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Homoplasy and Developmental Constraint: A Model and an Example from Plants[1]

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SYNOPSIS. Much attention has been paid to the role of developmental information in estimating phylogenetic relationships and, more recently, to the use of phylogenies in understanding the evolution of development. At the moment, however, we lack a sufficiently general theory connecting phylogenetic patterns of character evolution to properties of developmental systems. Here we outline a simple model relating homoplasy to the rate of character change and the number of evolvable states, both of which may reflect developmental constraints. Given a particular rate of character change, the fewer the evolvable states the more homoplasy is expected, and vice versa. The repeated evolution of a limited number of forms of bilateral flower symmetry may reflect constraints imposed by overall flower orientation and underlying mechanisms of differentiation.

INTRODUCTION

Studies of phylogeny and of development have long intersected. Darwin and Haeckel and their contemporaries were concerned mainly with how knowledge of development might inform phylogenetic inference. If ontogeny really did recapitulate phylogeny, then phylogenetic relationships might be determined directly by reference to ontogenetic sequences.

This same interest accompanied the emergence of phylogenetic systematics, where special emphasis was placed on how developmental sequences might be used in establishing the direction, or polarity, of character evolution (*e.g.*, Zimmermann, 1931; Hennig, 1966; see Donoghue and Kadereit, 1992). Debate over the theory and practical efficacy of ontogenetic methods of polarity assessment raged for over a decade in the pages of *Systematic Zoology* and *Cladistics,* touched off largely by Gareth Nelson's paper in 1978 (*e.g.*, Brooks and Wiley, 1985; de Queiroz, 1985; Kluge, 1985; Rieppel, 1985; Mabee, 1989, 1993; also see Weston, 1988, 1994). However, even as clarity was being achieved, the need seemed to dissipate. The widespread use of molecular characters, starting in the mid-1980s, shifted the discussion in the opposite direction. The main emphasis since then has been on how phylogenetic knowledge might be enlisted in understanding the evolution of development (see Fink, 1982, for the conceptual beginnings of this shift).

By now there are many examples in which phylogenetic hypotheses have helped orient developmental studies by suggesting the direction or likelihood of a particular character change, the phylogenetic location and correlates of evolutionary changes in development, the appropriate species for detailed comparison, etc. From plants, the recent work of Kramer and Irish (1999) provides a fine example: recognition that the model organisms *Arabidopsis* and *Antirrhinum* both represent a core eudicot clade within angiosperms motivated their study of expression patterns in B-class floral organ genes outside of core eudicots, in the related ranunculids. It is less obvious, however, whether any more general connection (beyond a list of specific examples) can be made between phylogenetic patterns and developmental systems.

Here we focus on homoplasy, which phylogenetic analyses reveal and allow us to quantify, and on how levels of homoplasy

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might relate to underlying developmental phenomena. Our primary aim is to highlight several parameters that will be critical to the development of a general theory connecting homoplasy and development, and to formulate a simple model relating these parameters to one another. Why do we wish to do this? It is *not* because we think that such an exercise will have an especially important bearing on phylogenetic inference itself. In fact, as argued elsewhere (Sanderson and Donoghue, 1996), the level of homoplasy in real datasets is not a very good predictor of confidence in phylogenetic inferences. Instead, we are motivated by the sense that evolutionary theory is incomplete if it fails to explain why some characters show more homoplasy than others. Evolutionary biologists should, we think, be able to make reasonably accurate *a priori* predictions about the behavior of particular characters, or classes of characters, and for this purpose some general theory is needed.

