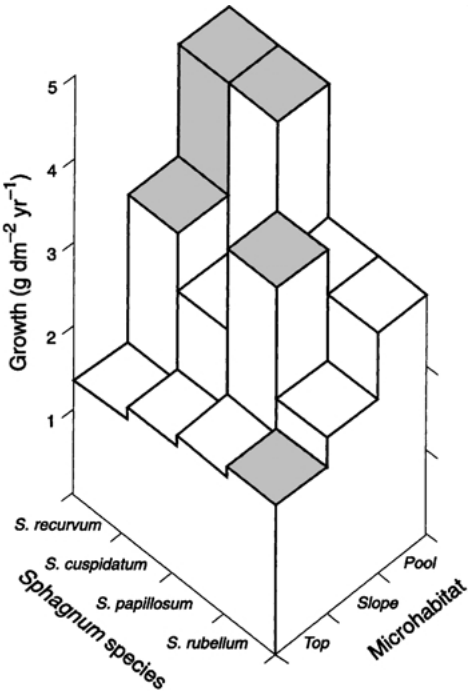

Figure 2. Height growth of four species of *Sphagnum* transplanted to the top, slope, or bottom (pool) of hummocks in highly infertile British bogs (after Silvertown, 1982, based on data of Clymo and Reddaway 1972). Shading indicates the microsite(s) each species typically dominates. Each species dominates the microsite(s) in which it has the highest growth rate relative to others, even if its growth there is less than what the same species achieves elsewhere. For example, *Sphagnum rubellum* grows far more rapidly in moist pools than on hummocks, but it grows faster atop hummocks than other species and dominates those microsites, while losing out to the faster-growing *S. recurvum* and *S. cuspidatum* in pools. These results (and similar ones for trees in bogs [Givnish and Montague, 1996]) flatly contradict the repeated assertion by Grime (1979) and colleagues (e.g., Grime *et al*., 1997) that plants in unproductive habitats achieve dominance not by outgrowing other species, but by growing more slowly and having less demand for resources.

plastic response is adaptive, where its growth rate is locally optimal and exceeds that of each of its competitors.

A more interesting question is whether organisms can show fully adaptive patterns of plasticity over a wide range and still have limited distributions. The answer appears to be yes – there are limits beyond which continuous variation in the expression of one strategy cannot maximize relative fitness, and species with a qualitatively different strategy are favored.

This principle is illustrated by the factors underlying the depth zonation of aquatic plants. In ponds, small lakes, and slow-flowing streams, emergent herbs with aerial leaves dominate shallow water (<ca. 1 m), attached floating-

leaved species like waterlilies dominate deeper water (<ca. 4 m), and submersed species dominate deeper water yet (<18 m) (Sculthorpe, 1967; Spence, 1982; Singer *et al.*, 1983). This zonation appears to reflect the influence of growth form on competition and the ability to maintain positive whole-plant carbon gain at different depths (Givnish, 1995). Emergents can overtop and exclude the shorter floating and submersed species at a given depth. But as the emergents spread into deeper water, they must allocate an increasing fraction of their biomass to the relatively unproductive petioles needed to hold their leaves above water. At their whole-plant compensation depth, the energetic income emergents obtain from photosynthesis should just balance the cost of building and maintaining leaves and their associated petioles and roots. Beyond this depth, they should show negative growth and dominance should shift to the next tallest growth form, the floating-leaved plants.

Floating-leaved plants should be able to spread into deeper water because their petioles are tethers, not cantilevers, and are far more slender (and thus, less costly) at a given depth than those of emergent plants. However, at some point they too will reach their whole-plant compensation depth. Beyond that, submersed species should dominate; they do not have direct access to light and atmospheric CO_2 at the water's surface, but also need not produce stems long enough to reach the surface (Givnish, 1995).

As expected, floating-leaved plants do allocate more to foliage than petioles at a given depth than emergent plants, and their allocation to leaves does drop more slowly with depth (Fig. 3). These trends appear to be adaptive, reflecting structural constraints on plant form. The more massive petioles of emergents are consistent with a general tendency for the diameters of self-supporting

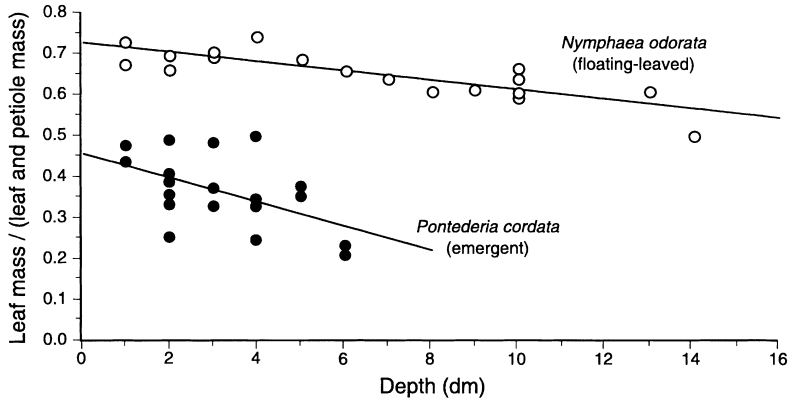

Figure 3. Proportional allocation of biomass to leaves vs. petioles as a function of depth in the emergent aquatic plant *Pontederia cordata* and the floating-leaved *Nymphaea odorata* (T.J. Givnish, unpublished data).

