Minireview

Floral induction and monocarpic *versus* polycarpic life histories

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Recent work in *Arabis alpina*, a perennial relative of *Arabidopsis*, has uncovered subtle differences in control of a gene that represses flowering which contributes to the polycarpic habit.

There are two extremes of life-history strategies in plants and animals - semelparity and iteroparity [1]. Semelparity is sometimes referred to as the 'big-bang reproductive strategy' [2], as semelparous species devote most of their energy and resources to maximizing the number of offspring in a single cycle of reproduction, and die soon after reproducing. Semelparity may be advantageous when the prospects for long-term survival are low. Iteroparous species, in contrast, reproduce multiple times, a strategy that may be advantageous when prospects for long-term survival are good.

In the plant kingdom, there are extreme examples of both strategies. At one end of the iteroparous spectrum are redwood trees, which can live for several thousand years with several thousand cycles of reproduction. In contrast, the popular semelparous research model *Arabidopsis thaliana* can complete its life cycle in less than two months, and once *Arabidopsis* produces a certain number of off-spring it rapidly senesces and dies, even under optimal growth conditions [3] (Figure 1).

Plants that live and reproduce for many years, such as redwoods, are often referred to as perennials. Plants such as *Arabidopsis* that typically survive only a single growing season are often referred to as annuals. However, the different life-history strategies of plants are better des-cribed by the terms monocarpic (semelparous; reproduces once and dies) and polycarpic (iteroparous; reproduces repeatedly), instead of annual and perennial, respectively. For example, perennial is hard to define, because there are plants that live for many years without flowering and then flower once and die. A striking example is the Haleakalā silversword, *Argyroxiphium sandwicense,* which may live for more than 50 years before flowering and dying (Figure 1).

The molecular basis for the death of monocarpic plants like *Arabidopsis* after reproduction is not well understood.

Plants develop from regions of stem cells called meristems. The shoot apical meristem (SAM) produces cells that differentiate into stems, leaves and flowers. In many monocarpic plants, including *Arabidopsis*, all active SAMs convert to flower production (that is, become inflorescence meristems). In *Arabidopsis*, when a certain number of seeds have been produced the inflorescence meristems stop growing, although they do not undergo terminal differentiation, and the whole plant senesces as the seeds mature [3]. Perhaps inflorescence meristem arrest after reproduction and the subsequent death is a specific genetic program in *Arabidopsis*, or perhaps the plants simply do not have the energy to sustain further growth from these inflorescence meristems - the plants 'burn out' in the effort to produce as many offspring as possible [3].

Thus, a key feature of polycarpy is to maintain a supply of meristems that are capable of vegetative growth; that is, SAMs that can produce shoots with leaves to sustain growth of the plant in future growth cycles. In a recent paper in *Nature* by Wang *et al.* [4], the polycarpic habit was studied in a relative of *Arabidopsis*, *Arabis alpina*, another member of the family Brassicaceae. *A. alpina* requires exposure to cold in order to flower (a phenomenon known as vernalization) [5]. However, as expected for a polycarpic plant, vernalization does not result in the flowering of all *A. alpina* SAMs. Those shoots of *A. alpina* that do flower cease growth and senesce during seed maturation similarly to shoots of *Arabidopsis*, but *A. alpina* maintains a supply of vegetative SAMs for another round of growth.

