

8 June 2007 | \$10

Science

 AAAS

Evolution and Development of Inflorescence Architectures

Przemyslaw Prusinkiewicz,¹ Yvette Erasmus,^{2*} Brendan Lane,¹ Lawrence D. Harder,³ Enrico Coen^{2†}

To understand the constraints on biological diversity, we analyzed how selection and development interact to control the evolution of inflorescences, the branching structures that bear flowers. We show that a single developmental model accounts for the restricted range of inflorescence types observed in nature and that this model is supported by molecular genetic studies. The model predicts associations between inflorescence architecture, climate, and life history, which we validated empirically. Paths, or evolutionary wormholes, link different architectures in a multidimensional fitness space, but the rate of evolution along these paths is constrained by genetic and environmental factors, which explains why some evolutionary transitions are rare between closely related plant taxa.

Organisms display great diversity in shape and architecture, but the range of observed forms represents only a small fraction of what is theoretically possible (1, 2). For example, when patterns of shell coiling are considered within a mathematically defined space of possible forms (morphospace), the observed forms are restricted to only a subregion of this space (3). One explanation for such restrictions is selection (4). However, it is likely that developmental and genetic mechanisms also play a role. For example, the absence of vertebrates with more than four limbs is thought to reflect an interplay between both developmental and selective constraints (5, 6). Developmental mechanisms restrict the range of genetic and phenotypic variation available for selection, whereas selection influences the evolution of developmental processes. Such two-way interactions can be unravelled using morphospaces based on developmental genetic mechanisms. We take this approach for the evolution of inflorescences, which has a history of both theoretical and molecular genetic analysis.

The arrangement of flowers on a plant reflects an iterative pattern of developmental decisions at the growing tips, or meristems. Each iteration occurs over a time interval known as a plastochron (7), during which a meristem may either switch to floral identity or continue to produce further meristems and, hence, branches. As the number of iterations rises, the number of the-

oretically possible structures increases rapidly (8). However, only a small subset of these structures corresponds to inflorescences observed in nature (Fig. 1). They are grouped into three broad architectural types: (i) panicles, which comprise a branching series of axes that terminate in flowers; (ii) racemes, which comprise axes bearing flowers in lateral positions or lateral axes that reiterate this pattern; and (iii) cymes, which comprise axes that terminate in flowers and lateral axes that reiterate this pattern (9–11) (Fig. 1, D to I). The appearance of each inflorescence type varies according to the arrangement of lateral meristems around the stem (phyllotaxy), the pattern of internode lengths, and additional variations on the three architectural themes. Although panicles, racemes, and cymes are all found among flowering plants, a restricted range of types is evident at local taxonomic levels: Genera seldom include species with both racemes and cymes.

These observations raise two related questions. First, what determines the extent of morphospace occupied by inflorescences in nature: Why do we find these three main architectural types and not more or fewer? Second, what constrains evolution within the occupied morphospace, imposing a local barrier between racemes and cymes?

A unifying inflorescence model. Previously, distinct developmental models have been postulated for different inflorescence types (12, 13), leading to a fractured view of phenotypic space. From an evolutionary perspective, however, inflorescence types should be related to each other through genetic changes. A developmental model that encompasses different architectural types within a single parameter space is thus needed. To construct it, we considered meristems giving rise to shoots or flowers as two extremes of a continuum. The variable that characterizes this continuum is called vegetativeness (*veg*), with high levels of *veg* corresponding to shoot meristem identity and low levels to flower meristem identity. The *veg* level may be related to many factors such as plant age, meristem position, internal state of a meristem, and the environment (14–17). For simplicity, we identify the factors influencing *veg* as plant age *t*, measured from the beginning of inflorescence development, and/or the internal state of the meristem.

If *veg* is high and does not change with time, an indeterminate vegetative branching structure is generated (Fig. 2A). For the plant to produce flowers, *veg* must decline in some or all meristems during growth. The simplest assumption is that *veg* decreases in all meristems equally. The resulting architecture is a panicle of flowers, which form at time *T* when low levels of *veg* are reached (Fig. 2B).

To generate further architectural types, we assume that meristems can be in one of two internal states, A and B, such that meristems in these states attain low levels of *veg* at different

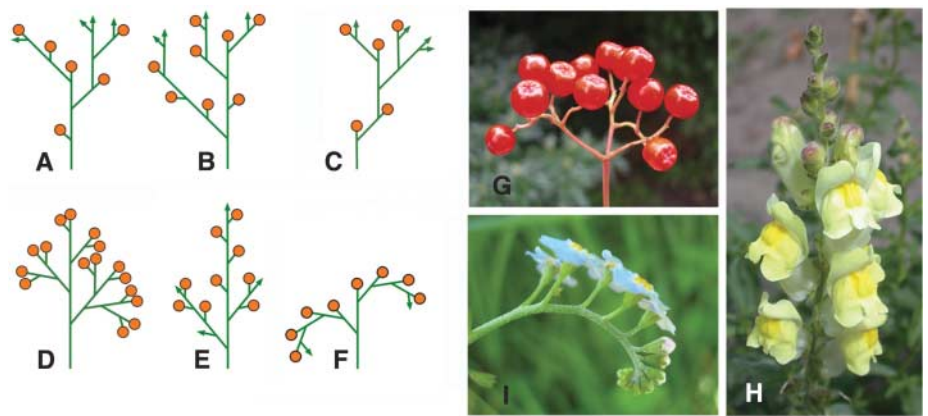


Fig. 1. Hypothetical and observed inflorescence structures. Arrows, meristems; circles, flowers. (A to C) Inflorescence structures not observed in nature. (D to F) Three main classes of inflorescence architectures: panicle (D), raceme (E), and cyme (F). (G to I) Species illustrating inflorescence types: (G) fruiting panicle of *Sorbus aucuparia*, (H) flowering raceme of *Antirrhinum majus*, (I) flowering cyme of *Myosotis arvensis*.

¹Department of Computer Science, University of Calgary, 2500 University Drive N.W. Calgary, Alberta T2N 1N4, Canada. ²Department of Cell and Developmental Biology, John Innes Centre, Colney Lane, Norwich NR4 7UH, UK. ³Department of Biological Sciences, University of Calgary, 2500 University Drive N.W. Calgary, Alberta T2N 1N4, Canada.

*Present address: Institute of Molecular Plant Science, Daniel Rutherford Building, Kings Buildings, Mayfield Road, Edinburgh, EH9 3JR, UK.

†To whom correspondence should be addressed. Email: enrico.coen@bbsrc.ac.uk

times, T_A and T_B . Suppose that the apical meristem of the main axis is in state A, whereas all lateral meristems are in state B. If $T_B < T_A$, lateral meristems will attain floral identity more quickly than does the apical meristem, yielding a raceme in the upper part of the plant (red path in Fig. 2C). However, lateral apices in the lower part of the inflorescence will also rapidly progress toward floral identity, producing a graded series of panicles (orange path in Fig. 2C). This

result is inconsistent with the structure of lateral branches of compound racemes, which typically do not terminate in flowers (Fig. 1E).

To resolve this discrepancy, we postulate that state B is transient. All lateral meristems are formed in state B, but they have two possible fates afterwards. If *veg* is sufficiently low, the meristem becomes a flower (red path, Fig. 2D). Otherwise, the meristem reverts to state A and produces a branch (orange path, Fig. 2D). The

rationale for this reversion is that a newly created meristem (state B) changes its identity once it becomes the terminal meristem of the next-order branch (state A). Biologically, state B represents the stage when a meristem is newly formed (immature), whereas state A represents a more advanced stage of meristem development (mature). We call the resulting model the transient model.

A key feature of the transient model is that it can generate cymes as well as racemes and panicles, thus accounting for these inflorescence types within a single framework. In cymes, lateral meristems repetitively create meristems in two different states: a terminal meristem giving rise to a flower, and a lateral meristem giving rise to a branch (Fig. 1F). This can be captured with the transient model by setting $T_A < T_B$. Immature lateral meristems then take longer to attain floral identity than do mature meristems, creating the reverse of the situation for racemes (Fig. 2E).

The region of morphospace generated by the transient model is illustrated in Fig. 3. The main diagonal ($T_A = T_B$) corresponds to panicles, flanked by racemes on one side ($T_A > T_B$) and cymes on the other ($T_A < T_B$). This planar region represents only a slice of the entire morphospace. For example, the hypothetical forms shown in Fig. 1, A to C, do not lie in this region and could only be generated with more complex mechanisms. The transient mechanism may therefore account for the restriction of observed inflorescence types to a small region of morphospace. Potentially adaptive architectures may not be attained in nature because they cannot be produced by developmental processes captured by the transient mechanism.

Integration of models with developmental and molecular genetics. To assess the plausibility of the transient model, we related it to underlying genetic mechanisms. We focused on two architectural genes from *Arabidopsis*, *TERMINAL FLOWER 1 (TFL1)* and *LEAFY (LFY)* (18–23), as they produce phenotypes that displace the wild-type plant in orthogonal directions in morphospace (Fig. 3). Compared to wild-type *Arabidopsis*, which produces branched racemes, mutants lacking *TFL1* activity produce inflorescences with short axes that terminate in flowers, whereas mutants lacking *LFY* produce highly branched inflorescences bearing shootlike flowers.