Such questions have already received some thoughtful attention. It is especially appropriate to single out Rupert Reidl, for whom such issues were of paramount importance (*e.g.*, Reidl, 1978). More recently, Pere Alberch (*e.g.*, 1982), Wallace Arthur (*e.g.*, 1988), Stuart Kaufman (*e.g.*, 1993), Daniel McShea (*e.g.*, 1996), Gunter Wagner (*e.g.*, 1989), David Wake (*e.g.*, 1991), and William Wimsatt (*e.g.*, 1986) have made noteworthy contributions to the development of a general theory along these lines. Unfortunately, however, this issue has seldom found its way into mainstream evolutionary literature, where the recurrence of form is instead generally treated as a collection of examples about which generalizations are unlikely or unnecessary. Ironically, Simon Conway Morris (1998, p. 219), whose main argument against Stephen Jay Gould's ''Wonderful Life'' (1989) rested on the phenomenon of convergence, expressed this outlook quite directly: ''I am not aware of a single synthesis, and perhaps one is not really necessary: afterall rampant convergence is not in dispute and piling up example after example might exhaust rather than instruct.'' While he certainly is right to note the absence of a general treatment, we think he is wrong about the desirability of a synthesis and the prospects for a more general theory.

BACKGROUND

Homoplasy and its measurement

Two (or more) conditions are considered states of the same character when one of the states is thought to have given rise to the other(s) during the evolution of the group, or that they were derived from some other state present in a common ancestor (transformational homology; see Donoghue, 1992). Likewise, each state of a character is assumed at the outset to be homologous in all taxa assigned that state, in the sense of having been retained from a common ancestor in which it evolved. For a character with two states (for example) the simplest assumption is that just one change of state (or step) occurred during the evolution of the group. However, a particular phylogenetic hypothesis (*e.g.*, derived from all of the characters under consideration) may entail extra state changes in one or more characters. The term homoplasy refers to such extra steps (see Sanderson and Hufford, 1996). Homoplasy is often said to encompass convergence, parallelism, and reversal. However, it is common (see Patterson, 1982) to distinguish convergence (analogous similarities that do not pass initial tests of homology and, therefore, are not included in phylogenetic datasets) from parallelism (independent origination, revealed via phylogenetic analysis, of a condition that did pass initial homology tests).

Homoplasy can be quantified using a variety of indices, the most popular of which has been the Consistency Index (CI; Kluge and Farris, 1969; see Archie, 1996). CI is the minimum number of state changes possible for a character, or an entire set of characters, divided by the number of state changes reconstructed as having occurred on a particular tree. As the number of extra steps increases, CI decreases. Other measures take into account additional factors, such as the maximum number of state changes possible (*e.g.*, Farris, 1989; Meier *et al.*, 1991; see Archie, 1996). The behavior of these indices has been studied using

both real datasets and simulations (see below), and it is clear that comparisons using any of them may be complicated by a variety of correlated factors and that no one measure is appropriate for all purposes (see Goloboff, 1991).

Levels of homoplasy

What do we know about levels of homoplasy? In general, as the number of taxa increases, so does the amount of homoplasy (Sanderson and Donoghue, 1989, 1996; Archie, 1989, 1996; Klassen *et al.*, 1991; Givnish and Sytsma, 1997*a*, *b*; Hauser and Boyajian, 1997). This reflects the fact that it is impossible to see very high levels of homoplasy in parsimony analyses of a small number of taxa and that more branches mean more opportunities (under parsimony) for character changes to occur (Sanderson and Donoghue, 1989; Goloboff, 1991; Klassen *et al.*, 1991). Correlations between CI and the number of characters or the taxonomic rank of the taxa in an analysis are generally not significant in meta-analyses of real datasets, though stronger relationships have been reported in simulations (*e.g.*, Klassen *et al.*, 1991; Archie, 1996; Givnish and Sytsma, 1997*a*).

Bearing in mind the correlation of CI with number of taxa, Sanderson and Donoghue (1989) compared levels of homoplasy in plant versus animal datasets, and in morphological versus molecular data, and did not find significant differences in either case (also see Donoghue and Sanderson, 1992). When many more molecular studies were included they found that morphological and DNA sequence datasets showed about the same levels of homoplasy, but that restriction fragment studies (which entailed mapping of restriction sites) tended to show less (Sanderson and Donoghue, 1996). Givnish and Sytsma (1997*b*) and Jansen *et al.* (1998) obtained similar results: restriction site studies generally showed less homoplasy than DNA sequence or morphological studies. Givnish and Sytsma (1997*b*) also reported a slight but significant difference between morphology and DNA sequences, and concluded that, in general, molecular data show less homoplasy than morphological data. However, when the 50 plant morphological datasets from Sanderson and Donoghue (1996; CI's recalculated with autapomorphies excluded) are added to their dataset the difference between morphology and sequences disappears (M. Donoghue, unpublished).

