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Parallelism in Flower Evolution and Development

Carolyn A. Wessinger[1] and Lena C. Hileman[2]

[1]Department of Biological Sciences, University of South Carolina, Columbia, South Carolina 29208, USA

[2]Department of Ecology and Evolutionary Biology, University of Kansas, Lawrence, Kansas 66045, USA; email: lhileman@ku.edu

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Abstract

Flower evolution is characterized by widespread repetition, with adaptations to pollinator environment evolving in parallel. Recent studies have expanded our understanding of the developmental basis of adaptive floral novelties—petal fusion, bilateral symmetry, heterostyly, and floral dimensions. In this article, we describe patterns of trait evolution and review developmental genetic mechanisms underlying floral novelties. We discuss the diversity of mechanisms for parallel adaptation, the evidence for constraints on these mechanisms, and how constraints help explain observed macroevolutionary patterns. We describe parallel evolution resulting from similarities at multiple hierarchical levels—genetic, developmental, morphological, functional—which indicate general principles in floral evolution, including the central role of hormone signaling. An emerging pattern is mutational bias that may contribute to rapid patterns of parallel evolution, especially if the derived trait can result from simple degenerative mutations. We argue that such mutational bias may be less likely to govern the evolution of novelties patterned by complex developmental pathways.

1. INTRODUCTION

Angiosperms (flowering plants) began diversifying on the order of 140 million years ago (reviewed in Sauquet & Magallón 2018), and the diversity of flower form among extant species today is breathtaking. Current floral diversity reflects evolutionary optimization of reproductive output under variable environmental conditions. Reproductive success has been optimized through shifts in mating system, including shifts in biotic (animal) and abiotic (wind, water) pollination strategies (Barrett 2002, Stebbins 1970). Since the beginning of flowering plant diversification, much of the evolution of flowers has been linked to biotic pollination (Gottsberger 2016, Hu et al. 2008), and more than 85% of current species utilize animals for pollination services (Ollerton et al. 2011). Therefore, adaptive floral evolution that facilitates shifts to available pollinators, and enhances pollen transfer when animals visit, is ubiquitous (Faegri & van der Pijl 1979, Fenster et al. 2004, Ollerton et al. 2009).

Adaptive floral evolution has resulted in massive floral trait convergence and parallelism that reveal repeated adaptive trait evolution in response to similar pollinator environments. For the most part, the repeatedly evolved traits discussed in this review are termed parallelisms (although similarities derived from different floral organs, e.g., some repeated origins of nectar spurs, may be more accurately described as convergences). Parallel floral trait evolution reflects developmental changes that increase complexity from the relatively simple ancestral angiosperm flower, followed in some cases by reversals in complexity. This review describes patterns of parallelism and developmental processes associated with transitions toward flower complexity, including sympetaly (petal fusion), bilateral flower symmetry, and initiation of nectar spurs and heterostyly (pollen- and ovule-bearing reproductive organs of different lengths to reduce self-pollination), as well as quantitative changes that enhance pollen transfer, including evolutionary changes in floral tube and nectar spur lengths.

Our understanding of floral trait parallelism has been facilitated by advances in the flowering plant phylogeny onto which floral traits are now being extensively mapped, revealing patterns of parallel trait evolution (e.g., Sauquet et al. 2017, Wessinger et al. 2019, Wu et al. 2018, Zhong et al. 2019). At the same time, recent research has led to an unprecedented understanding of the developmental and genetic processes that shape diverse aspects of flower form (reviewed here and in Kramer 2019, Moyroud & Glover 2017, Smyth 2018, Woźniak & Sicard 2018). Through integration, we can begin to identify biologically meaningful connections between patterns of trait evolution and the developmental genetic processes that shape those traits. Our goal is to begin answering three fundamental questions of floral trait evolution.

First, to what extent do repeatedly evolved floral traits utilize similar developmental and genetic processes? Analogous to Abouheif's (1997) hierarchical approach to integrating morphology with development and genes when considering trait homology, we consider trait parallelism in the same hierarchical context. Parallelism can be identified in flower function (e.g., transitions to a specific pollinator), morphology (e.g., transitions to similar organ dimensions), development (e.g., transitions via similar cellular processes), genetic pathways, genes, and specific causal mutations. We review examples in which repeated floral trait evolution is coupled with parallel or divergent developmental and genetic processes, highlighting the utility of a hierarchical approach.

Second, what constraints direct floral trait evolution to follow parallel developmental genetic paths? Our review of a subset of well-studied floral traits emphasizes the fact that nearly all flower diversification requires one or both of the following processes: (*a*) hormone signaling (usually auxin) to initiate patterns of cell proliferation and (*b*) modifications to patterns of cell division and/or cell expansion to achieve adaptive variation in floral organ dimensions. The gene regulatory networks that affect these processes are extremely complex, and divergent genetic changes are

often employed. Yet, despite diverse and often divergent genetic mechanisms for parallel trait evolution, we identify some similarities at the level of hormone signaling.

Third, are the patterns of floral trait evolution and genetic processes underlying trait evolution reciprocally illuminating? We discuss whether and how patterns of trait evolution are likely shaped by genetic mechanisms, such that a given pattern of trait evolution points to specific genetic mechanisms, and by extension, whether we may predict patterns of trait evolution from descriptions of genetic mechanisms. This concept seems to apply to traits produced by relatively simple genetic mechanisms. Evaluating whether this idea holds for floral traits produced through complex genetic pathways will require additional insights into both trait evolution and development.

2. MACROEVOLUTIONARY PATTERNS OF FLORAL TRAIT EVOLUTION

The flower itself is a complex novelty of plant evolution (Friedman 2009). The ancestral flower was likely quite simple compared with the flower complexity we see among extant species. The ancestral form is predicted to have been bisexual, with unfused sterile perianth organs of similar shape (radial symmetry) surrounding unfused reproductive organs (Sauquet et al. 2017). This represents the common ground plan that is retained in some lineages, such as the family Nymphaceae (water lilies and relatives), and on which all subsequent floral trait evolution is based (Smyth 2018). An emerging theme is the parallel evolution of a more complex floral form from this simpler ancestral condition. Many parallel transitions toward increased complexity occur at a broad taxonomic scale and characterize major flowering plant lineages. These include evolutionary transitions from free to fused floral organs and from radially to bilaterally symmetrical flowers, as well as the origins of nectar spurs and heterostyly.

Floral organ fusion has given rise to a diversity of specialized floral traits. The most elaborate of these is arguably the specialized pollinaria of orchids and milkweeds, which facilitate precise pollen movement between flowers. Pollinaria are derived from fusion between stamens and pistils, two different floral organ types (i.e., adnation). Examples of fusion between the same type of floral organ (i.e., connation) are common. For example, carpel-to-carpel fusion, leading to a single syncarpous ovary, evolved early in diversification of eudicots and monocots. Sympetaly has evolved multiple times (Reyes et al. 2018, Stull et al. 2018, Zhong & Preston 2015), leading to corolla tubes and keel petals (**Figure 1*a*,*b***), which define major flowering plant lineages such as Lamiales (snapdragon, sages, and relatives), Fabaceae (peas, beans, and relatives), Polygalaceae (milkworts and relatives), and Zingiberales (banana, bird of paradise, and relatives).