We incorporated genes into the transient model by postulating that *TFL1* increases *veg* and *LFY* reduces *veg* in meristems (Fig. 4A). The wild-type *Arabidopsis* architecture results from high *TFL1* and low *LFY* activity in state A meristems (yielding high *veg*) and high *LFY* and low *TFL1* activity in state B meristems (yielding low *veg*). This pattern of gene expression is produced by assuming that: (i) *LFY* and *TFL1* inhibit each other (18, 20, 22), (ii) *TFL1* expression is inhibited in state B, (iii) *LFY* is less sensitive to *TFL1* inhibition in state B [this enhances *LFY* in B meristems and allows *LFY* to attain high activity even in plants overexpressing

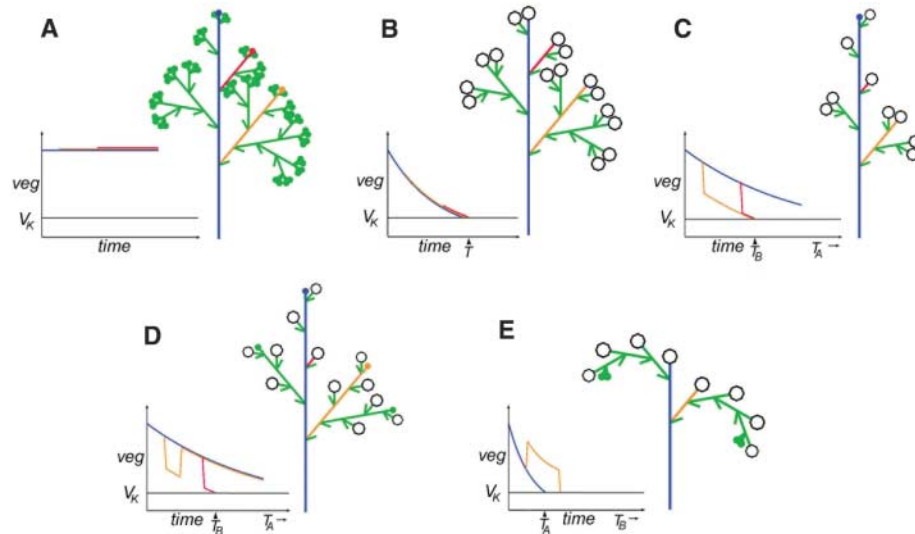


Fig. 2. Architectures and time course of *veg* decline for various inflorescence models. Small filled circles, meristems; white circles, flowers. Colors highlight paths of representative meristems: main meristem, blue; lowest lateral meristem, orange; third lateral meristem from bottom, red. Plots show the time course of *veg* decline in selected meristems after their initiation. (A) Level of *veg* does not change with time; an indeterminate vegetative branching structure results. (B) *veg* declines at a similar rate in all meristems and yields flowers upon reaching threshold V_K at time T ; a panicle results. (C) *veg* in apical meristems (state A) reaches threshold V_K at time $T_A > T_B$ for lateral meristems (state B). The resulting structure is a compound raceme, with lower branches terminating in flowers. (D) Transient model in which lateral meristems are initially in state B but revert to state A if *veg* does not reach the threshold V_K ; a raceme with indeterminate branches is produced for $T_B < T_A$. (E) Transient model in which $T_B > T_A$ yields a cyme.

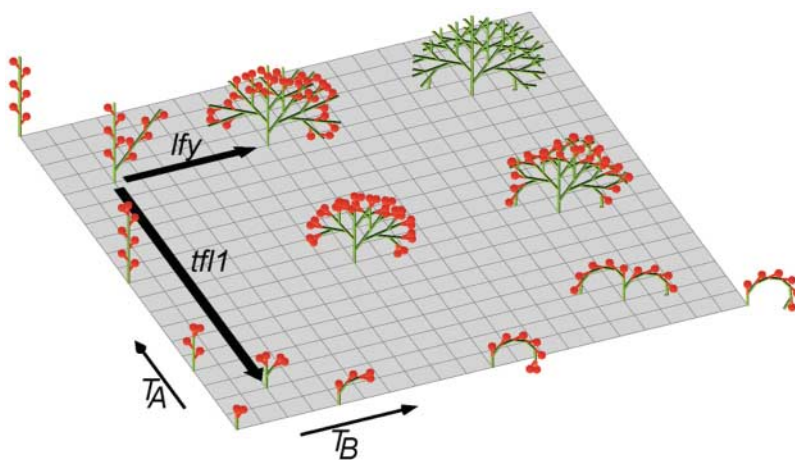


Fig. 3. Morphospace for the transient model. Different phenotypes are generated by varying the times T_A and T_B at which flowers begin to form. Values along each axis range from 0 to 10 plastochrons. Black arrows, pointing away from the wild-type architecture of *Arabidopsis*, indicate the effect of *tfl1* and *lfy* mutations [under inductive conditions (20)]. Inflorescences are shown at six plastochrons.

TFL1 (24)], and (iv) the levels of *TFL1* and *LFY* increase with plant age (21, 25). The shootlike flowers observed in *lfy* mutants (20) and plants overexpressing *TFL1* (22, 24) are captured by assuming that they correspond to intermediate *veg* levels, lying between the levels for normal shoots and flowers.

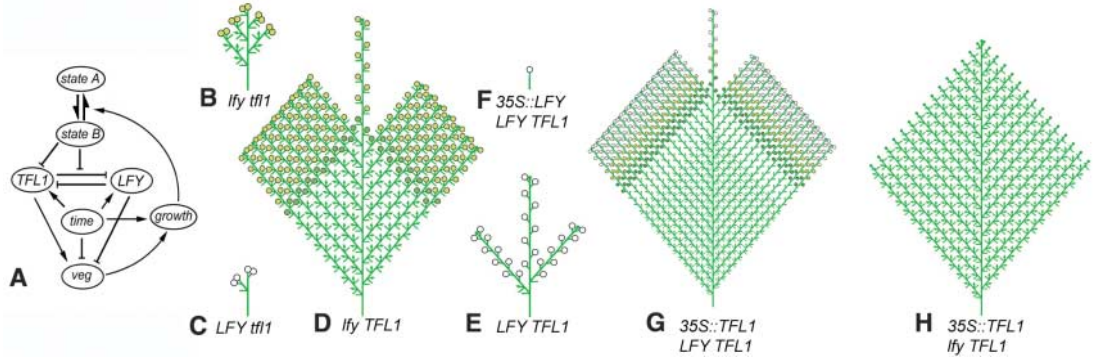
With these assumptions, the transient model largely accounts for mutant and overexpression phenotypes of *Arabidopsis* (Fig. 4, B to H, and

SOM text) and for observed patterns of gene expression. According to the model, a lateral meristem that gives rise to a branch is initially in state B, which corresponds to relatively low *TFL1* and high *LFY*. This pattern reverses when the meristem switches back to state A. These features agree with experimental data: *TFL1* expression is low in newly formed lateral primordia and only increases later (22). Furthermore, *LFY* is expressed early in primordia that will give rise

to lateral branches but is absent from mature lateral shoot meristems (25, 26). The transient model thus provides a functional explanation for observed expression patterns.

Evolutionary origins and implications of the transient mechanism. Both the range of architectural types observed in flowering plants and molecular genetic data lend support to the transient mechanism, but the evolutionary origin of this mechanism is unclear. To elucidate it, we

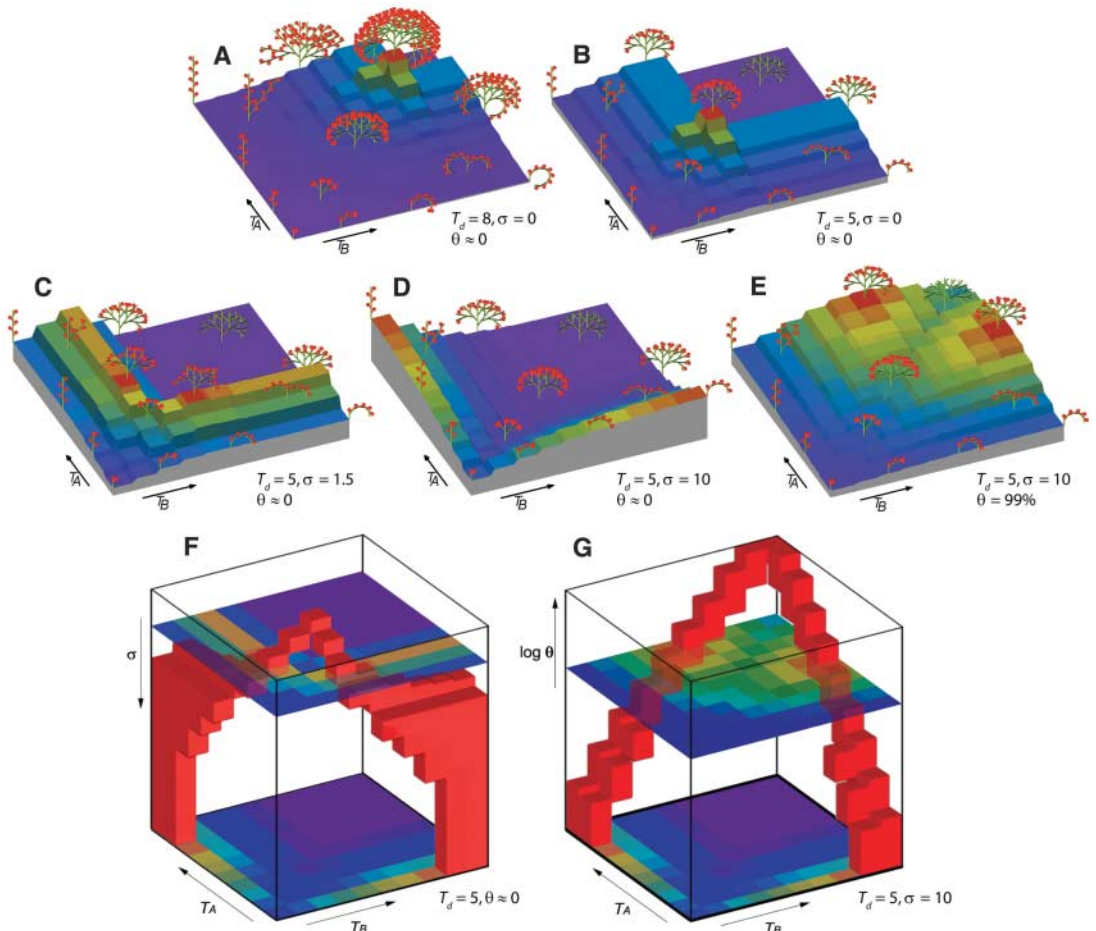
Fig. 4. Incorporating *LFY* and *TFL1* genes into the transient model for *Arabidopsis*. (A) Interactions between genes, time, *veg*, and growth underlying the model. Growth increases the number of modules and hence influences the spatial pattern of gene expression. Gene activity affects *veg* and hence influences whether a meristem will continue to generate more modules or whether it will cease growing. Arrowheads indicate up-regulation; bars, down-regulation. Growth promotes production of meristems in state A or B, with state B reverting to A unless the floral threshold is reached. (B to H) Wild-type, mutant, and transgenic phenotypes generated by the model with the