Several other contrasts have been analyzed. For example, Donoghue and Sanderson (1994) subdivided ten flowering plant morphological datasets into pubescence characters, leaf characters, and flower characters, and found similar levels of homoplasy across the three classes. de Quieroz and Wimberger (1993) compared morphological versus behavioral datasets (and morphological versus behavioral characters within datasets) and found little difference in homoplasy between these. Foster *et al.* (1996), on the other hand, compared display and non-display behavioral traits across eight animal datasets and suggested that display characters may show less homoplasy than non-display characters.

Most studies have focused on characters in which variation is coded as discrete states (*e.g.*, presence or absence of a morphological structure or a restriction site), but some attention has also been given to levels of homoplasy in continuously varying characters. Ackerly and Donoghue (1998) formulated a continuous-character analog of the Retention Index (Farris, 1989; see Archie, 1996), which they called the Quantitative Convergence Index (QVI). Application of this measure to a set of leaf and plant architectural characters in maples (*Acer*) revealed exceptionally high levels of homoplasy in most of these traits (Ackerly and Donoghue, 1998). It is important to note, however, that they measured homoplasy only in characters that were mapped onto the phylogeny after the fact (and which might not have passed initial similarity tests), as opposed to characters that were included in the analysis from the outset (as in the meta-analyses discussed above). In general, the former are expected to show more homoplasy than the latter.

A SIMPLE MODEL

The most obvious finding of the studies reviewed above is that there is great variation in homoplasy among characters within

any of the classes of characters examined. Some characters undergo little change and show little homoplasy; others in the same datasets undergo many changes and show high levels of homoplasy. Can we identify parameters that will partition any set of characters into those with high levels of homoplasy and those with low levels? We could, of course, continue to examine standard contrasts (*e.g.*, molecules versus morphology) in the hope of eventually identifying significant differences from which generalizations could be drawn. Alternatively, we could develop a model from which some predictions could be derived. What follows is an attempt along these lines.

Given a particular tree (*i.e.*, holding the number of taxa, tree shape, and branch lengths constant), we expect lower rates of character change to yield lower levels of homoplasy than higher rates. However, the outcome depends on another variable, namely the number of evolvable states of a character. If the number of states were unlimited, then each change (no matter how many actually occured) might lead to a different state, and we would see little or no recurrence. In contrast, if the number of states were strictly limited (*e.g.*, to two alternative states), then a high rate of change, over a large enough tree, guarantees a high level of homoplasy.

These simple expectations are borne out by a number of observations. For example, the connection between rate and homoplasy was verified in a simulation by Archie (1996) and, indirectly, in a simulation that examined levels of homoplasy as a function of the percentage of the characters that changed along each branch of a tree (Hauser and Boyajian, 1997). Results obtained by Ackerly and Donoghue (1998) for continuously varying characters are consistent with the predictions relating rate and number of evolvable states to homoplasy. In a system with (in effect) an unlimited number of states they found no correlation between the amount/rate of character change and level of homoplasy.

In practice, rate of character change and number of states may be correlated. If rates are very low then few states will be observed, even if, in theory, there were many evolvable states. Likewise, high rates of change might tend to result in the evolution of more states. Under these circumstances, low rate characters with few states might exhibit about the same amount of homoplasy as high rate characters with more states. There are, however, other possibilities. Importantly, a high rate of change does not guarantee the evolution of more states, because this may be limited for developmental or functional reason. If such constraints existed, then a high rate of change would lead instead to toggling back and forth between the few possible states, and hence to high levels of homoplasy.

To explore these relationships further we carried out a set of computer simulations. Given an evolutionary rate (probability of change), μ, a character C with N states, and a phylogeny with root node R:

(1) R was initialized as state 0.