During the diversification of flowering plants, bilateral flower symmetry has evolved well over 100 times from an ancestral condition of radial symmetry (Reyes et al. 2016). These transitions represent the evolution of additional complexity, where the basic floral plan is elaborated to include distinct developmental fates for dorsal and ventral sides of flowers (**Figure 2*a*,*b***). Similar to organ fusion, the evolution of bilateral flower symmetry defines major lineages of flowering plants (e.g., Fabaceae, Lamiales, Zingiberales, and the orchid family, Orchidaceae).

Nectar spurs are tubular outgrowths of (usually) petal tissue that hold nectar for visiting pollinators (**Figure 3*a***). These novel structures represent evolutionary complexity, since spurs represent a local region of differentiated petal tissue with a novel developmental fate. Unlike sympetaly and symmetry, nectar spurs do not define major lineages, but they have evolved many times during flowering plant diversification and are well studied in multiple groups, for example, *Aquilegia* (columbine), *Delphinium* (larkspur), *Linaria* (toadflax), and *Pelargonium* (Cullen et al. 2018, Hodges 1997, Jabbour & Renner 2012, Puzey et al. 2012, Tsai et al. 2018).

Sympetaly, bilateral symmetry, and nectar spurs all function to filter pollinators with specific morphologies and to improve conspecific pollen transfer efficiency (Armbruster 2014, Endress

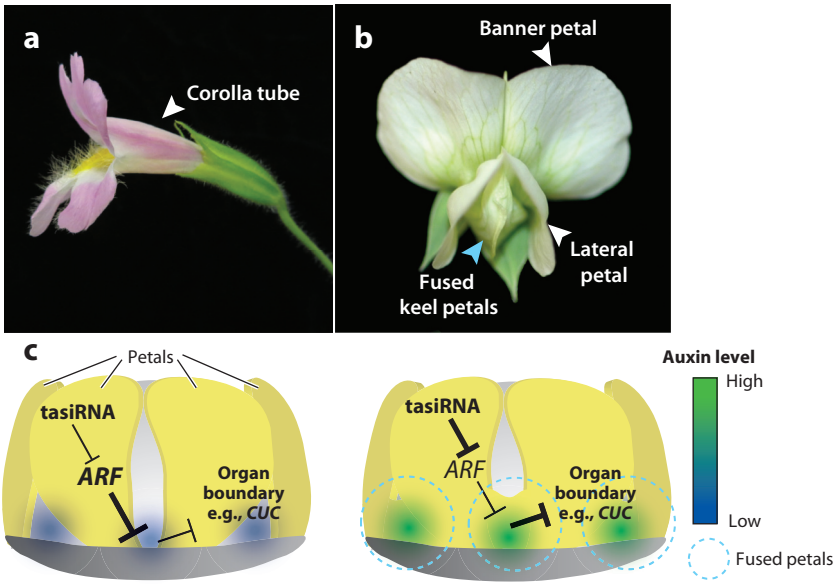

Figure 1

Examples and hypothesized developmental basis of sympetaly. (*a*) *Mimulus lewisii* flower with corolla tube formed by congenital petal fusion (*white arrowhead*). (*b*) *Pisum sativum* flower with keeled ventral petals formed by postgenital fusion (*blue arrowhead*). (*c*) A model for the developmental basis of petal fusion (Ding et al. 2018), whereby variation in regulation of the organ polarity program [e.g., *AUXIN RESPONSE FACTOR* (*ARF*)] regulated by *trans*-acting small interfering RNAs (tasiRNAs) affects interpetal levels of auxin (high levels in *green*, low levels in *blue*). High auxin levels are hypothesized to promote interpetal cell proliferation and negatively regulate the organ boundary genetic program [e.g., *CUPULIFORMIS* (*CUP*)].

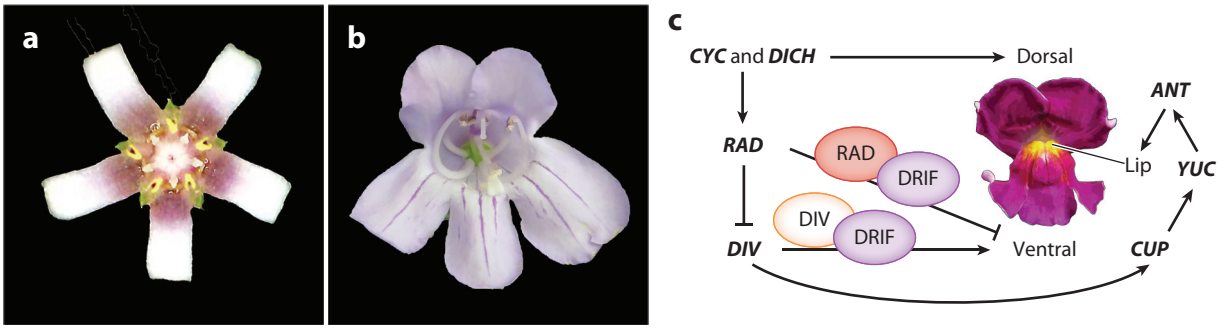

Figure 2

Examples of flower symmetry and the developmental basis of bilateral symmetry in *Antirrhinum majus* (snapdragon). (*a*) Radially symmetrical flower of *Crassula exilis*. (*b*) Bilaterally symmetrical flower of *Penstemon virgatus*. (*c*) The developmental program that regulates bilateral symmetry in snapdragon. *CYCLOIDEA* (*CYC*), *DICHOTOMA* (*DICH*), and *RADIALIS* (*RAD*) determine dorsal flower development. *DIVARICATA* (*DIV*) determines ventral flower development. Ventral identity is precluded from the dorsal side by RAD protein competitively excluding DIV from interacting with DIVARICATA RADIALIS INTERACTING FACTORs (DRIFs). *DIV* specifically affects ventral lip development by putatively altering cell proliferation at the site of curvature through regulation of *CUPULIFORMIS* (*CUP*), *YUCCA* (*YUC*), and *AINTEGUMENTA* (*ANT*).

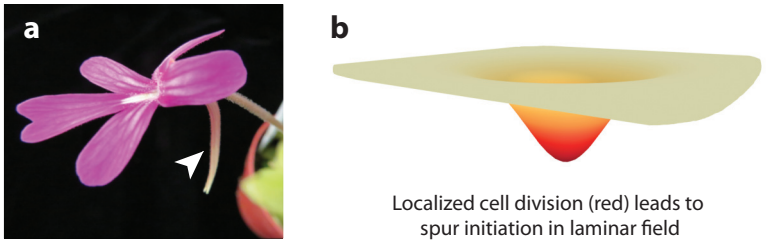

Figure 3

Nectar spur initiation. (*a*) *Pinguicula* sp. (butterwort) with a petal-derived spur (*arrowhead*). (*b*) Nectar spurs, regardless of tissue origin, require a localized zone of cell proliferation to initiate outgrowth (*red area*). Elevated auxin and meristem identity have each been implicated in establishing a focal region of cell proliferation.