interactions shown in (A), assuming inductive conditions (20). Circles indicate flowers, color-coded according to *veg* levels. White, normal flower; yellow/green, shootlike flower). Arrows indicate branches. (For detailed explanation, see SOM.)

interactions shown in (A), assuming inductive conditions (20). Circles indicate flowers, color-coded according to *veg* levels. White, normal flower; yellow/green, shootlike flower). Arrows indicate branches. (For detailed explanation, see SOM.)

Fig. 5. (A to E) Two-dimensional fitness landscapes. Fitness levels are indicated by height and color. For each genotype, fitness is calculated over seasons with an average duration T_d and standard deviation σ , assuming that fraction θ of mature plants survives from one season to the next. Plants illustrate the architecture generated at time T_d . (A) For annuals with fixed growth duration, the optimal inflorescence is a panicle, represented by a single adaptive peak. (B) If T_d is reduced, the optimal architecture is a less highly branched panicle. (C) When σ is increased, two peaks arise corresponding to compound racemes and cymes. (D) With a further increase in σ , the peaks diverge. Optimal architectures are simple racemes and cymes. (E) Increased longevity θ shifts the peaks toward panicles. (F and G) Regions of high fitness in 3D fitness spaces. Horizontal sections correspond to the high-fitness regions in 2D fitness landscapes. (F) Path capturing the relation between architecture and σ . Colored sections correspond to figures (C) (top) and (D) (bottom). (G) Wormhole capturing the relation between architectures and longevity θ . Colored sections correspond to figures (D) (bottom) and (E) (top).



Wormhole capturing the relation between architectures and longevity θ . Colored sections correspond to figures (D) (bottom) and (E) (top).

considered the fitness of different architectures in the region of morphospace generated by the transient model (SOM text).

With unlimited pollination and a growing season of fixed length, the optimal inflorescence architecture is a panicle. This is because the plant can delay flowering by keeping its meristems in a vegetative state until the latest time needed for fruit production, thus maximizing branching and the number of fruits (Fig. 5, A and B). However, if the length of the growth seasons varies from year to year, a plant that delays flowering too long may fail to produce any fruits by the end of a short season. This would reduce fitness, particularly if annual seed production is essential for genotype survival. The best strategy may thus be for a plant to “hedge its bets” (27–32) and generate flowers sequentially during a season, so that some flowers are produced early if the season is short but more flowers can still be produced later if the season is long. Racemes and cymes, in which only a fraction of meristems switch to floral identity at any time, may then have higher fitness values than panicles. This is illustrated in Fig. 5, C and D, in which racemes and cymes form separate adaptive peaks in the fitness landscape for annual plants, with the positions of the peaks depending on the standard deviation of growth duration.

To the extent that the impact of variable season length increases from tropical to more temperate conditions, our model predicts higher frequencies of racemes and cymes in temperate compared with tropical climates, and the opposite trend for panicles. To test these predictions, we extracted the incidence of each architectural type in different climatic zones from the Watson and Dallwitz database of angiosperm families (33) (fig. S1A). Although this database is not ideal for our purposes, as it aggregates characters for all species in the same family, it reveals significant trends. Consistent with our predictions, cymes are relatively more frequent in more temperate conditions ($P < 0.01$), whereas panicles show the opposite trend ($P < 0.001$). Racemes are also more frequent in temperate than tropical conditions, but not significantly ($P > 0.2$).

In addition to the environmental influences, positions of peaks in the theoretical fitness landscapes depend on factors under genetic control, such as plant longevity. If a plant is perennial (i.e., lives more than 1 year), the negative impact of short seasons in an uncertain environment is reduced by spreading the risk over multiple years (Fig. 5E). We found no significant associations of raceme or panicle architectures with longevity, but cymes are significantly less common in families with only woody perennials and more common in families with annuals, as predicted (fig. S1B) ($P < 0.05$). Furthermore, this trend is observed in temperate but not tropical families. Although we have focused on the effects of environmental uncertainty, similar theoretical fitness landscapes involving interactions between internal and external factors

could also be generated by considering the effects of limitations on pollination rate.

Although these results provide a rationale for sequential patterns of flower production, they do not explain why the transient mechanism evolved rather than other developmental mechanisms. One possibility is that the transient mechanism arose more readily because it built on the pre-existing developmental process of meristem establishment. Establishing a new lateral meristem requires a period of time during which genes needed for meristem maintenance become activated (34). The transient mechanism could have arisen by coupling *veg* levels to this basic transition of meristems from a newly initiated (immature) to an established (mature) state. Thus, the evolution of the transient mechanism may reflect an earlier developmental genetic constraint that biased the variants available for selection toward this mechanism (6).

Evolution between racemes and cymes. In uncertain or pollinator-limited environments, racemes and cymes can represent equivalent adaptive solutions separated by a valley in the fitness landscape (Fig. 5, C to E). Such low-fitness valleys may have been circumvented in various ways during evolution. One is through changes in the environment. Figure 5F shows fitness landscapes that have been calculated for different degrees of environmental uncertainty and combined to form a three-dimensional (3D) fitness space, with the vertical axis representing the standard deviation in growth duration. An inverted U-shaped high-fitness region connects racemes, panicles, and cymes. Connecting paths may also occur through variation in genetically controlled factors. For example, a path emerges when plant longevity is varied along the vertical axis (Fig. 5G). Connecting paths of this type are common features of higher dimensional genotypic spaces (35, 36). We propose the general term “evolutionary wormhole” for such high-dimensional connections.

Movement along evolutionary wormholes that connect racemes and cymes appears limited, as individual genera seldom include both architectures. This constraint may arise because moving along wormholes requires coordinated changes of several parameters, involving multiple genetic steps. Large jumps between regions of high fitness are thus unlikely; moreover, gene segregation would generate low-fitness phenotypes. In addition, mutations required to move along some stretches of a wormhole, such as crossing between racemes and cymes, involve changes in regulatory interactions rather than simple loss or reduction of function. For example, creating a cyme in *Arabidopsis* would require changing the *LFY* and *TFL1* promoters or other genes that interact with *LFY*, *TFL1* or *veg*, such that *veg* becomes high in state *B* and low in state *A* (the reverse of wild type). Such mutations are likely to be rare. Thus, evolution of inflorescences is constrained by the nature of the developmental genetic mechanism, as well

as by the interaction between organism and environment.

Conclusion. The diversity of inflorescence architectures reflects an interplay between development and selection at several levels. We propose that a relatively simple developmental mechanism—the transient model—underlies the restriction of inflorescence types to a small region of morphospace. This mechanism offers a selective advantage in dealing with environmental limitations and uncertainty over a simpler mechanism that can generate only panicles. Although similar advantages might also be achieved through other developmental mechanisms, the transient mechanism may have evolved because it co-opted a previously available developmental transition from the immature to mature state of apices. Within the confines of the morphospace spanned by the transient model, inflorescence architectures cannot evolve freely but are restricted to following paths of high fitness or evolutionary wormholes. The combination of theoretical and experimental approaches described here shows how development and selection can interact during evolution to carve out biological forms from the vast space of theoretical possibilities.