(2) Descendant nodes of R were traversed recursively, with the following procedure followed for each node D: the probability μ was evaluated to determine whether a character state change occurred along the branch leading from D's direct ancestor. If a change did occur, the state assigned to D was drawn from a uniform distribution of all possible states of C (*i.e.*, characters had a 1/N chance of changing to the same state). If a change did not occur, then D was assigned the same state as its immediate ancestor.

(3) The consistency index was calculated for C, and we took the inverse of this value as the measure of homoplasy.

We conducted simulations on a pectinate (comb-like) 16-taxon tree over a range of evolutionary rates (mutation probability) and evolvable character states, and calculated average homoplasy from 1,000 simulated characters for each combination of variables. The results are shown in Figure 1.

As expected, homoplasy shows a negative relationship with number of character states for all values of evolutionary rate, and a positive relationship with rate for every number of evolvable states. Experiments with completely symmetrical bifurcating trees yielded similar results. The ef-

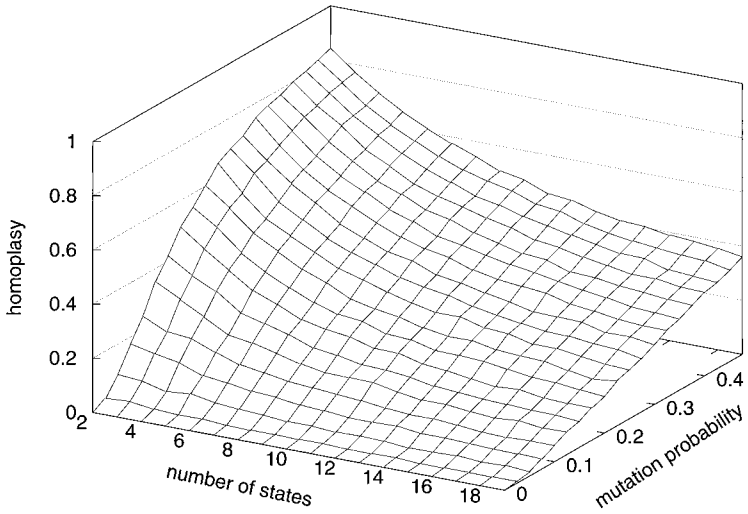

FIG. 1. Results of simulations of character evolution on a pectinate (comb-like) phylogeny with 16 terminal nodes, showing the relationship between homoplasy (measured as one minus the consistency index, averaged over 1,000 characters), the number of evolvable character states, and the probability of mutation (character state change) along internal branches. Homoplasy increases with the probability of change and decreases with the number of evolvable character states.

fect of using trees with more taxa is to shift the surface to the right along the x-axis; a larger tree allows more branches along which changes can occur, and therefore will, on average, show more homoplasy for any particular combination of rate and number of states.

This model suggests a variety of tests using real datasets, but these are more difficult than one might suppose. On the surface one could compare levels of homoplasy in characters with more versus fewer states, such as restriction site characters with two states, nucleotide characters with four states, and amino acid characters with twenty. The difficulty in such comparisons is that the number of evolvable states (the critical parameter in our model) may be far fewer than the maximum number possible in theory, owing to developmental or functional constraints (see below). This is perhaps most obvious in molecular data, where, for example, transitions are typically favored over transversions, and substitutions of amino acids with similar properties (hydrophobicity, etc.) are favored over those with very different properties. An alternative would be to view the number of observed states of a character (within or across datasets) as reflecting constraint, and see how well this predicts homoplasy. Homoplasy comparisons among classes of characters that differ in rate of change would be instructive, though this may often be confounded by the rate/number of states correlation. Based on rate differences among codon positions, for example, one might expect the most homoplasy at 3rd sites. The fact that this is not always the case (*e.g.*, Olmstead *et al.*, 1998; Sennblad and Bremer, 2000) may reflect severe constraints on the acceptable substitutions at first and second sites. That is, although there may be fewer changes at 1st and 2nd sites, there may also be more homoplasy owing to functional constraints on the number of states.