2001, Fenster et al. 2004, Stebbins 1970, Thomson & Wilson 2008). Therefore, each of these traits is considered to be an adaptation to maximize outcross mating success. The advantages of outcrossing include the maintenance of heterozygosity, often associated with increased relative fitness, and the avoidance of inbreeding depression (Darwin 1876, Husband & Schemske 1996).

Other evolutionary trends toward greater floral complexity involve the evolution of developmental polymorphisms, in which alternative developmental fates, controlled by genetic polymorphism, are expressed in different individuals. Examples include the evolution of heterostyly and dioecy (separate sexes). In heterostyly, genotypic variation at causal loci controls alternate spatial arrangements of anthers and stigmas that reduce self-pollination and reinforce outcrossing (**Figure 4**). Heterostyly occurs in at least 28 families and is thought to have arisen at least 23 independent times (Barrett 2002, Barrett et al. 2009, Naiki 2012). Reciprocal placement of

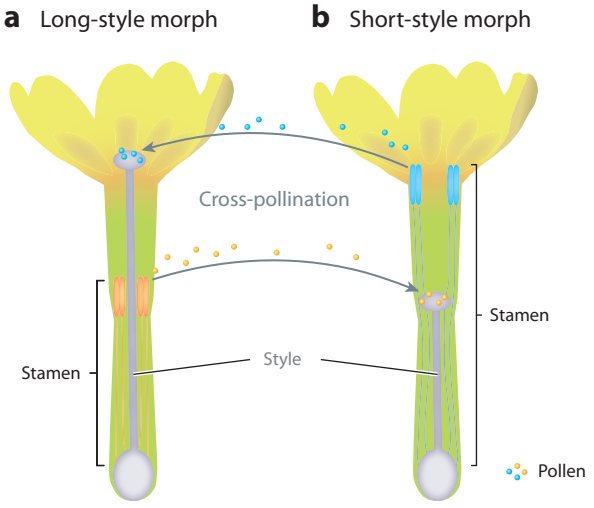

Figure 4

Heterostyly dimorphism. Allelic variation across tightly linked genes at a single locus determines stamen and style length dimorphism in heterostylous species. (*a*) L-morph flowers have long styles and short stamens. (*b*) S-morph flowers have short styles and long stamens. These alternative arrangements of reproductive organs (L- and S-morphs) promote outcrossing and reduce self-pollination.

reproductive organs in heterostyly promotes outcrossing through segregated pollen deposition on pollinators' bodies (Kohn & Barrett 1992, Simon-Porcar et al. 2015, Zhou et al. 2015).

Following origins of floral complexity, reverse transitions toward simpler ancestral forms do occur, and are often clustered within lineages. Evolutionary patterns of gain and loss for these complexity traits represent the superimposition of these two processes. Reversals toward ancestral forms appear to occur on a more rapid timescale than do gains. For example, a single origin of sympetaly in the ancestor of Lamianae (Lamiales, Solanales, and allied orders) has been followed by at least five reversals to free petals (Stull et al. 2018). In Lamiales, a single origin of bilateral symmetry has been followed by at least eight reversals to radial symmetry (Zhong et al. 2017). Similarly, in Malpighiaceae, bilateral symmetry is a shared ancestral trait and at least four lineages have independently reverted to radial symmetry (Zhang et al. 2013). Once gained, nectar spurs can be lost (Ballerini et al. 2019, Fernández-Mazuecos et al. 2019, Hodges 1997), but assessment of the relative rate of gain versus loss requires further investigation. Losses of heterostyly are extremely common within heterostylous lineages and reflect selection for a highly selfing mating strategy. For example, a single origin of heterostyly in *Primula* is followed by several independent losses across the genus (de Vos et al. 2014, Mast et al. 2006, Zhong et al. 2019). A selfing strategy associated with loss of heterostyly can be favored when pollinators are rare or unreliable and when inbreeding depression is minimal, allowing the transmission advantage associated with selfing to be realized (reviewed in Busch & Delph 2012).

Not all floral parallelisms involve qualitative changes in complexity like those described above. Evolutionary transitions between quantitative aspects of floral organ dimensions (size and shape) are common. Changes in petal size, corolla tube, and nectar spur length occur even within genera (**Figure 5*a–c***) and are frequently evolutionarily labile in multiple directions, without a clear bias in directionality. For example, in *Linaria* there have been repeated evolutionary transitions between narrow and wide corolla tubes and between shorter and longer nectar spurs (Cullen et al. 2018).

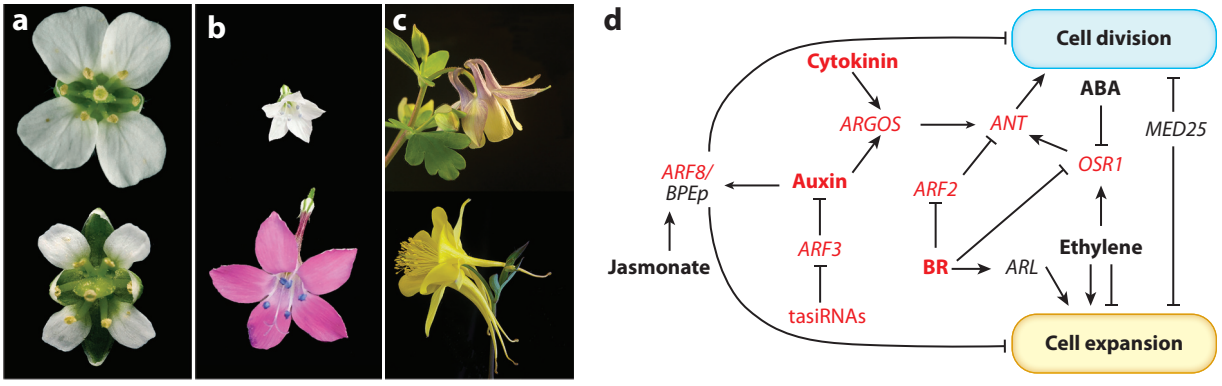

Figure 5

Evolution of flower dimensions through changes in cell proliferation and/or expansion. (*a*, *top*) *Capsella grandiflora* and (*bottom*) *Capsella rubella* differ in flower size as a result of selection for selfing in *C. rubella*. (*b*, *top*) *Saltugilia australis* and (*bottom*) *Saltugilia splendens* differ in corolla tube length (and petal size) as a result of selection imposed by flower–pollinator interactions. (*c*, *top*) *Aquilegia brevistyla* and (*bottom*) *Aquilegia chrysantha* differ in nectar spur length as a result of selection imposed by flower–pollinator interactions. (*d*) The genetic programs regulating the balance between cell proliferation and cell expansion are complex and rely on hormone regulation. Some genes jointly affect both cell proliferation and cell expansion (e.g., *ARF8/BPEp* and *MED25*). Hormones are in bold; genes and hormones discussed in this review are in red. Abbreviations: *ANT*, *AINTEGUMENTA*; *ARL*, *ARGOS-LIKE*; *ARF*, *AUXIN RESPONSE FACTOR*; *BPE*, *BIG PETAL*; BR, brassinosteroid; *MED*, *Mediator of RNA polymerase II*; tasiRNAs, *trans*-acting small interfering RNAs. Photographs provided by (*a*) Adrien Sicard, (*b*) Jacob Landis, and (*c*) Evangeline Ballerini.