References and Notes

- G. R. McGhee, *Theoretical Morphology* (Columbia Univ. Press, New York, 1999).
- K. J. Niklas, *The Evolutionary Biology of Plants* (Univ. of Chicago Press, Chicago, 1997).
- D. M. Raup, A. Michelson, *Science* **147**, 1294 (1965).
- D. M. Raup, *J. Paleontol.* **41**, 43 (1967).
- R. Raff, *The Shape of Life* (Univ. of Chicago Press, Chicago, 1966).
- J. Maynard Smith *et al.*, *Q. Rev. Biol.* **60**, 265 (1985).
- R. O. Erickson, F. J. Michelini, *Am. J. Bot.* **44**, 297 (1957).
- I. M. H. Etherington, *Proc. R. Soc. Edinb.* **59**, 153 (1939).
- W. Troll, *Die Infloreszenzen I* (Gustav Fischer Verlag, Stuttgart, Germany, 1964).
- W. Troll, *Die Infloreszenzen II* (Gustav Fischer Verlag, Jena, Germany, 1969).
- F. Weberling, *Morphology of Flowers and Inflorescences* (Cambridge Univ. Press, Cambridge, 1992).
- P. Prusinkiewicz, A. Lindenmayer, *The Algorithmic Beauty of Plants* (Springer-Verlag, New York, 1990).
- E. A. Kellogg, in *Monocots: Systematics and Evolution*, K. L. Wilson, D. A. Morrison, Eds. (CSIRO, Collingwood, Australia, 2000), pp. 84–88.
- G. G. Simpson, C. Dean, *Science* **296**, 285 (2002).
- T. Sachs, *Plant Cell Environ.* **22**, 757 (1999).
- D. Barthelemy, *Acta Biotheor.* **39**, 309 (1991).
- L. E. Gatsuk, O. V. Smirnova, L. I. Vorontsova, L. B. Zaugol'nova, L. A. Zhukova, *J. Ecol.* **68**, 675 (1980).
- F. Parcy, K. Bomblies, D. Weigel, *Development* **129**, 2519 (2002).
- D. Weigel, J. Alvarez, D. R. Smyth, M. F. Yanofsky, E. M. Meyerowitz, *Cell* **69**, 843 (1992).
- E. A. Schultz, G. W. Haughn, *Development* **119**, 745 (1993).
- D. Bradley, O. Ratcliffe, C. Vincent, R. Carpenter, E. Coen, *Science* **275**, 80 (1997).
- O. J. Ratcliffe, D. J. Bradley, E. S. Coen, *Development* **126**, 1109 (1999).
- S. Shannon, D. R. Meeks-Wagner, *Plant Cell* **3**, 877 (1991).
- O. J. Ratcliffe *et al.*, *Development* **125**, 1609 (1998).
- M. A. Blazquez, L. N. Soowal, I. Lee, D. Weigel, *Development* **124**, 3835 (1997).
- J. Long, M. K. Barton, *Dev. Biol.* **218**, 341 (2000).
- K. R. Hopper, T. Rosenheim, T. Prout, J. Oppenheim, *Oikos* **101**, 219 (2003).

28. D. King, J. Roughgarden, *Theor. Popul. Biol.* **21**, 194 (1982).
 29. D. Cohen, *J. Theor. Biol.* **12**, 119 (1966).
 30. D. King, J. Roughgarden, *Theor. Popul. Biol.* **22**, 1 (1982).
 31. P. Haccou, Y. Iwasa, *Theor. Popul. Biol.* **47**, 212 (1995).
 32. E. Kussell, S. Leibler, *Science* **309**, 2075 (2005).
 33. L. Watson, M. J. Dallwitz, *The Families of Flowering Plants: Descriptions, Illustrations, Identification, and Information Retrieval*. Version: 29 July 2006 <http://delta-intkey.com> (1992 onward).
 34. T. Keller, J. Abbott, T. Moritz, P. Doerner, *Plant Cell* **18**, 598 (2006).
 35. A. C. Whibley *et al.*, *Science* **313**, 963 (2006).
36. S. Gavrillets, *Fitness Landscapes and the Origin of Species*, S. Levin, H. Horn, Eds., Monographs in Population Biology (Princeton Univ. Press, Princeton and Oxford, 2004).
 37. We thank J. Avondo for help with visualizing 3D fitness landscapes, M. Claus for helpful discussions on bet-hedging, and C. Thébaud for advice on inflorescence databases. This research was funded by grants from Human Frontier Science Program (E.C. and P.P.), Natural Sciences and Engineering Research Council of Canada (P.P. and L.H.), and the Biotechnology and Biological Sciences Research Council, UK (E.C.).

Supporting Online Material

www.sciencemag.org/cgi/content/full/1140429/DC1
 Materials and Methods
 SOM Text
 Fig. S1
 References
 SOM Programs

25 January 2007; accepted 30 April 2007
 Published online 24 May 2007;
 10.1126/science.1140429
 Include this information when citing this paper.

Marine Radiocarbon Evidence for the Mechanism of Deglacial Atmospheric CO₂ Rise

Thomas M. Marchitto,^{1,2,*} Scott J. Lehman,^{1,2,*} Joseph D. Ortiz,³ Jacqueline Flückiger,² Alexander van Geen⁴

We reconstructed the radiocarbon activity of intermediate waters in the eastern North Pacific over the past 38,000 years. Radiocarbon activity paralleled that of the atmosphere, except during deglaciation, when intermediate-water values fell by more than 300 per mil. Such a large decrease requires a deglacial injection of very old waters from a deep-ocean carbon reservoir that was previously well isolated from the atmosphere. The timing of intermediate-water radiocarbon depletion closely matches that of atmospheric carbon dioxide rise and effectively traces the redistribution of carbon from the deep ocean to the atmosphere during deglaciation.

Radiocarbon measurements of calendrically dated hermatypic corals (1) and planktonic foraminifera (2, 3) indicate that the radiocarbon activity ($\Delta^{14}\text{C}$) of the atmosphere during the latter part of the last glacial period [~20,000 to 40,000 years before the present (yr B.P.)] ranged from ~300 to 800 per mil (‰) higher than it was during the pre-nuclear modern era (Fig. 1C). Although reconstructions of Earth's geomagnetic-field intensity predict higher cosmogenic ^{14}C production rates during the glacial period, production was apparently not high enough to explain the observed atmospheric enrichment (2–5). Rather, a substantial fraction of the atmosphere's $\Delta^{14}\text{C}$ buildup must have been due to decreased uptake of ^{14}C by the deep ocean. This requires a concomitant ^{14}C depletion in a deep-ocean dissolved inorganic C reservoir that was relatively well isolated from the atmosphere. Renewed ventilation of this reservoir could theoretically explain the drop in atmospheric $\Delta^{14}\text{C}$ (Fig. 1C) and the rise in atmospheric CO₂ (6) across the last deglaciation. Most workers point to the Southern Ocean as a

locus of deglacial CO₂ release, based on the similarity between atmospheric CO₂ and Antarctic temperature records (6) and on numerous conceptual and numerical models (7–9). If correct, we would expect some signature of the low- ^{14}C deep-ocean C reservoir to be spread to other basins via Antarctic Intermediate Water (AAIW). Here, we report a strong radiocarbon signal of the deglacial release of old C, recorded in an intermediate-depth sediment core from the northern edge of the eastern tropical North Pacific.

Intermediate water $\Delta^{14}\text{C}$ reconstruction. Marine sediment multi-core/gravity-core/piston-core triplet from sediment layer MV99-MC19/GC31/PC08 was raised from a water depth of 705 m on the open margin off the western coast of southern Baja California (23.5°N, 111.6°W) (10). The site is today situated within the regional O₂ minimum zone that exists because of a combination of high export production and poor intermediate-water ventilation. Various sediment properties in MC19/GC31/PC08 vary in concert with the so-called Dansgaard-Oeschger (D-O) cycles that characterized the Northern Hemisphere climate during the last glacial period (11). Originally discovered in Greenland ice cores, D-O cycles also exist in a number of lower-latitude locations that were probably teleconnected to the North Atlantic region through the atmosphere (2, 12, 13). Off the coast of Baja California, the sedimentary concentrations of organic C, Cd, Mo, and benthic foraminifera all decreased sharply during D-O stadials (cold periods in Greenland) (11, 14). Together, these proxies are consistent with reduced productivity during stadials, caused

by either decreased coastal upwelling or a deepening of the regional nutrient related to the mean state of the tropical Pacific (11).

Diffuse spectral reflectance (DSR) provides a 1-cm resolution stratigraphy for GC31/PC08. After R-mode factor analysis, the third factor of DSR (Fig. 1A) exhibits the strongest correlation to the productivity proxies and to Greenland climate (11). We used this DSR record to apply a calendar-age model to MC19/GC31/PC08, based on correlation to $\delta^{18}\text{O}$ (an air-temperature proxy) in Greenland ice core GISP2 (Greenland Ice Sheet Project 2) (15). Resulting calendar ages were then combined with 50 benthic foraminiferal radiocarbon ages [19 of which were published previously (10)] to calculate age-corrected intermediate-water $\Delta^{14}\text{C}$ (16). To evaluate the partitioning of ^{14}C between the atmosphere and the ocean, we compared intermediate-water $\Delta^{14}\text{C}$ to that of the atmosphere (Fig. 1C), as reconstructed from tree rings (17), U-Th-dated corals (1, 17), and planktonic foraminifera from Cariaco Basin off Venezuela (3). Calendar ages for Cariaco Basin were originally based on the correlation of lithologic climate proxies to the GISP2 $\delta^{18}\text{O}$ record (2), which has been layer-counted with visual and chemical techniques (15). However, Hughen *et al.* (3) recently demonstrated that the Cariaco Basin ^{14}C calibration yields much better agreement with coral results older than ~22,000 yr B.P. when an alternate age model is used, based on correlation to the U-Th-dated Hulu Cave speleothem $\delta^{18}\text{O}$ record from eastern China (13). Because DSR in GC31/PC08 is more similar to the Greenland isotope record than to the lower-resolution Hulu Cave record, we continued to use the GISP2 correlation but applied simple provisional age adjustments to GISP2 older than 23,400 yr B.P., using four tie points to Hulu Cave (Fig. 1B and fig. S1). We do not suggest that this age model is necessarily superior to the original one (15), but this exercise is necessary for comparing our data to the most recent (and most consistent) atmospheric $\Delta^{14}\text{C}$ reconstructions (1, 3, 17). The resulting age model for MC19/GC31/PC08, based on 21 tie points, yields a very constant sedimentation rate (fig. S2) and gives us confidence that our calendar-age assignments for ^{14}C samples between tie points are reliable to within a few hundred years (table S1).