LIMITS ON THE NUMBER OF STATES

The model presented above highlights the number of states of a character as an important predictor of levels of homoplasy. What, then, determines the number of states? Unfortunately, there are several possibilities, and in real cases these may be difficult to distinguish.

One possibility is that the number of states is largely an artifact of taxonomic practice and/or the level of resolution of

particular methods. Initial homology criteria are not applied with the same level of stringency in all cases, depending on available evidence (see Donoghue and Sanderson, 1994). In morphology, for example, relevant details of anatomy and development are often lacking, and decisions about states are based then on superficial appearances. In general, this results in the recognition of fewer states, and hence increased homoplasy. If additional data were available, differences between conditions initially scored as being the same might become apparent, leading to the recognition of additional states. It is common, in fact, for authors to highlight, after the fact, subtle differences among states discovered to have arisen independently (*e.g.*, see discussion of *Viburnum* fruit color in Baldwin *et al.*, 1995). Related to this point is the desire (seldom made explicit) to recognize fewer states for morphological characters in order to obtain a reasonably well-resolved hypothesis of relationships. Afterall, if every taxon were scored as having a separate state, based on subtle differences, the character would lose its power to resolve phylogenetic relationships. This tendency to ''lump'' may result in errors, perhaps especially in cases of reduction (*e.g.*, see discussion of megaspore walls in Gnetales and angiosperms in Donoghue and Doyle, 2000).

Restriction enzyme analyses provide an example of a limitation on the number of states imposed by the method of detection. Restriction sites are scored as being present or absent, but losses of a site might occur by changes in any of the constituent nucleotides (DeBry and Slade, 1985). If we could compare the underlying sequences directly, we might find that different nucleotide substitutions accounted for the loss of a site in different taxa, and score them as having different states. On the surface, presence–absence scoring should increase the level of homoplasy, yet, as noted above, restriction site data tend to show high CI's. The reason for this is still unclear, but it is worth noting that all nucleotide positions within a restriction site are probably not equally free to vary (owing to functional constraints), so that presence–absence scoring might relatively accurately reflect changes in just one or two nucleotides within the site. Furthermore, restriction site mapping becomes untenable when levels of divergence are too great, so that such studies have a built-in mechanism for excluding characters that might otherwise inflate the level of homoplasy. We suspect that much higher levels of divergence are tolerated in molecular sequence analyses and in morphology than in restriction site studies.

While it is certain that methodological artifacts do exist, and may in part be responsible for the numbers of states that are recognized, it is equally certain that this is not the whole story. There are several obvious and presumably powerful forces that limit the number of states observed to a subset of those that are theoretically attainable (Wake, 1991; also see Meyer, 1999, on the ''re-awakening'' of developmental mechanisms; and Wagner, 2000, on the ''exhaustion'' of morphological character states). One of these is selection on function. Some states are not seen simply because they are relatively disadvantageous and are weeded out. Other states may arise frequently, being accessible adaptations to particular circumstances. Properties of developmental systems also might bias or channel the variability that emerges from the process, rendering some outcomes more likely than others (see Maynard Smith *et al.*, 1985). Even if developmental systems were infinitely malleable, and any state could eventually be achieved, the existence of a particular developmental system at a particular point in phylogeny renders some changes more likely than others. In the next section we provide a concrete example of what we believe to be a developmental constraint on the number of states and hence the level of homoplasy.

A PLANT EXAMPLE

Recently, we analyzed the evolution of flower symmetry using a large composite tree of asterid angiosperms (Donoghue *et al.*, 1998; Ree and Donoghue, 1999), a group of some 65,000 species that includes most of the flowering plants with sympetalous corollas (petals fused into a tube). We concluded (see Fig. 2) that the first asterids