Evolutionary changes in corolla tube, reproductive organ, or nectar spur dimensions can facilitate pollen placement on coadapted pollinators while excluding others, thereby promoting pollinator specialization as a mechanism to maximize outcross mating success. However, transitions to very small flowers reflect selection for a highly selfing mating strategy, and transitions to small selfing flowers are often asymmetrical with transitions back to large outcrossing flowers unlikely (e.g., Baldwin et al. 2011).

3. DEVELOPMENTAL TRANSITIONS TOWARD FLOWER COMPLEXITY

3.1. The Developmental Basis of Sympetaly

Petal primordia initiate either in a spiral arrangement (e.g., magnolia flowers) or in the whorled arrangement common to most eudicot and monocot species. Sympetaly, which forms corolla tubes or keeled petals, occurs on a floral ground plan in which petal primordia initiate in a whorled arrangement such that lateral petal boundaries are adjacent to one another, facilitating petal-to-petal fusion (**Figure 1**). The initiation of flower organs, including petals, in either a spiral or whorled arrangement results from positional information established by early auxin foci on the floral meristem, reinforced by cytokinin signaling leading to localized cell proliferation (reviewed in Rast & Simon 2008, Smyth 2018). But exactly how evolutionary transitions between spiral and whorled arrangements occur remains largely a mystery.

Sympetaly occurs by two primary processes—congenital and postgenital fusion (reviewed in Specht & Howarth 2015, Verbeke 1992, Zhong & Preston 2015). Petal primordia that undergo congenital fusion (e.g., Lamiales; **Figure 1*a***) are united through the connection and extension of a meristematic region underlying the already-initiated petal primordia. In postgenital fusion (e.g., Fabaceae; **Figure 1*b***), petals develop from distinct primordia that merge through a process of epidermal union. Given these divergent processes, nonparallel genetic mechanisms may underlie independent transitions to sympetaly. Determining the genetic basis of petal fusion has focused largely on model species in Lamiales and Solanales that show congenital fusion (e.g., *Antirrhinum*, *Mimulus*, *Petunia*), and emerging insights are beginning to suggest parallel genetic mechanisms.

Characterization of *Mimulus lewisii* mutants with loss of petal fusion led Ding et al. (2018) to propose a compelling model for congenital sympetaly. The model centers on regulation of auxin at the interpetal primordia boundaries (**Figure 1*c***). In species with free petals, auxin levels are low between initiating petal primordia (Heisler et al. 2005, Reinhardt et al. 2003), where an organ boundary genetic program that maintains distinction between adjacent petals is upregulated (reviewed in Rast & Simon 2008). In *M. lewisii*, formation of the corolla tube is associated with high levels of auxin between petal primordia, consistent with cell proliferation in the interpetal primordia region.

The *M. lewisii* loss-of-fusion mutants suggest that evolutionary changes in the organ polarity program lead to elevated auxin levels between petal primordia, resulting in corolla tube growth. The organ polarity program determines adaxial/abaxial (top/bottom) identity of laminar organs (e.g., leaves and petals) and regulates auxin for laminar growth. Within this program, AUXIN RESPONSE FACTOR (ARF) proteins, specifically *Arabidopsis* ARF3, are known to repress auxin accumulation (Simonini et al. 2017). In *M. lewisii*, negative regulation of *ARFs* by *trans*-acting small interfering RNAs (tasiRNAs) is associated with high levels of interpetal auxin (**Figure 1*c***). Mutants defective for tasiRNA processing have elevated *ARF* expression levels, reduced auxin levels between petal primordia, and reduced petal fusion (Ding et al. 2018). The hypothesis presented by Ding et al., that changes to interpetal auxin levels mediated by the polarity program determine sympetaly, is consistent with petal loss-of-fusion mutants in *Ipomoea* (*feathered*; Iwasaki & Nitasaka

2006) and *Petunia* (*maewest*; Vandenbussche et al. 2009). Both of these mutations occur in genes that are components of the adaxial/abaxial polarity program.

Elevated auxin levels are hypothesized to negatively regulate the organ boundary program (Furutani et al. 2004, Vernoux et al. 2000; reviewed in Rast & Simon 2008). This negative regulation allows confluence between developing *M. lewisii* petals. The model postulating upregulation of auxin between petal primordia, leading to downregulation of the organ boundary program (**Figure 1*c***), is in line with results from *Antirrhinum majus* (snapdragon), which clearly show that where the corolla tube develops, the organ boundary gene *CUPULIFORMIS* (*CUP*) is downregulated (Rebocho et al. 2017). The *CUP* homolog in *Petunia*, *NO APICAL MERISTEM* (*NAM*), along with an additional organ boundary gene, *HANABA TARATSU* (*HAN*), has also been implicated in the development of fused petals (Preston et al. 2019, Souer et al. 1996, Zhong et al. 2016). However, recent results demonstrate a role for *NAM* and *HAN* in promoting organ fusion, not maintaining organ boundaries (Preston et al. 2019, Zhong et al. 2016).

Because sympetaly has evolved multiple times, employing divergent developmental mechanisms (i.e., congenital and postgenital fusion), at first glance it would seem unlikely that parallel changes to auxin accumulation mediated by the polarity program evolve repeatedly. Tantalizingly, the tasiRNA–ARF pathway is implicated in formation of the legume keel (Yan et al. 2010, Zhou et al. 2013), derived from postgenital fusion of two petals (**Figure 1*b***) (Crozier & Thomas 1993). How the tasiRNA–ARF pathway affects the organ boundary pathway at late stages of petal development, after petal organ boundaries have been established, remains unknown. Still, these studies from Lamiales/Solanales and Fabaceae point to parallel developmental genetic mechanisms leading to divergent forms of sympetaly.

3.2. The Developmental Basis of Flower Symmetry

Breaking radial symmetry requires the evolution of distinct developmental trajectories on the dorsal versus ventral side of a developing flower (**Figure 2**). Our understanding of the genetic control of dorsal and ventral identity in bilateral flower symmetry comes primarily from research in snapdragon (Lamiales). Early in snapdragon flower development, even before flower organ primordia are visible, the flower symmetry genes *CYCLOIDEA* (*CYC*) and *DICHOTOMA* (*DICH*) are expressed on the dorsal side of the developing flower meristem. This pattern of early dorsal-restricted expression has been identified in some but not all species with radially symmetrical flowers (Busch et al. 2011, Cubas et al. 2001, Zhong & Kellogg 2015), leaving it unclear whether restricted expression predates the evolution of bilateral flower symmetry. In snapdragon flowers, expression persists in the developing dorsal organs through later stages of maturation (Luo et al. 1996, 1999). Their dorsal-restricted expression sets in motion a cascade of genetic interactions (**Figure 2*c***) that lead to differential development of flower organs along the dorsoventral flower axis affecting the petal, stamen, and carpel whorls.