Baja California intermediate-water radiocarbon activities are plotted in red in Fig. 1C. The

¹Department of Geological Sciences, University of Colorado, Boulder, CO 80309, USA. ²Institute of Arctic and Alpine Research, University of Colorado, Boulder, CO 80309, USA. ³Department of Geology, Kent State University, Kent, OH 44242, USA. ⁴Lamont-Doherty Earth Observatory, Columbia University, Palisades, NY 10964, USA.

*These authors contributed equally to this work.

†To whom correspondence should be addressed. E-mail: tom.marchitto@colorado.edu

‡Present address: Environmental Physics, Institute of Biochemistry and Pollutant Dynamics, Eidgenössische Technische Hochschule Zürich, 8092 Zürich, Switzerland.

Supporting Online Material for

Evolution and Development of Inflorescence Architectures

Przemyslaw Prusinkiewicz, Yvette Erasmus, Brendan Lane, Lawrence Harder and Enrico Coen*

*To whom correspondence should be addressed: enrico.coen@bbsrc.ac.uk

This file includes:

Materials and methods

Description of general models for Fig. 2

Description of *Arabidopsis* model for Fig. 4

Detailed description of phenotypes in Fig. 4 (B-H)

Fitness calculations for Fig. 5

Figure S1

Supplemental references

Materials and methods

Arabidopsis plants of various genotypes were grown as described previously (S1) to count lateral branches for comparison with the simulation model. The model was written in the L-system-based programming language L+C (S2), and the simulations were performed with the L-studio modelling environment (S3). L-studio was also used to compute fitness data and visualize 2D fitness landscapes. Images of 3D fitness spaces were obtained with volume visualization software QtVolView (S4). Statistical analysis of the incidence of cymes, racemes and panicles among angiosperm families (data from (S5)) assessed generalized linear models (S6) as implemented in the genmod procedure of SAS/STAT®, version 9.1.3 (S7).

Description of general models for Fig.2

A parameterized model was specified in the L-system-based language L+C (2). Inflorescences are described in terms of four types of module: apex, internode, leaf and

flower. Each module has a set of local attributes associated with it, such as internal state and *veg*, whose values can vary at different stages of development. At the start of growth, the inflorescence consists of two modules: an apex and an internode. The number of modules increases during growth through the application of L-system productions. At regular time intervals (plastochrons), the fate of each apex is decided by choosing between two possible outcomes: (a) production of a metamer (i.e., a structure consisting of an internode and a lateral apex with a subtending leaf), or (b) transformation into a flower. The decision is based on the level of *veg*, which in turn depends on global time t and state of the apex. If *veg* in an apex is above a threshold value V_K , the apex will produce a metamer, otherwise it will be replaced by a flower that terminates further growth of the axis.

If *veg* stays at a constant high level, repeated application of the production rules gives rise to the reiterated branching structure shown in Fig.2A. For the illustration in Fig.2B, *veg* is computed as an exponential function of time t : $veg = \alpha e^{-\beta t}$, where α and β are constants. This gives rise to a panicle, since all apices reach the threshold value V_K at the same time $T = -\frac{\ln(V_K / \alpha)}{\beta}$.

To generate other architectural classes, a further module attribute is introduced: apical state. For the first of the resulting models (Fig.2C), apices are assumed to maintain the state they had at the time when they were formed. The apex of the main axis is in state A , while all lateral apices are in state B . The level of *veg* in state A apices is computed as above: $veg = \alpha e^{-\beta t}$. For apices in state B , *veg* is reduced by a constant amount δ : $veg = \alpha e^{-\beta t} - \delta$. This means that state apices in state A will reach the threshold value V_K at time $T_A = -\frac{\ln(V_K / \alpha)}{\beta}$ (as in Fig.2B), while apices in state B will reach the threshold at the

earlier time $T_B = -\frac{\ln((V_K + \delta)/\alpha)}{\beta}$. An inflorescence consisting of an upper simple raceme and paniculate lower branches that terminate in flowers results. The size of each part depends on the values of constants α , β , δ , and V_K .

Racemes with indeterminate branches are generated with the transient model. As in the previous model, only state B apices are generated in lateral positions. If veg in the state B apex falls below the threshold value V_K within time interval T_T from the inception of this apex, the apex assumes flower identity. In contrast, if veg does not reach V_K within this period of time, the apex reverts to state A and assumes vegetative identity. The parameter T_T thus determines the period of the immaturity of the apex, during which its fate is determined. This model generates a raceme with indeterminate branches as shown in Fig. 2D.

The transient model can also generate cymes. This is achieved by giving a negative value to the constant δ in the equation for evaluating veg in state B (see above). As a result, state B apices have a higher level of veg than state A apices. If the duration of state B is more than one plastochron ($T_T > 1$), state B apex will have time to generate one or more branches before reverting to state A , yielding a cyme (Fig. 2E).

Description of *Arabidopsis* model for Fig.4

Aims and overview

The main aim of the *Arabidopsis* model is to relate the generic transient model of inflorescence development to molecular genetic mechanisms. The resulting model should account for data on mutant and over-expression phenotypes as well as for gene expression data for key architectural genes. The second aim is to illustrate how molecular genetic data

can be incorporated into architectural models to capture the two-way interaction between gene activity and growth. The third aim is to capture additional features observed in mutants such as the generation of shoot-like flowers and the presence or absence of bracts.

The modelling framework is similar to that described above for the generic transient model. The key difference is that *veg* in an apex is now a function of gene activity as well as time. Gene activity in an apex in turn depends on time, gene interactions, and apex state (*A* or *B*). To capture these interactions, each apex has an additional series of attributes involved in computing gene activity levels, whose values can change as the plant grows. Because many of the details on concentrations and kinetics of gene products are unknown, some simplifications and arbitrary assumptions have been made while constructing the model. For example, we adopt simple linear or hyperbolic functions to capture promoting or inhibitory effects. We also assume that many interactions are combined multiplicatively, by analogy with the law of mass action. At present there are no experimental data to quantitatively evaluate our assumptions, and more realistic functions could be incorporated in the future as more become known. However, this does not detract from the main aims of the model described above.

In the following sections we outline: (1) the basic mathematical functions employed in the model, (2) the way gene activity levels are computed as a function of time, gene interactions and module state, and (3) the effects of *veg* and the way it is computed.

(1) Basic functions

Dependence on global time t measured in plastochrons is implemented with a piecewise linear function $E_{T_{ON}, T_M}(t)$:

$$E_{T_{ON}, T_M}(t) = \begin{cases} 0 & \text{for } t < T_{ON} \\ \frac{t - T_{ON}}{T_M - T_{ON}} & \text{for } T_{ON} \leq t \leq T_M \\ 1 & \text{for } t > T_M \end{cases} \quad (1)$$

This function increases linearly with time from zero at $t = T_{ON}$ to one at $t = T_M$.

Promoting effects are captured with:

$$Y_L = P_{Y \leftarrow X}(X_L) = 1 + K_{Y \leftarrow X} X_L, \quad (2)$$

where $P_{Y \leftarrow X}(X_L)$ denotes the promoting effect of factor X on factor Y , and $K_{Y \leftarrow X} > 0$ is a constant that determines the sensitivity of factor Y to the factor X . The subscripted variables X_L and Y_L denote the levels of the corresponding factors; other subscripts are introduced further down. This function evaluates to 1 when $X_L=0$ and increases linearly with X_L . Factor X_L could be level of gene activity, *veg* or state.

Inhibitory effects are captured with the function:

$$Y_L = I_{Y \leftarrow X}(X_L) = \frac{1}{1 + K_{Y \leftarrow X} X_L}, \quad (3)$$

where $I_{Y \leftarrow X}(X_L)$ denotes the inhibitory effect of factor X on factor Y , and $K_{Y \leftarrow X} > 0$ is a constant that determines the sensitivity of factor Y to factor X . This function evaluates to 1 when $X_L=0$ and decreases hyperbolically with increasing X_L . As with the promoting function, factors X_L and Y_L could be levels of gene activity, *veg* or state.

Factors may act in a chain. A factor Y , influenced by factor X , may itself affect another factor Z . For example, the expression $I_{Z \leftarrow Y}(I_{Y \leftarrow X}(X_L))$ may represent a situation where a transcription factor X down-regulates the expression of a gene Y , which in turn down-regulates the expression of a gene Z .

A combined effect of several factors X_1, X_2, \dots, X_n acting in parallel on the same factor Y is simulated by multiplying individual contributions. For example, the product

$P_{Y \leftarrow X_1}(X_{1L})P_{Y \leftarrow X_2}(X_{2L})I_{Y \leftarrow X_3}(X_{3L})$ represents a promoting effect of factors X_1 and X_2 , combined with an inhibitory effect of factor X_3 , on factor Y .

(2) Gene activity levels

The overall level (G_L) of the product of a gene G reflects the combined activities of the promoter (G_P) and coding (G_C) regions. This is calculated as the product

$$G_L = G_P G_C . \quad (4)$$

We assume that, for a constitutive promoter with a wild-type coding region, $G_P = G_C = 1$, so the overall gene activity level G_L is also 1. If G_P and G_C are both reduced to 0.1, G_L is equal to 0.01, which represents 10% of a transcription factor with 10% activity producing 1% overall activity. The advantage of this formulation is that it is easy to swap promoters between genes. Thus, activity level of a construct with promoter of gene G_1 driving coding region of gene G_2 can be computed as $G_{1P}G_{2C}$. The formulation also allows alleles with different strengths to be generated simply by varying G_C . If more than one gene with the same coding region is present, then the value of G_L corresponds to the sum of the individual gene activity levels.