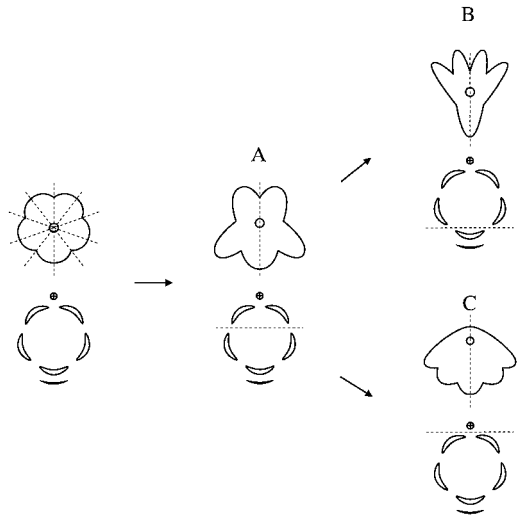

FIG. 2. Possible evolutionary paths from a radially symmetrical (actinomorphic) flower in the inferred ancestor of asterid angiosperms (shown at left) to the most common forms of bilaterally symmetrical (zygomorphic) flowers found among Asteridae. Each flower is depicted as a pair of diagrams: an outline of the corolla, viewed face-on (above), and a floral diagram showing the orientation of petals relative to the stem (above) and a subtending bract (below). Dashed lines on upper diagrams indicate axes of symmetry and those on the lower diagrams show planes of greatest dorso-ventral differentiation. Given a flower with five petals, a medially positioned ventral petal, and a single dorso-ventral axis of symmetry, three basic forms of bilateral symmetry are possible: (A) two dorsally oriented petals and three ventrally oriented petals (2:3 pattern); (B) four dorsal petals and a single ventral petal (4:1 pattern); and (C) all five petals ventrally oriented (0:5 pattern).

probably had radially symmetrical (actinomorphic, or polysymmetric) corollas, and that bilaterally symmetrical (zygomorphic, or monosymmetric) corollas evolved at least eight times independently, and were then secondarily lost at least nine times within bilaterally symmetrical clades (especially within the lamiid clade that includes the snapdragons, mints, and their relatives). These results are robust over a range of weights applied to changes in the two directions (Donoghue *et al.*, 1998; Ree and Donoghue, 1998). Furthermore, maximum likelihood analyses, taken at face value, suggest that transitions from a bilateral to a radial corolla are more likely than changes in the other direction (Ree and Donoghue, 1999).

Within each of the major bilaterally symmetrical clades, it appears that there have been frequent changes in the form of zygomorphy (Donoghue *et al.*, 1998). In the most common pattern, two upper (dorsal, adaxial) petals are differentiated from three lower (ventral, abaxial) petals (Fig. 2A). We refer to this as the 2:3 pattern. The 4:1 pattern features four upper and one lower petal (Fig. 2B), and in the 0:5 form all five petals are shifted to the lower side of the flower (Fig. 2C). Ignoring for the moment a few very important exceptions (see below), these three forms have each evolved repeatedly, while other readily imaginable forms (3:2, 1:4, etc.) are not encountered. Why do we see the recurrence of certain forms and not others?

We suggest that a large part of the explanation relates to the nature of flower development in asterids. Ancestrally, and most commonly, asterid flowers are oriented such that, from the inception of organ primordia on the flower meristem, there are five petals, one of which is in a medial position on the lower (ventral, abaxial) side of the flower (all flowers in Fig. 2). As long as this arrangement is maintained, and given an underlying mechanism for dorso-ventral differentiation, then only the three commonly observed forms of bilateral symmetry (2:3, 4:1, 0:5) are possible (Fig. 2). Given this limitation on the number of states, changes in the form of symmetry (for whatever reason) have resulted in considerable homoplasy.

Regarding developmental mechanisms underlying these patterns, we know a great deal about one 2:3 flower, the common snapdragon (*Anthirrinum majus*), thanks to the work of Enrico Coen and colleagues (*e.g.*, Coen, 1996; Lou *et al.*, 1996). In this case normal development entails expression of a gene called CYCLOIDEA (among several others) in the dorsal, but not in the ventral, region of the flower primordium. It is possible therefore, that evolutionary shifts in the form of bilateral symmetry have entailed changes in the zone of CYCLOIDEA expression (*e.g.*, over a larger region of the primordium in 4:1 flowers than in 2:3 flowers), and the effects of this on downstream genes (Donoghue *et al.*, 1998). This is