structures in plants to scale as their length raised to the 1.0–1.5 power (McMahon, 1973; Niklas, 1993); by constrast, tensile structures have diameters that are independent of length but scaling as the 0.33 power of the mass or drag they bear (Peterson *et al*., 1982; Givnish, 1995). Removal experiments (T.J. Givnish, unpublished data) confirm that emergents competitively exclude floaters from shallow water, but floaters do not exclude emergents from deeper water. So, while both emergents and floaters appear to show smoothly varying, adaptive patterns of plasticity in petiole allocation and design with depth, there are limits to the range of depths over which each of their patterns can yield a competitive advantage. The depth distributions of emergent, floating, and submersed species are, I would argue, a consequence of their individual plasticities of energy allocation to petioles, which in turn help determine their whole-plant compensation depth. Each growth form reaches its outer depth limit not because its allocation to petioles is maladapted to the structural constraints it faces, but because its adaptive variation of allocation with depth ultimately paints the plant into a corner: adaptive plasticity in the same developmental plan leads to negative carbon balance, and competitive demise, beyond a certain depth.

This kind of situation may have deep generality. Phenotypic plasticity often arises through the subtle modulation of stereotyped developmental patterns (see *Which plant traits are plastic*? above). Sun- and shade-adapted leaves in a given plant are generally still produced on branches with the same phyllotaxis, using the same basic pattern of lamina and vein development, and with the same arrangements of leaves, buds, and flowers. Small, plastic shifts in these stereotyped developmental patterns can arise in a variety of ways (e.g., heterochrony [Alberch *et al*., 1979]), be favored by selection in straightforward ways, and be adaptive over a range of habitats. But as conditions vary continuously beyond this range, survival or competitive success may require a discontinuous shift to another developmental pattern. Such a shift may require selection for a different developmental pathway, not merely the tinkering of an existing pathway – and thus, be less likely to arise.

The achievement of plasticity through modulation of a particular developmental pattern may impose important constraints on plasticity thus achieved. One potential example is the use of telomeres in vertebrates to measure and limit the number of cell divisions. This mechanism, which is thought to have evolved to help prevent cancer or other forms of uncontrolled cell proliferation, may prove to be a fundamental constraint on age plasticity and evolutionary lability. In addition, the use of primary growth for tissue production in most vertebrates may ultimately prove to be one of the factors most responsible for senescence; the resulting lack (in most instances) of a developmental program for intercalating new tissue in areas damaged or lost would interfere with selection for long life or immortality.

Ecological constraints on the evolution of plasticity in plants

As discussed above, the evolution of plasticity is limited by its costs and limits to its benefits (DeWitt *et al*., 1998; Schlichting and Pigliucci, 1998). Of these costs and limits, perhaps the most difficult to circumvent are those involving the detection of reliable environmental cues and the lag times between such detection and the development of adaptive phenotypes (Schlichting and Pigliucci, 1998). We have seen how unproductive conditions – by reducing rates of plant growth and imposing a fundamental lower limit on lag times – can limit the evolution of adaptive responses to unpredictable and rapidly changing conditions. Here I would like to conclude by outlining a previously overlooked – and perhaps crucial – constraint on the evolution of adaptive plasticity based on signal detection and integration; this constraint arises as a result of spatio-temporal autocorrelations in environmental conditions, possibly joined by the costs of sensing and integrating various environmental signals.

Optimal plant form, physiology, and behavior should, in general, be context-specific. Ideal stomatal conductance, for example, depends on a plethora of external conditions, such as irradiance, sunfleck duration, humidity, air temperature, wind speed, soil nitrogen and phosphorus levels, soil moisture, hydraulic conductivity, temperature, and oxygen content, and the expected rainfall, temperature, irradiance, and daylength in the near future (see Cowan, 1986; Givnish, 1986c; Chazdon *et al*., 1996). Producing an optimal, competitively successful phenotype through adaptive plasticity thus requires, in principle, the detection and integration of a large number of environmental signals.

Cybernetic costs have long been raised as a potential barrier to the evolution of plasticity (see review by DeWitt *et al*., 1998). However, the tiny amount of energy and nutrients allocated to DNA – together with the relatively large fraction of non-coding 'junk' DNA in the genomes of most eukaryotes, and the widespread success of polyploid plants – argue against any strong constraint on plasticity imposed by DNA costs. But signal detection and transduction may be a different story. Construction and maintenance of organs, tissues, and/or biochemical pathways required for sensing specific environmental signals (e.g., photoperiod, R:FR ratio, soil humidity) require the allocation of resources far more costly than picograms of DNA. As a consequence of these costs, natural selection should favor an organism's reliance on the fewest, most reliable signals. It seems unlikely, however, that plants constantly re-evolve the capacity to detect particular signals; indeed, the conservation of phytochromes and ethylene receptors across higher plants (see above) argue that a 'tool box' of receptors – and their associated energetic costs – can be considered fixed. More

important in favoring reliance on a few reliable signals may be the fitness costs of using unreliable, uninformative, or misleading signals to guide development.