As with the general models described in the earlier section, each apex in the model creates three new modules every plastochron: an internode, axillary apex and subtending leaf (the inflorescence starts with a single apex). The pattern of inflorescence development largely depends on how the *LFY* and *TFL1* promoters are regulated within the modules. The promoter levels depend on time, gene regulatory interactions and module state. These dependencies are described below.

Promoter level as a function of time

If the promoter level of a gene G depends only on time, we have:

$$G_P(t) = E_{T_{ON}, T_M}(t). \quad (5)$$

Promoter level as a function of gene interactions

The inhibitory effect of *TFLI* on the *LFY* promoter is expressed as:

$$LFY_P = I_{LFY \leftarrow TFL}(TFL_L). \quad (6)$$

Similarly, the inhibitory effect of *LFY* on the *TFLI* promoter is expressed as:

$$TFL_P = I_{TFL \leftarrow LFY}(LFY_L). \quad (7)$$

It is known from experiments (S8,S9) that the *LFY* and *TFLI* expression can gradually increase over time. This effect is captured by combining Eqs. 5, 6 and 7 as follows:

$$LFY_P = E_{T_{ON}, T_M}(t) I_{LFY \leftarrow TFL}(TFL_L), \quad (8)$$

$$TFL_P = E_{T_{ON}, T_M}(t) I_{TFL \leftarrow LFY}(LFY_L). \quad (9)$$

Promoter level as a function of module state

In the model, module state has an impact on the promoter levels of *TFLI* and *LFY* genes. The effect of state on *LFY* gene expression is captured by assuming that the *LFY* promoter is less sensitive to inhibition by *TFLI* in state *B* than in state *A*. To this end, we use a function $I_{(LFY \leftarrow TFL) \leftarrow s}(s_L)$, with $s_L = 0$ in state *A*, and $s_L = 1$ in state *B*. The overall level of the *LFY* promoter is expressed by the following modification of Eq. 8:

$$LFY_P = E_{T_{ON}, T_M}(t) I_{LFY \leftarrow TFL}(I_{(LFY \leftarrow TFL) \leftarrow s}(s_L) TFL_L) \quad (10)$$

The sensitivity coefficient has value $K_{(LFY \leftarrow TFL) \leftarrow s} = 100$ (c.f. Eq. 3). With this value, the impact of *TFLI* on the promoter level of *LFY* in state *B* is reduced to 1% of its sensitivity in state *A*.

The effect of module state on the promoter level of gene *TFL1* is captured with a function $I_{TFL \leftarrow s}(s_L)$, where $s_L = 0$ in state *A*, and $s_L = 1$ in state *B*, as previously assumed for the function $I_{(LFY \leftarrow TFL) \leftarrow s}(s_L)$. Taking time and down-regulation of *TFL1* by *LFY* into consideration (Eq. 9), the overall level of the *TFL1* promoter is expressed by the formula:

$$TFL_P = E_{T_{ON}, T_M}(t) I_{TFL \leftarrow LFY}(LFY_L) I_{TFL \leftarrow s}(s_L). \quad (11)$$

The sensitivity constant $K_{TFL \leftarrow s}$ (Eq. 3) is equal to 9. For a given level of the *LFY* protein, the level of the *TFL1* promoter in state *B* is thus reduced to 10% of the level in state *A*.

(3) Effect and control of *vegetativeness*

Effects of veg on plant development

As with the general models, if *veg* in an apex falls below the value V_K , the apex is replaced by a determinate structure. For the *Arabidopsis* model, the determinate structure could correspond to a shoot-like flower or a normal flower. Shoot-like flowers correspond to intermediate levels of *veg* which are below the threshold V_K required for determinacy but above the level in normal flowers. Wild-type plants show a sharp transition up the plant from producing shoots to normal flowers in lateral positions, while *lfy* or *35S::TFL1* plants show a more gradual transition that results in the production of intermediate determinate structures with some shoot-like features. The transitions in different genotypes can be captured by assuming that once a determinate structure is formed, its *veg* level continues to be computed for a short period (1 plastochron) and is then fixed. In wild-type plants, this period of continuing *veg* evaluation gives time for shoot-like intermediates to be bypassed. By contrast, in *35S::TFL1* plants *veg* levels decrease more slowly, and a gradual transition from mutant to normal flowers occurs. In *lfy* mutants *veg* also falls slowly, but furthermore it does not attain the low levels of normal flowers (see below); this leads to intermediate shoot-like characteristics of all determinate structures.

The levels of *veg* affect not only the switch from apices to flowers, but also the extent to which subtending leaves develop. Wild-type *Arabidopsis* plants have leaves subtending lateral shoots but not flowers. In *lfy* mutants, leaves or bracts typically subtend both the shoots and shoot-like flowers that grow in place of normal flowers; this indicates that *LFY* plays a role in preventing bract development (*S10*, *S11*). Plants over-expressing *TFL1* form intermediate structures comprising shoots without subtending leaves (*S12*, *S13*). In the model, these features are captured by computing *veg* level for a newly initiated leaf as it is done for the lateral apex that is subtended by this leaf. The justification is that both apex and subtending leaf emerge from a common primordium or anlage (*S11*, *S14*). The computation of *veg* continues for a short period of time, T_L , after which *veg* level in the leaf no longer changes. Leaf development is arrested when the level of *veg* in the leaf falls below a threshold value ($veg \leq V_{LF}$) and the level of *LFY* exceeds another threshold value ($LFY_L > LFY_{TH}$).

Control of veg in various genotypes

The level of *veg* in the model is a function of time (plant age) and gene activity. Gene activity in turn depends on time, gene interactions and module state (see earlier section). The architectures generated by the model depend on the times T_A and T_B , at which the *veg* values fall below the intermediate value V_K .

Wild type

To account for the phenotypes of various genotypes (see below), the level of *veg* in an apex of wild type is computed as a function of gene activity and time:

$$veg = \max \begin{cases} V_0 - K_{BAS} P_{veg \leftarrow LFY}(LFY_L) I_{veg \leftarrow TFL}(TFL_L)t \\ V_M I_{veg \leftarrow LFY}(LFY_L) \end{cases} \quad (12)$$

where V_0 is the initial level of veg . To account for eventual formation of mutant flowers in $lfy\ tfl1$ mutants, veg declines with time with a basic rate constant K_{BAS} . This time-dependent decrease in veg is promoted by LFY and inhibited by $TFL1$. In addition, veg is kept above a minimal level, which decreases as LFY activity increases. This is captured by LFY inhibiting the minimal level of veg (veg_m) such that the minimum level is low when LFY is on, and is raised to V_M when LFY is off. In wild type, veg values in state B meristems are lower than veg values in state A meristems, due to a difference in expression of LFY and $TFL1$. Consequently, $T_B < T_A$, resulting in a racemose inflorescence (Fig. 4E).

$lfy\ tfl1$ double mutant

In the $lfy\ tfl1$ double mutant the inflorescence has a few branches which terminate in mutant determinate structures. Equation 12 evaluates to:

$$veg = \max \begin{cases} V_0 - K_{BAS} t \\ V_M \end{cases} \quad (13)$$

Thus veg declines at a linear basic rate K_{BAS} and eventually reaches a minimum value V_M . The resulting determinate structures will be mutant because veg does not fall below V_M . In this double mutant, there is no difference in veg values between state A and state B meristems, resulting in a panicle (Fig. 4B).

$tfl1$ single mutant

Here Equation 12 takes the form:

$$veg = \max \begin{cases} V_0 - K_{BAS} P_{veg \leftarrow LFY}(LFY_L)t \\ V_M I_{veg_m \leftarrow LFY}(LFY_L) \end{cases} \quad (14)$$

The function $P_{veg \leftarrow LFY}(LFY_L)$ captures the promotive effect of LFY_L on decline in veg , and

$I_{veg_m \leftarrow LFY}(LFY_L)$ describes the effect of LFY_L on reducing the minimum value of veg .

According to this equation, the *tfl1* single mutant inflorescence forms flowers earlier than the *lfy tfl1* double, and the flowers are wild-type. Furthermore, there is no difference in the expression of *LFY* between meristems in state *A* and *B* (c.f. Eq. 10), $T_B = T_A$, and a panicle results (Fig. 4C). If *TFL1* activity is halved rather than eliminated (which in reality would correspond to a *TFL1/tfl1* heterozygote), the phenotype generated by the model differs from wild-type by just one branch. Given that the number of branches in real wild-type plants varies between 1 and 3, the model captures the recessive nature of the *tfl1* mutation.

lfy single mutant

Here equation 12 evaluates to:

$$veg = \max \begin{cases} V_0 - K_{BAS} I_{veg \leftarrow TFL} (TFL_L) t \\ V_M \end{cases} \quad (15)$$

Thus in a single *lfy* mutant, *veg* will decline at a slow rate according to the value of $K_{veg \leftarrow TFL}$ and, since *veg* cannot fall below V_M , the determinate structures eventually produced will be shoot-like.