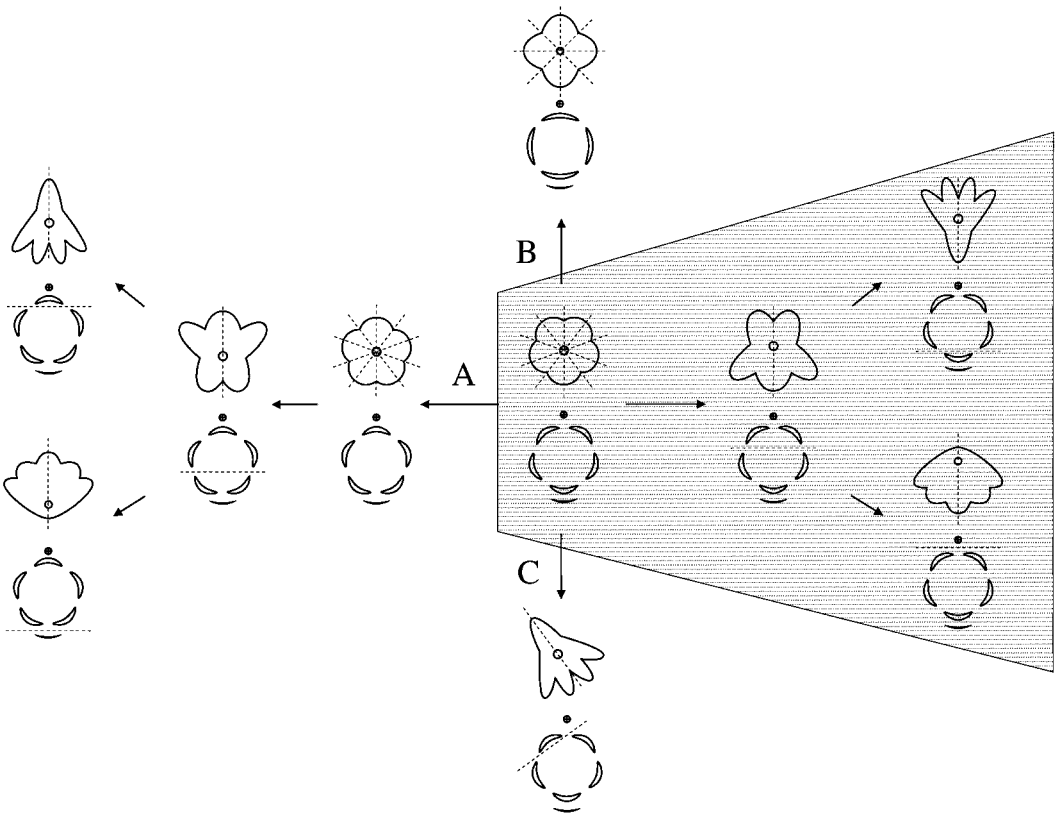

FIG. 3. Examples of evolutionary modifications of the ancestral flower form in asterid angiosperms (other modifications are thought to have occurred, but are not shown here). The shaded area represents the common transitions, as shown in Figure 1. (A) A 180° shift in the position of petal primordia on the meristem results in a medially positioned dorsal petal and allows the evolution of 3:2, 1:4, and 5:0 forms of bilateral symmetry. Other shifts away from the ancestral condition include: (B) reduction in the number of petals (derived from bilaterally symmetrical flowers in some lineages, as in *Plantago*; see text); and (C) assymetry, with differentiation along an axis oblique to the dorso-ventral axis (derived from bilaterally symmetrical flowers in some lineages, as in *Centranthus*; see text).

strictly conjecture, however, as the appropriate comparisons have not yet been made.