CYC and *DICH* belong to the class II lineage of TCP [named after *TEOSINTE BRANCHED 1* (*TB1*) in *Zea mays* (maize; Doebley et al. 1997), *CYC* in *Antirrhinum majus* (snapdragon; Luo et al. 1996), and *PCF* in *Oryza sativa* (rice; Kosugi & Ohashi 1997)] family transcription factors (Martín-Trillo & Cubas 2010) and are paralogs resulting from a gene duplication event that occurred much more recently than the origin of bilateral flower symmetry in the snapdragon lineage (Gübitz et al. 2003, Hileman & Baum 2003). CYC and DICH positively regulate *RADIALIS* (*RAD*), a MYB family transcription factor. Similar to that of *CYC* and *DICH*, expression of *RAD* is restricted to the dorsal flower meristem and developing dorsal organs (Corley et al. 2005). A key regulator of ventral petal identity is *DIVARICATA* (*DIV*) (Galego & Almeida 2002). *DIV* and *RAD* are paralogs of one another. *DIV* is expressed both ventrally and dorsally, but in order to exclude

ventral identity from the dorsal side of the flower, dorsal-restricted RAD protein competitively excludes DIV protein interactions required for DIV function (Raimundo et al. 2013).

Symmetry genes in snapdragon determine dorsal and ventral fates by affecting patterns of cell division and/or cell expansion (Cui et al. 2010, Green et al. 2010). TCP family genes are known to broadly affect patterns of cell division, expansion, and differentiation (Martín-Trillo & Cubas 2010); therefore, *CYC* and *DICH* may determine dorsal patterns of division and expansion directly or indirectly via *RAD*. Recent research has begun to elucidate the mechanisms underlying complex shape formation of the snapdragon ventral lip (**Figure 2*c***). During stages of development when the ventral petal undergoes sharp curvature through localized cell proliferation, the following genes are expressed at the site of curvature: *DIV* (Galego & Almeida 2002), *CUP*, *YUCCA1* (Rebocho et al. 2017), and *AINTEGUMENTA* (*ANT*) (Delgado-Benarroch et al. 2009). *YUCCA1* is an auxin biosynthetic gene associated with auxin accumulation and initiation of localized cell proliferation. *ANT* belongs to the *AINTEGUMENTA-LIKE/PLETHORA* (*AIL/PLT*) gene family, which is known to be auxin responsive (Krizek 2011), potentially placing *ANT* downstream of *YUCCA1*. Rebocho et al. (2017) provide compelling evidence that *YUCCA1* is positively regulated by CUP, and that *CUP* in turn is positively regulated by DIV. Together, these analyses begin to shed light on how symmetry genes may shape floral organ development across the dorsoventral axis by affecting regulators of cell proliferation via auxin signaling.

Functional data from across eudicots support parallel recruitment of a *CYC*-dependent program in multiple origins of bilateral flower symmetry. In the sunflower family (Asteraceae), bilaterally symmetrical ray flowers have evolved more than once (Panero & Funk 2008), and different *CYC*-like paralogs appear to have been recruited independently to direct ray flower development (Broholm et al. 2008; Chapman et al. 2012; Fambrini et al. 2011, 2018; Garcês et al. 2016; Kim et al. 2008). In papilionoid legumes, three *CYC*-like genes are responsible for dorsoventral flower patterning. In *Lotus japonicus*, *CYC1* and *SQUARE PETALS* (*SQU*) function redundantly to pattern the dorsal banner petal, while *KEELED WINGS IN LOTUS* (*KEW*) functions primarily to maintain identity of the lateral petals distinct from the ventral keel petals (Feng et al. 2006, J. Wang et al. 2010, Z. Wang et al. 2008, Xu et al. 2013) (banner, lateral, and keel petals of *Pisum sativum*, a close relative of *Lotus japonicus*, are labeled in **Figure 1*b***). In Brassicaceae, a close relative of *Arabidopsis*, *Iberis amara*, develops bilaterally symmetrical flowers and the differential growth of dorsal compared with ventral petals results from dorsal-specific petal expression of the *CYC*-like gene *TCP1* (Busch & Zachgo 2007). In addition to these functional studies, research focusing on the spatial distribution of *CYC*-like gene expression in both monocots and eudicots supports independent recruitment of a *CYC*-dependent program for bilateral flower symmetry (e.g., Bartlett & Specht 2011, Citerne et al. 2017, Howarth et al. 2011, Jabbour et al. 2014, Preston & Hileman 2012, Zhang et al. 2010, Zhao et al. 2018).

Independent recruitment of a *CYC*-dependent program to shape bilateral flower symmetry requires the program to be regulated such that *CYC*-like gene expression is restricted to the dorsal (or ventral) side of the developing flower. How *CYC*-like genes evolve restricted expression along the dorsoventral axis is not well understood. Only in snapdragon, through characterization of the *backpetals* mutant, do we know that *CYC* expression would be continuous across the flower except for a regulatory sequence in its promoter that negatively regulates *CYC* on the ventral side of the developing flower (Luo et al. 1999). Whether similar mechanisms explain the independent origins of restricted *CYC*-like expression remains unknown.

Across multiple eudicots, genetic studies point to loss of dorsal-restricted *CYC*-like gene expression in independent reversals to radial flower symmetry. In *Plantago*, *Callicarpa*, *Mentha*, and *Tengia* (Lamiales), *Microsteria* and *Psychopterys* (Malpighiaceae), and *Cadia* (Fabaceae), reversals to radial symmetry are associated with expanded expression of *CYC*-like genes across the

dorsoventral floral axis (Citerne et al. 2006, Pang et al. 2010, Preston et al. 2011, Zhang et al. 2013, Zhong et al. 2017). In *Callicarpa* and *Mentha*, this association is accompanied by expanded or absent *RAD*-like gene expression, respectively (Zhong et al. 2017). A few additional independent reversals to radial symmetry in Malpighiaceae, as well as in *Bournea* and *Lycopus* (Lamiales), are associated with conservation of dorsal-restricted *CYC*-like gene expression (Zhang et al. 2012, 2013; Zhong et al. 2017; Zhou et al. 2008). This finding suggests potentially more complicated mechanisms than a simple loss of dorsal-specific regulation (reviewed in Hileman 2014). In *Lycopus*, radially symmetrical flowers seem to have evolved through loss of *RAD*-like gene expression (Zhou et al. 2008).