In *lfy*, *veg* values in state *B* meristems are lower than *veg* values in state *A* meristems, due to a difference in expression of *TFL1*. Consequently, $T_B < T_A$, resulting in a racemose inflorescence, although it is more highly branched than wild type because the difference between T_B and T_A is not as large (Fig. 4D). If *LFY* activity is halved rather than eliminated (which in reality would correspond to a *LFY/lfy* heterozygote), the phenotype generated by the model differs from wild-type by 2 extra branches. This is consistent with experimental observations showing that *LFY/lfy* heterozygotes can have 1 or 2 more branches than wild-type (S8).

Effects of constitutive expression

For constitutive *LFY*, *veg* declines rapidly according to Equation 12, both because LFY_L is always high and because TFL_L is low due to the inhibitory effect of *LFY* on the *TFLI* promoter (Eq. 11). Thus the inflorescence rapidly terminates with a flower (Fig. 4F).

For constitutive *TFLI*, *veg* declines slowly according to Equation 15 both because TFL_L is high and because LFY_L is low due to the inhibitory effect of *TFLI* on the *LFY* promoter (Eq. 10). Eventually flowers will form because the *LFY* promoter in state *B* is relatively insensitive to the effects of *TFLI* (Fig.4G). Moreover, the inflorescence will be a raceme, since $T_B < T_A$. The transition in subtending leaves is also captured by the model as the threshold *veg* level at which leaves abort is higher than the threshold for formation of determinate structures ($V_{LF} > V_K$). Flowers are no longer formed in the model if constitutive *TFLI* is combined with *lfy*, in agreement with observations (Fig. 4H).

The L-system code for the general and *Arabidopsis* models is available on request.

List of parameters

$V_0 = 100$	initial <i>veg</i> level
$V_M = 10$	minimum <i>veg</i> level while <i>LFY</i> is off
$V_{LF} = 35$	<i>veg</i> level below which leaves abort
$V_K = 20$	<i>veg</i> level below which apex switches to determinate structure
$K_{BAS} = 13$	basic rate constant for decline in <i>veg</i>
$LFY_{TH} = 0.1$	level of <i>LFY</i> gene activity above which leaf can abort
$K_{LFY \leftarrow TFL} = 10$	inhibitory effect of <i>TFLI</i> on <i>LFY</i>
$K_{TFL \leftarrow LFY} = 5$	inhibitory effect of <i>LFY</i> on <i>TFLI</i>
$K_{(LFY \leftarrow TFL) \leftarrow s} = 100$	inhibitory effect of state <i>B</i> on sensitivity of <i>LFY</i> to <i>TFLI</i> inhibition
$K_{TFL \leftarrow, s} = 9$	inhibitory effect of state <i>B</i> on <i>TFLI</i> expression

$K_{veg \leftarrow LFY} = 3$	promotive effect of <i>LFY</i> on rate of <i>veg</i> decline
$K_{veg \leftarrow TFLI} = 19$	inhibitory effect of <i>TFLI</i> on rate of <i>veg</i> decline
$K_{vegm \leftarrow LFY} = 10$	inhibitory effect of <i>LFY</i> on minimum <i>veg</i>
$T_L = 0.5$	age in plastochrons when leaf abortion is sensitive to <i>veg</i>
$T_T = 1$	duration in plastochrons of state <i>B</i> (immature)
$T_{ON} = 2$	global time in plastochrons when <i>LFY_p</i> and <i>TFLP_p</i> begin to be activated
$T_M = 8$	global time in plastochrons at which <i>LFY_p</i> and <i>TFLP_p</i> are fully activated

Detailed description of phenotypes in Fig. 4 (B-H)

(B) Ground state (*lfy tfl1* double mutant). *Veg* declines at a basic rate until it reaches an intermediate level in which shoot-like flowers are formed (*S10*). (C) Effect of adding *LFY* to the ground state (*tfl1* mutant). The level of *LFY* increases with plant age in both state *B* and state *A* meristems, causing a decrease of *veg* and formation of normal flowers (*S10*, *S15*). In addition, *LFY* combined with low *veg* suppresses development of the bract supporting the second lateral flower. (D) Effect of adding *TFLI* to the ground state (*lfy* mutant). *TFLI* is expressed in the main state *A* meristem, delaying the formation of a terminal shoot-like flower. In meristems giving rise to the lower branches, *TFLI* expression is initially low (state *B*) and returns to the high level as meristems switch to state *A*. The resulting transient drop in *veg* is insufficient to initiate shoot-like flowers. The drop reaches lower *veg* levels as the plant ages, eventually causing initiation of shoot-like flowers (*S10*, *S11*). As *veg* declines relatively slowly, the first few determinate structures have higher *veg* (darker green) than later ones, capturing the gradual transition to more “flower-like” structures in *lfy* mutants. (E) Wild type. The effects of *LFY* and *TFLI* are amplified through their regulatory interactions (*S13*). As a result, the number of lateral branches decreases with respect to the *lfy* mutant to two, and normal flowers are formed as in the *tfl1* mutant. (F) Effect of ectopic expression of *LFY*. Constitutive *LFY* causes a fast

decrease in *veg*, both because of *LFY*'s direct effect on *veg* and because *LFY* inhibits *TFL1* expression. Consequently, the main meristem is transformed into a flower before the lateral apices have been formed (S16). **(G)** Effect of ectopic expression of *TFL1*. Constitutive *TFL1* reduces the drop of *veg* in state *B* lateral meristems, leading to the formation of many branches. The transient drop of *veg* in state *B* meristems created along each axis eventually becomes sufficient to suppress bracts, then to initiate shoot-like flowers (green to yellow), and finally to initiate development of normal flowers (white) (S13) **(H)** Ectopic expression of *TFL1* in a *lfy* mutant background. Level of *veg* drops slowly in all meristems giving a structure that repetitively branches without forming flowers (S13).

Fitness calculations used to generate Fig. 5

We consider the development of a population of plants captured by the transient model. The genotype of each plant is summarily represented by two parameters, T_A and T_B , as described in the main text and in the Description of General Models section of the Supplementary Information. The total number of flowers borne by a plant with the genotype (T_A, T_B) during a growth season of duration Ω is denoted $f_{A,B}(\Omega)$. For simplicity, we assume that the number of seeds produced by a plant is the same as the number of its flowers.

The number of plants present during the next season, relative to the number present during the current season, is calculated with the equation

$$\varphi_{A,B}(\Omega) = \theta + \kappa f_{A,B}(\Omega),$$

where θ is the fraction of plants that will survive past the current season, and κ is the fraction of seeds that germinate. (We used $\kappa = 1\%$ in the simulations shown in Figure 5.)

Over a sequence of N seasons of durations $\Omega_1, \Omega_2, \dots, \Omega_N$, the number of plants produced from a single initial plant of genotype (T_A, T_B) will thus be

$$\varphi_{A,B}(\Omega_1)\varphi_{A,B}(\Omega_2)\dots\varphi_{A,B}(\Omega_N) = \prod_{i=1}^N \varphi_{A,B}(\Omega_i).$$

The average per-year growth of the population of seeds is the geometric mean of this number (*S17, S18*):

$$\bar{\varphi}_{A,B}(\Omega_1, \Omega_2, \dots, \Omega_N) = \left(\prod_{i=1}^N \varphi_{A,B}(\Omega_i) \right)^{\frac{1}{N}}.$$

If the sequence of growth duration is not given, but growth durations are drawn from some distribution p , the average-per-year population growth is the weighted geometric mean,

$$w_{A,B}(p) = \left(\prod_{\Omega} (\varphi_{A,B}(\Omega))^{p(\Omega)} \right)^{\frac{1}{\sum_{\Omega} p(\Omega)}},$$

where $p(\Omega)$ is the probability that a season will have duration Ω . (The sum of probabilities $\sum_{\Omega} p(\Omega)$ may be less than one, for example, when probabilities $p(\Omega)$ are computed with a normal distribution truncated to a finite interval.) The above formulas are often represented in logarithmic forms:

$$\log \bar{\varphi}_{A,B}(\Omega_1, \Omega_2, \dots, \Omega_N) = \frac{1}{N} \sum_{i=1}^N \log \varphi_{A,B}(\Omega_i)$$

and

$$\log w_{A,B}(p) = \frac{1}{\sum_{\Omega} p(\Omega)} \sum_{\Omega} p(\Omega) \log \varphi_{A,B}(\Omega),$$

which relate the geometric means to the more familiar arithmetic means. Given a distribution of growth-season durations, p , we use the average-per-year population growth $w_{A,B}(p)$ as the measure of fitness (*S17*) of genotype (T_A, T_B) .