In any geographic area, a few signals (e.g., photoperiod) may correlate well with the many aspects of the environment to which a plant must be adapted (e.g., temperature, rainfall). Within that area, selection should favor reliance on those signals, favoring the rapid evolution of transcriptional regulators tied to the detectors of those signals, and targeting promoters upstream of regulatory and structural genes required to produce an adaptive phenotype (see *Mechanisms of plasticity*). Because the environment is spatially/spatio-temporally autocorrelated, a system of signal detection and transduction which is adaptive in one microsite is likely to be adaptive in other, similar microsites nearby. In a restricted geographical area, gene flow within and between populations of a given species should tend to homogenize the system of signal detection and transduction used – and hence, the pattern of phenotypic response to environmental variation.

But the interaction of local selection and gene flow can limit the extent to which organisms can adapt to particular microenvironments. Correlations among environmental factors seen in one geographic area or set of environments must ultimately break down or reverse themselves in others. Monthly mean rainfall and temperature are positively correlated with each other and daylength in Wisconsin, for example, while rainfall is negatively correlated with daylength and temperature in California. Growing season length and soil moisture may be positively correlated among alpine sites buried by snow to a particular depth, but negatively correlated among sites buried to different depths. If a plant is to produce an adaptive phenotype across any broad environmental gradient via regulatory control, at some point it must undergo a quantum shift in the signals it uses, not merely the responses to those signals. Gene flow among nearby populations might make such a quantum shift difficult, if not impossible, and – together with the parallel selection pressures for a particular regulatory system in a given landscape of habitats, imposed by the scale of environmental autocorrelation – impose a fundamental constraint on the range of conditions over which a species can show adaptive plasticity. This constraint, I propose, should be a leading factor helping to limit species' distributions along ecological and geographic gradients, and their ability to displace competitors, evade predators, and dominate local ecosystems over evolutionary time scales – and, hence, an issue of deep and abiding interest to ecologists and evolutionary biologists.

Tantalizing support for this view comes from an analysis of the environmental factors correlated with the production of seasonal forms in members of the satyrid butterfly genus *Bicyclus* in subequatorial Africa (Roskam and Brakefield, 1999). The wet-season form has large eyespots and conspicuous

bands, while the dry-season form has a more cryptic coloration. The false eyes appear to involve predator deception during the wet season, when the butterflies actively fly through vegetation, while crypsis prevails during the dry season, when the butterflies mostly rest on dried foliage (Roskam and Brakefield, 1996; Brakefield, 1997). In savannas south of the equator, rainfall and temperature are often positively related to each other and production of the wet-season form – suggesting that both factors (or their correlates) may be serving as signals there. However, in savannas and wet forests north of the equator, rainfall and temperature are negatively related, and only rainfall shows a significant correlation with production of the wet-season form – suggesting that only rainfall is serving as a signal there. These inferences regarding the nature of regulatory control in species of different regions are yet, however, to be tested by experimental manipulation.

Strategies in studying developmental plasticity

The importance of understanding the molecular bases of phenotypic plasticity is threefold. First, detailed information on the fitness benefits and costs of morphs produced by various genotypes in different environments alone is *not* sufficient to predict the genotype(s) favored by natural selection – we also need to understand the underlying genetics (e.g., allelic vs. epistatic relationships among genotypes, effects of a few major loci or numerous loci of small effect) (e.g., see van Tienderen, 1991, 1997; Gabriel and Lynch, 1992; Zhivotovsky *et al.*, 1996). Second, if we are to test the hypothesis just advanced that gene flow and environmental autocorrelation impose a fundamental constraint on the evolution of phenotypic plasticity, then we need to understand the details of the actual regulatory cascade operating in different populations, how they differ from each other as a function of distance and ecological divergence between populations, and how they respond to various environmental signals. More generally, if we are to test the hypothesis that underlying many patterns of phenotypic plasticity are minor modulations of a fixed bauplan (i.e., no "metaplasticity"), then we also need to know the molecular underpinnings of plastic norms of reaction. Finally, knowledge of the molecular bases of phenotypic plasticity is fundamental to understanding exactly how changes in plant form, physiology, and behavior have evolved – through changes in structural or regulatory genes, transcriptional regulators, signal detectors or signal transducers. Understanding these bases is likely to illuminate similar processes underlying the evolution of all aspects of phenotypic diversity on earth, and will require several new forms of collaboration by molecular biologists, geneticists, developmental biologists, ecologists, and evolutionary biologists.

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