3.3. The Developmental Initiation of Nectar Spurs

In their multiple origins, nectar spurs derive from a variety of floral tissues (Endress 2001). In *Aquilegia* (columbines, Ranunculaceae; **Figure 5*c***), *Centranthus* (Caprifoliaceae), and *Linaria* (Plantaginaceae), nectar spurs develop as tubular outgrowths from the laminar petal or corolla tube surface (**Figure 3**) (Cullen et al. 2018, Damerval & Becker 2017, Mack & Davis 2015). In *Delphinium*, nectar spurs are uniquely integrated into both inner and outer whorl sepals and petals (Jabbour & Renner 2012). Even in closely related *Aquilegia*, which has evolved nectar spurs independently from those in *Delphinium*, nectar spurs develop in only the inner whorl petals. *Impatiens* (Balsaminaceae) develops nectar spurs from outer whorl sepals (Young 2008). Interestingly, in *Pelargonium* (Geraniaceae), nectar spurs develop from intercalary growth within the receptacle, resulting in a long cavity that appears to be (but is not) a sepal-derived spur fused to the pedicel (Tsai et al. 2018).

The initiation of tubular outgrowths requires a new signal on the laminar surface (e.g., developing petal or sepal) that leads to a focused area of cell division (**Figure 3*b***). Once a nascent spur initiates, spur elongation may occur through processes of additional cell division and/or cell expansion. Variation in early cell division or late cell expansion may contribute to interspecific variation in spur length (see discussion in Section 4.3, below). Research focused on a few model species representing independent origins of nectar spurs points to divergent developmental mechanisms.

In *Aquilegia*, auxin signaling is implicated in the initiation of a discrete cell proliferation zone on the developing petal laminar surface, which results in an out-pocket, or cup, forming the nascent spur (Ballerini et al. 2019, Yant et al. 2015). Evidence supporting this model comes from gene expression studies showing that an *Aquilegia* homolog of a gene implicated in auxin biosynthesis, *CYTOCHROME P450 FAMILY 71A* (*CYP71A*), is significantly upregulated at the earliest stages of localized spur initiation within the petal field. In addition, genes downstream in auxin signaling, including *ARF3/ETTIN*, *ARF8*, and *SMALL AUXIN UPREGULATED RNA* (*SAUR*), are upregulated at the same early stages. Notably, *SAUR* is implicated in promotion of cell expansion (Spartz et al. 2012), and cell expansion may be critical for spur elongation in *Aquilegia* (Puzey et al. 2012).

Another group of genes implicated in spur development is the *KNOX* genes. These genes are not upregulated in the *Aquilegia* spur, but instead appear to be important for nectar spur development in relatives of snapdragons. Snapdragon flowers do not develop a nectar spur but do produce a nectar sac (gibba) at the proximal end of the ventral corolla tube. In close relatives (e.g., *Linaria*), the gibba develops into a nectar spur. Snapdragon mutants constitutively overexpressing *STM-like class I KNOX* genes produce a tubular outgrowth on the ventral petal that is reminiscent of *Linaria* spurs (Golz et al. 2002). These ectopic tubular outgrowths can be interpreted as a duplicated corolla tube or a spurlike structure, but either way they suggest a divergent mechanism of

spur initiation. This divergent mechanism requires ectopic expression on the already-developing petal surface of a novel meristematic region from which spur outgrowth is organized, presumably initiated by a novel pattern of *KNOX* expression. In *Linaria*, homologs of these *KNOX* genes show a surprisingly broad pattern of expression in the differentiating dorsal and ventral petals—not perfectly but somewhat overlapping with the zone of nectar spur development. In snapdragon, in contrast, *KNOX* genes exhibit the canonical expression pattern restricted to undifferentiated meristematic tissues (Box et al. 2011, Golz et al. 2002).

This class of *KNOX* genes has been recruited for compound leaf development (reviewed in Nikolov et al. 2019), indicating the potential for *KNOX*-driven developmental complexity outside of meristems. Notably, *KNOX* genes are not upregulated in the developing *Aquilegia* spur (Yant et al. 2015). That auxin-responsive proteins function to downregulate *KNOX* genes at meristem edges in order for differentiation to occur (Heisler et al. 2005) further supports the hypothesis that auxin-driven spur development (in *Aquilegia*) and *KNOX*-driven spur development (in *Linaria*) represent divergent developmental genetic mechanisms. While nectar spurs can be lost (e.g., in *Aquilegia* and Antirrhineae, the tribe to which *Linaria* belongs; Ballerini et al. 2019, Fernández-Mazuecos et al. 2019), the developmental basis of spur loss has not been extensively studied.

4. DEVELOPMENTAL TRANSITIONS IN FLOWER DIMENSIONS WITH SHIFTS IN MATING SYSTEM AND PRIMARY POLLINATOR

4.1. Hormone-Responsive Pathways Control Floral Organ Size and Shape

Similar to the initiation of a petal lip or nectar spur, floral organ dimensions result from two primary phases of organ growth: an initial period of cell division followed by a period of cell expansion. Whereas the initiation of floral organs centrally involves auxin signaling, the two phases of organ growth are influenced by multiple plant hormones, including auxin, and diverse genes in regulatory networks acting downstream of hormones (**Figure 5*d***). Details of these networks and candidate genes identified in the model species *Arabidopsis* and *Antirrhinum* have recently been extensively reviewed (e.g., Krizek & Anderson 2013, Moyroud & Glover 2017). It is clear that many genetic interactions contribute to the duration and rate of cell division and to the degree of cell expansion; therefore, variation in floral dimensions may often be polygenic.

Several major themes emerge from genetic studies of floral organ size control. First, organ size is influenced by the intersection of several different plant hormones that, in combination, affect development (**Figure 5*d***). An illustrative case is the gene regulatory network involving *ANT*, which promotes cell division by positively regulating cell cyclin genes in *Arabidopsis* (Mizukami & Fischer 2000). Multiple regulators of *ANT* have been described, including *AUXIN-REGULATED GENE INVOLVED IN ORGAN SIZE* (*ARGOS*) (Krizek 1999, Mizukami & Fischer 2000), *ORGAN SIZE RELATED 1* (*OSR1*) (Feng et al. 2011), and *ARF2* (Vert et al. 2008). Importantly, these *ANT* regulators are themselves regulated by diverse hormones: *ARGOS* is upregulated by auxin and cytokinin (Hu et al. 2003), *OSR1* is upregulated by ethylene (Feng et al. 2011), and *ARF2* is likely sensitive to brassinosteroids (BRs) and auxin signals (Vert et al. 2008). These findings point to the importance of flexible coregulation of a single network by a variety of plant hormones during flower development. Second, specific hormones can be involved in both cell division and cell expansion processes, through different regulatory pathways. This is true at least for ethylene (Feng et al. 2011, van Es et al. 2018) and BRs (Hu et al. 2006, Vert et al. 2008). Third, there are individual genes that affect both cell division and expansion processes (**Figure 5*d***) (Feng et al. 2011, Varaud et al. 2011, Xu & Li 2011). Finally, important mechanisms for limiting organ size include ubiquitin-mediated degradation of positive growth factors (Disch et al. 2006, Li et al.