In constructing Figure 5, we assumed normal distribution of growth season length, truncated to the interval from 0 to 20 plastochrons and sampled discretely with a resolution of one plastochron. The probability density $f(\omega)$ of the normal distribution with mean Ω_d and standard deviation σ is:

$$f(\omega) = \frac{1}{\sigma\sqrt{2\pi}} \exp\left(-\frac{1}{2}\left(\frac{\omega - \Omega_d}{\sigma}\right)^2\right).$$

For growth durations expressed in plastochrons, the probability of a growth duration of Ω plastochrons is thus

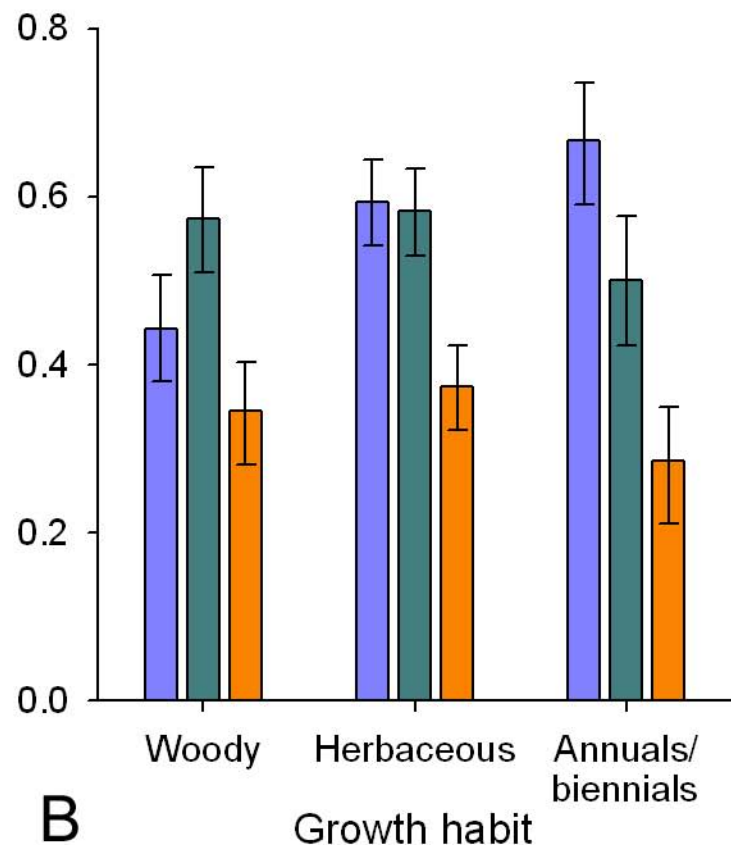
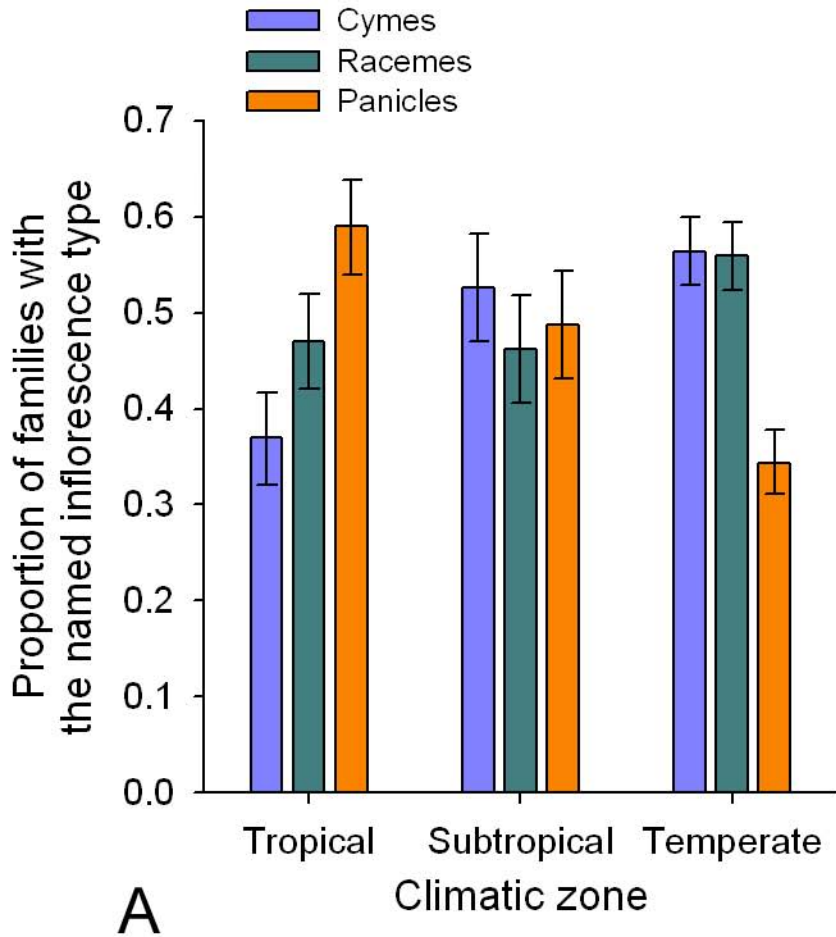
$$p(\Omega) = \int_{\Omega}^{\Omega+1} f(\omega) d\omega \approx f(\Omega).$$

Figure S1

Incidence of cymes, racemes and panicles (mean \pm SE) among angiosperm families in different climatic zones (**A**) and with contrasting growth habits (**B**). Data are from (S5) (thyrses are included within the panicle class in this database).

(**A**) Cymes are relatively more common in families with temperate ranges, whereas panicles occur most frequently in tropical families. Family categories include species with only tropical ranges, some species with subtropical, but not temperate ranges, or some species with temperate ranges ($n = 373$ families). Analyses of generalized linear models (S6) found contrasting associations for the three inflorescence types. Climate affects the proportion of families with cymes (likelihood-ratio test, $G_2 = 10.23$, $P < 0.01$), and panicles ($G_2 = 17.27$, $P < 0.001$), but not with racemes ($G_2 = 3.22$, $P > 0.2$). Cymes occur more commonly among temperate families than among tropical families, whereas panicles exhibit the opposite pattern.

(B) Cymes are more common in families with some annual species than in exclusively woody families. Family categories include: only woody species, some or only herbaceous perennial species, or some annual/biennial species. Analysis of all available families from this perspective was complicated by the strong association between growth habit and climate range ($G_4 = 89.66$, $P < 0.001$) that exists because exclusively tropical families tend to be exclusively woody and do not include annual or biennial species. Because our hypothesis for the effects of longevity applies primarily in environments with relatively brief growing periods, we restricted our analyses of the influences of growth habit to families with some temperate species ($n = 194$ families). These analyses found significant associations with growth habit for cymes ($G_2 = 5.78$, $P > 0.05$: test for the expected pattern of woody < herbaceous perennials < annuals/biennials, $G_1 = 4.48$, $P < 0.05$). In contrast, neither the incidence of racemes ($G_2 = 0.84$, $P > 0.5$) nor panicles ($G_2 = 1.00$, $P > 0.5$) varied significantly with growth habit for families with some temperate families. A separate analysis (not illustrated in the figure) that compared the effects of growth habit on the incidences of different inflorescence types between families with only tropical species and those with only temperate species detected a significant interaction for cymes ($G_1 = 5.85$, $P < 0.025$), but not for racemes ($G_1 = 0.25$, $P > 0.5$), or panicles ($G_1 = 0.16$, $P > 0.5$). All of the six families in this analysis that include some annual species have exclusively temperate ranges, and five of these families include cymose species. This incidence of cymes is significantly higher than that observed for exclusively woody, temperate families (mean = 14.3%, lower standard error = 7.1%, upper standard error = 12.1%: $G_1 = 6.49$, $P < 0.05$), which also include cymose species less commonly than temperate families with some perennials (mean = 57.1%, LSE = 13.4%, USE = 12.5%: $G_1 = 4.94$, $P < 0.05$). In contrast, cymes were equally uncommon among tropical families with exclusively woody species (mean = 36.7%, LSE = 5.2%, USE = 5.6%) or some perennial species (mean = 27.8%, LSE = 9.3%, USE = 11.7%: $G_1 = 0.51$, $P > 0.4$).



Supplemental references

- S1. L. Mundermann, Y. Erasmus, B. Lane, E. Coen, P. Prusinkiewicz, *Plant Physiol* **139**, 960 (2005).
- S2. R. Karwowski, P. Prusinkiewicz, *Electronic Notes in Theoretical Computer Science* **86**, 19 (2003).
- S3. P. Prusinkiewicz, *Acta Horticulturae* **630**, 15 (2004).
- S4. K. Lee *et al.*, *Plant Cell* **18**, 2145 (2006).
- S5. L. Watson, M. J. Dallwitz, *The families of flowering plants: descriptions, illustrations, identification, and information retrieval. Version: 29th July 2006* <http://delta-intkey.com> (1992 onwards).
- S6. P. McCullagh, J. A. Nelder, *Generalized linear models* (Chapman & Hall, London, ed. 2nd, 1989).
- S7. C. SAS Institute Inc. SAS OnlineDocumentaion 9.1.3. (SAS Institute Inc., NC, 2004).
- S8. M. A. Blazquez, L. N. Soowal, I. Lee, D. Weigel, *Development* **124**, 3835 (1997).
- S9. D. Bradley, O. Ratcliffe, C. Vincent, R. Carpenter, E. Coen, *Science* **275**, 80 (1997).
- S10. E. A. Schultz, G. W. Haughn, *Development* **119**, 745 (1993).
- S11. D. Weigel, J. Alvarez, D. R. Smyth, M. F. Yanofsky, E. M. Meyerowitz, *Cell* **69**, 843 (1992).
- S12. O. J. Ratcliffe *et al.*, *Development* **125**, 1609 (1998).
- S13. O. J. Ratcliffe, D. J. Bradley, E. S. Coen, *Development* **126**, 1109 (1999).
- S14. J. Long, M. K. Barton, *Developmental Biology* **218**, 341 (2000).
- S15. S. Shannon, D. R. Meeks-Wagner, *Plant Cell* **3**, 877 (1991).
- S16. D. Weigel, O. Nilsson, *Nature* **377**, 495 (1995).
- S17. P. Haccou, Y. Iwasa, *Theoretical Population Biology* **47**, 212 (1995).
- S18. K. R. Hopper, T. Rosenheim, T. Prout, J. Oppenheim, *OIKOS* **101**, 219 (2003).