There are several potentially independent elements of the underlying system (petal number, dorso-ventral differentiation, flower orientation), and modification of any one of these opens up the possibility of new forms of bilateral symmetry (Fig. 3). Perhaps most obviously, there could be a change in the number of petals (*e.g.*, a reduction to four petals in Fig. 3B), which would open up possibilities such as 1:3,3:1, etc. In asterids, shifts from five to four petals have occurred on a number of occasions, some of these within radially symmetrical clades (*e.g.*, as in Rubiaceae). In some cases a change from five to four has accompanied a shift from bilateral to radial symmetry, as in the common plantain (*Plantago*) within ''Scrophulariaceae'' (Donoghue *et al.*, 1998; Reeves and Olmstead, 1998; Endress, 1998). Another possibility is a shift from strictly dorso-ventral differentiation of the flower primordium to a mechanism yielding assymetrical flowers (as in Fig. 3C). This has seldom occurred within asterids, but there are several documented cases, such as in *Centranthus* within Valerianaceae (Endress, 1999). In this case, differentiation of one of the two dorsal petals from the other four petals yields what appears to be a 1:4 pattern.

However, perhaps the most interesting and common ''escape'' from the typical asterid syndrome involves a 180° reorientation, such that the medial petal is in the dorsal position from the inception of organ differentiation (Fig. 3A). Such a shift occurred within the ericad lineage (Ericaceae), and where bilateral flowers have evolved within this group, as within the familiar *Rhododendron*, we encounter the unfamiliar 3:2 flower form. Similarly, but quite independently, in the lobelia lineage (Lobeliaceae) we see the same 180° shift in underlying orientation, and again encounter 3:2 flowers, as in *Nemacladus* and its relatives. Curiously, in many lobeliads the flowers undergo ''resupination,'' twisting 180° much later in development (after the flowers are well formed). Consequently, most lobeliad flowers, as they are eventually encountered by pollinators, have reverted to the common 2:3 orientation. This phenomenon suggests that selection for the typical asterid arrangements may also play an important role in limiting the number of states.

The point of this example is that as long as typical asterid flower development is maintained, a limited number of forms of zygomorphy are possible, and other forms are effectively off limits. We see toggling between the standard 2:3, 4:1, and 0:5 patterns because overall orientation and mechanisms of dorso-ventral differentiation, which are established very early in flower development, appear to be more conserved than down-stream features, including overall symmetry and, in particular, the forms of bilateral symmetry.

This is most certainly not to say, however, that the underlying constraints in this system (petal number, dorso-ventral differentiation, flower orientation) are absolute. In fact, as we have emphasized, each of these characters has undergone change within asterids (and elsewhere in angiosperms). Breakdown of any one of the constraining factors renders the standard forms of bilateral symmetry off limit, but opens up a new set of possible forms. As emphasized in Figure 3, a 180° change in flower orientation in several clades made possible 3:2, 1:4, and 5:0 flower forms.

CONCLUSIONS

Activity at the intersection of phylogenetic systematics and developmental biology has shifted over the years. The old problem of inferring character polarity from ontogenetic sequences has faded into the background as phylogenetic attention has increasingly focused on the analysis of molecular data. Emphasis is now placed on how knowledge of phylogeny can be put to use in understanding the evolution of development. At this point, however, general theory at this intersection is rather limited. In particular, although properties of developmental systems are presumably in some way responsible for observed patterns of homoplasy, we still lack a predictive theory relating these phenomena. Our hope is to draw attention to the need for such a theory. In particular, we have highlighted the need to better understand how the rate of character change and limitations on the number of evolvable states interact in determining levels of homoplasy.

A variety of factors bear on the number of states entered into phylogenetic analyses, including artifacts of taxonomic practice and methods of analysis. Ultimately, however, selection on function and properties of developmental systems place bounds on the number of realized states. Wake (1991) argued, with examples from salamanders, that developmental constraint was a primary cause of homoplasy. We suggest that the same is true in plants. The flower symmetry case highlighted here provides one example, but we suppose there are many others. For instance, constraints related to the individuation of floral organs and to the timing of developmental events may help explain characteristic changes in the number of flower parts (*e.g.*, repeated addition and subtract of whole sets of stamens). To whatever degree developmental systems place limits on character evolution, we have argued that they will influence patterns in homoplasy. Conversely, the analysis of homoplasy will illuminate our understanding of development.

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