2008) and the action of TCP family transcription factors that promote cell differentiation (Huang & Irish 2015).

4.2. The Developmental Basis of Flower Size Transitions with Selfing

A model for studying developmental changes responsible for reduced flower size associated with self-pollination is *Capsella rubella* (Brassicaceae) (**Figure 5*a***). This species has evolved flowers that are fivefold smaller than those of its outcrossing sister species, *Capsella grandiflora*, largely through reduced cell division in floral organs (Sicard et al. 2011). Fine-mapping of quantitative trait loci in the interspecific cross has identified causal mutations at two loci contributing to differences in cell division. The first is a petal-specific enhancer of *STERILE APETALA* (*SAP*) that encodes an F-box protein component of an E3 ubiquitin ligase (Sicard et al. 2016). This ubiquitin ligase promotes cell division by targeting negative regulators of cell proliferation (Wang et al. 2016). *C. rubella* has acquired mutations to this enhancer that reduce *SAP* gene expression, resulting in reduced flower size. The second locus is *CYP724A1*, a gene in the BR synthesis pathway (Fujikura et al. 2018). The *C. rubella CYP724A1* allele has mutations conferring greater splicing efficiency that results in higher gene expression, which in turn increases BR levels. These higher BR levels inhibit cell division and lead to smaller flowers (Fujikura et al. 2018). These data point to the precise regulation of cell division and cell expansion by hormones. Depending on the context, hormone increases may promote or inhibit cellular processes. The quantitative genetic basis of changes in overall size has also been studied in other species (e.g., *Mimulus guttatus*; Kelly & Mojica 2011); however, the underlying developmental pathways have not yet been identified.

4.3. The Developmental Basis of Flower Dimension Transitions with Pollinator Shifts

The developmental basis of floral evolution associated with pollinator transitions has been investigated in several genera. In *Petunia*, flower shape evolution in association with adaptation to hawkmoth and hummingbird pollinators has occurred through changes to both cell division and expansion. Hawkmoth-pollinated *Petunia axillaris* has evolved increased corolla tube length relative to bee-pollinated *Petunia integrifolia* through increased cell division and cell expansion (Stuurman et al. 2004). Elongated stamen filaments and styles in hummingbird-pollinated *Petunia exserta* have involved primarily cell division (Hermann et al. 2015). In *Saltugilia* (**Figure 5*b***), flower size variation associated with adaptation to different pollinators results primarily from differences in cell expansion (Landis et al. 2016), whereas in *Lithospermum*, flower size variation involves primarily changes in cell division (Cohen 2016). Candidate genes for these pollinator-associated evolutionary transitions have not been reported.

Similar to corolla tube length, spur length evolves in response to specific pollinators. For example, hummingbird- and hawkmoth-pollinated species have longer spurs compared with bee-pollinated relatives. Studies in different lineages point to divergent mechanisms underlying spur length differences among closely related species. Among closely related *Linaria* species, differences in cell division early in spur patterning explain most interspecific spur length variation (Cullen et al. 2018). However, among closely related *Aquilegia* species (**Figure 5*c***), differences in cell expansion at later stages of differentiation explain most interspecific variation in spur length (Puzey et al. 2012). In the unique spurs of *Pelargonium*, both cell division and cell expansion processes jointly determine interspecific spur length differences (Tsai et al. 2018). Given the developmental complexity of cell division and expansion networks in floral tissue (**Figure 5*d***), there are many target loci that could, in theory, generate adaptive variation in spur length. One appealing

candidate for spur length variation due to cell expansion is the *SAUR*-dependent pathway, implicated in *Aquilegia* spur cell elongation (Yant et al. 2015).

4.4. The Developmental and Genetic Bases of Heterostyly

Evolution of heterostyly requires complete linkage of major effect alleles that cause reciprocal differences in reproductive organ length (**Figure 4**). For example, with distyly, a major effect allele that causes short styles is linked to a major effect allele at a second locus that causes long stamen filaments. Often, these loci are also linked to a self-incompatibility locus. The set of linked loci is termed the *S*-locus supergene, and high linkage disequilibrium is maintained by suppressed recombination (Barrett & Shore 2008, Charlesworth 2016). In multiple genera, suppressed recombination results from *S*-locus hemizygosity, with *S*-locus genes present in one morph and completely absent in the other (Cocker et al. 2018, Kappel et al. 2017, Shore et al. 2019, Ushijima et al. 2012, Yasui et al. 2012).

In theory, major effect alleles for organ length polymorphism could arise in any of the diverse networks that affect floral organ cell division or cell expansion, as long as mutations can specifically affect a single floral whorl (e.g., styles but not stamen filaments). Only a small number of loci responsible for style and/or stamen length in heterostylous taxa have been identified, yet it is already clear that diverse loci are recruited into *S*-locus supergenes. In *Primula*, allelic differences causing style length variation via changes in cell expansion are caused by the presence or absence of a *CYP734A50* homolog in the *S*-locus (Huu et al. 2016, Li et al. 2015, Nowak et al. 2015). *CYP734A50* is known to function in BR degradation. Individuals with the *S*-locus haplotype containing *CYP734A50* have short styles because of increased BR degradation that causes reduced cell expansion in style tissue (Huu et al. 2016, Nowak et al. 2015). It is not yet known how this locus influences organ length in the style only. In *Turnera*, variation in style length is caused by the presence or absence of a *BAHD* acyltransferase homolog that likely functions to inactivate BRs (Shore et al. 2019). Interestingly, *Primula* and *Turnera* have functionally converged on BR-dependent mechanisms for style length polymorphism, albeit through distinct components of BR regulation. The loci affecting stamen length in *Primula* and *Turnera* have been identified as homologs of the B-class organ identity gene *GLOBOSA* (Nowak et al. 2015) and of *S-PROTEIN HOMOLOG 1* (*SPH1*) (Shore et al. 2019), respectively. However, it is currently unclear how allelic variation at these loci determines stamen length.

Reversals from heterostyly to homostyly associated with transitions to selfing occur relatively rapidly (e.g., de Vos et al. 2014, Mast et al. 2006, Zhong et al. 2019). Homostyly can be caused by loss-of-function mutations in one or more genes in the hemizygous *S*-locus. In both *Primula* and *Turnera*, loss-of-function mutations at the style length loci (*CYP734A50* and *BAHD*, respectively) inactivate their repressive effects, resulting in so-called long-homostyle phenotypes, in which style length is similar to stamen length (Huu et al. 2016, Shore et al. 2019). Accordingly, short-homostyle mutants in these two systems result from loss-of-function mutations to the filament length loci (*GLOBOSA* and *SPH1*) (Li et al. 2016, Shore et al. 2019).

5. CONSTRAINTS SHAPING PARALLEL AND DIVERGENT PROCESSES

The floral traits reviewed above exhibit evolutionary parallelisms in the context of function, but often involve nonparallel changes to organ-level development. For example, sympetaly can be congenital or postgenital; bilateral flower symmetry can derive from developmental differences in the perianth, the stamen whorl, or both; and nectar spurs can be derived from different floral organ tissues. In most cases, these novel traits require a signal establishing new patterns of cell proliferation. Sympetaly requires initiation of cell proliferation between otherwise distinct organs; spur

development requires focused cell proliferation within a laminar surface. Data point to a parallel process of novel auxin foci as an initiating signal. This is not surprising, since auxin accumulation is a primary mechanism by which organ outgrowth occurs in plants and likely represents a significant constraint on patterning mechanisms.

Once localized cell division is patterned, the input and interplay between cell division and cell expansion required to shape developing organs are complex (**Figure 5*d***). It is not surprising that studies point to multiple components of this pathway affecting organ dimensions. Which loci are the target of selection is likely constrained by potential pleiotropy, with genes already acting in an organ-specific manner reducing off-target effects (e.g., a *GLOBOSA* homolog in stamen length variation). However, many genetic changes channel response through the BR hormone pathway (e.g., petal size in *Capsella*; style length in *Primula* and *Turnera*). BR-dependent pathways may be particularly flexible targets for adaptive evolution of decreased floral organ length. Aside from being implicated in both cell division and expansion, BR-dependent mechanisms seem to be tightly controlled by hormone concentrations: Either an increase or a decrease in BR concentration inhibits organ growth (Fujikura et al. 2018). Therefore, multiple genetic mechanisms may result in reduced organ growth by disturbing BR levels away from levels that maximize cell division or expansion. Genes involved in the degradation of growth-promoting factors (including BRs) show a pattern of parallel recruitment, again pointing to the importance of the levels of critical growth-promoting factors.

6. RECIPROCAL ILLUMINATION BETWEEN PHYLOGENETIC PATTERNS AND GENETIC MECHANISMS

Adaptive transitions in floral traits are ultimately limited by the availability of suitable mutations. This can cause genetic constraints that shape patterns of trait evolution if mutations causing certain traits arise much more frequently than mutations causing transitions to other traits or reversals to the ancestral state. Such mutational biases may help explain the tempo and relative reversibility of parallel transitions. We see this relationship for two well-studied traits, each with a relatively simple genetic basis: first, the parallel evolution of self-compatible (SC) from self-incompatible (SI) mating systems, and second, the parallel evolution of red flowers from bluish ancestors associated with transitions from bee to hummingbird pollination. In both cases, we find lineages in which parallel evolution in the forward direction (to SC or red flowers) is significantly more common than reversals to the ancestral condition (SI or blue) (Igic & Busch 2013, Wessinger et al. 2019). SC and red flowers are often produced through loss-of-function mutations to SI genes and anthocyanin pathway genes, respectively (reviewed in Shimizu & Tsuchimatsu 2015, Wessinger & Rausher 2012). The target size for mutations that disrupt gene function is much larger than that of mutations that can restore gene function to a degraded gene or pathway. Therefore, genetic constraints may contribute to the extreme asymmetry in transition rates between SI and SC, and between blue and red flowers. These represent additional constraints beyond those clearly imposed by selection from pollinator environment.

It is less apparent that mutational biases, in addition to selective processes, contribute to phylogenetic patterns in the floral traits reviewed above. These morphological traits are generated through developmental pathways that can be substantially more complex than those for SI and flower color. With this additional complexity, we lack a clear expectation that certain transitions more reliably involve frequently arising loss-of-function mutations, or other types of mutations with relatively large target size. Naïvely, we might assume that, following the origins of additional morphological complexity (e.g., sympetaly, bilateral symmetry, or spurs), secondary reversals to the ancestral condition might involve loss-of-function mutations that dismantle developmental complexity. We currently have limited information that this is the case.

Reversals from bilateral to radial flower symmetry are common and may be coupled to loss-of-function mutations at *CYC* regulatory sequences that eliminate dorsal-restricted expression, analogous to snapdragon *backpetals* (Luo et al. 1999). However, some reversals rely on other genetic mechanisms that do not mimic *backpetals*, but may also have large target size (e.g., loss of floral *RAD* expression in *Lycopus*). Transitions between unfused and fused corollas, as well as between spurred and unspurred flowers, seem to occur with appreciable frequency in certain angiosperm lineages (Ballerini et al. 2019, Hodges 1997, Reyes et al. 2018, Stull et al. 2018). However, we have a limited picture of phylogenetic patterns for these traits, making an assessment of relative reversibility difficult. In addition, we have scant information on the developmental bases for reverse transitions. Additional data on developmental mechanisms and macroevolutionary patterns for sympetaly and nectar spurs will allow further insights into the relationship between pattern and process.

For evolutionary transitions in floral dimension traits (e.g., flower and organ size), we expect minimal effects of mutational bias on patterns of trait evolution. Given the extremely complex regulatory networks involving both promotive and repressive pathways (**Figure 5*d***), frequently arising loss-of-function mutations could lead to either increases or decreases in size. Thus, we expect any asymmetries in the rates of transitioning between different flower dimensions to be shaped primarily by selective constraints. For example, transitions toward, but not away from, longer nectar spurs in *Aquilegia* are hypothesized to involve selective constraints imposed by moth pollinators (Whittall & Hodges 2007). An exception is the reversal from heterostyly to homostyly involving loss-of-function mutations to hemizygous genes at the *S*-locus (Huu et al. 2016, Li et al. 2016, Shore et al. 2019). In this case, the evolution of a hemizygous *S*-locus in heterostyly acts as a simple genetic locus, easily disrupted by mutation, echoing the mechanism for transitions from SI to SC. These loss-of-function mutations help explain the relatively rapid pace of transitions from heterostyly to homostyly and may generate a genetic constraint on reversals, helping to explain the asymmetrical pattern of parallel transitions.

7. SUMMARY

Approaching parallel trait evolution through the hierarchical lens has proven useful for understanding which levels of organization and patterning exhibit similarity. While we have identified examples of both parallel and divergent mechanisms, ranging from function through tissues to molecular changes, most revealing has been the central role of hormones in floral trait evolution. We see this both in the repeated establishment of novel floral traits and in evolutionary modifications associated with transitions to selfing and between pollinators. Traditionally, floral evolutionary/developmental research has focused on conservation and diversification of gene expression and function. Of course, identifying causal mutations for trait evolution is the holy grail, but the synthesis presented here suggests that research focused on the role of hormones in trait novelty will provide critical and novel insights. Genetic constraints shape not only the paths through which development proceeds but also the macroevolutionary patterns of trait evolution. Traits that have a simple genetic basis and that derive through loss-of-function mutations provide a clear opportunity for reciprocal illumination. Whether these insights extend to more complex developmental patterning is less clear. What is clear is that when, as in the case of heterostyly, complex trait development is traced to simple genetic mechanisms analogous to those underlying flower pigment evolution or the loss of self-incompatibility, reciprocal illumination is possible.

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