From polycarpy towards monocarpy

Wang *et al.* [4] identified an *A. alpina* mutant, *perpetual flowering 1* (*pep1*), that does not require vernalization for flowering. Furthermore, in non-vernalized *pep1* mutants, a greater fraction of SAMs become inflorescence meristems than in vernalized wild-type plants. Therefore, *PEP1* is required both to create a vernalization requirement and to ensure that a certain fraction of SAMs remain vegetative. Previous work in *Arabidopsis* has established that *FLOWERING LOCUS C* (*FLC*), a gene encoding a MADS-domain transcription factor, is a flowering repressor that prevents SAMs from flowering in the fall and creates a vernalization requirement [5]. Thus, Wang *et al.* [4]
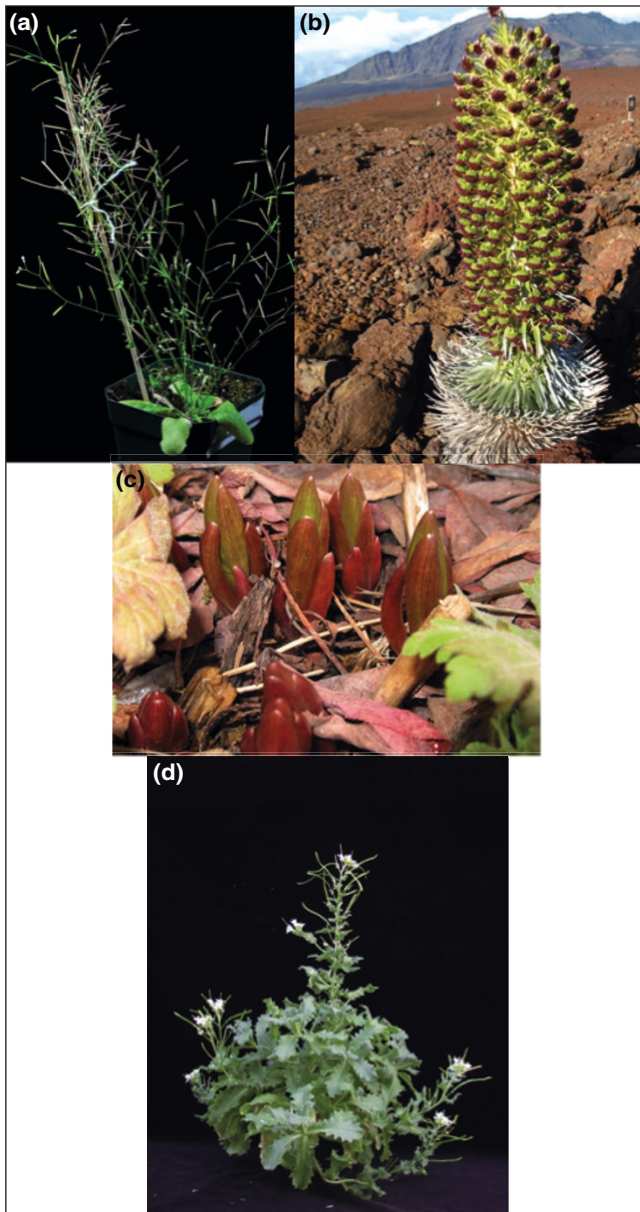
Figure 1

Examples of monocarpic and polycarpic plants. **(a)** A plant of *Arabidopsis thaliana* that has produced sufficient seed and is entering the phase of whole-plant senescence characteristic of many monocarpic plants. All of the shoots are floral, and this plant will soon die, despite being kept in optimal growth conditions. **(b)** Like *A. thaliana,* the monocarpic Haleakalā silversword dies after reproduction. But unlike *A. thaliana,* the silversword typically grows for several decades before flowering. **(c)** The above-ground parts of many polycarpic perennials that are adapted to temperate climates do senesce each year as winter approaches, and new growth emerges from below-ground parts of the plant in the following spring, as illustrated by this member of the lily family. **(d)** *Arabis alpina* is a polycarpic relative of *A. thaliana*. Whereas all shoots of *A. thaliana* undergo the floral transition, some *A. alpina* shoots remain vegetative to permit further growth and flowering in future years. *A. alpina* is a short-lived perennial that does not 'die back' in preparation for winter. Image of *A. alpina* courtesy of Maria Albani.

hypothesized that *PEP1* might be the *A. alpina* homolog of *FLC*, and demonstrated that this is indeed the case. What is interesting is that vernalization only transiently results in *PEP1* repression in *A. alpina*; this is in contrast to the situation in *Arabidopsis*, in which vernalization can result in a stable repression of *FLC* [5]. Only those *A. alpina* SAMs that actually initiate flowers during cold exposure produce flowering shoots when warm temperatures return. Even quite long periods of cold exposure are not sufficient to convert all SAMs to flowering, and the resumption of *FLC* expression in the non-flowering SAMs in warm temperatures ensures that these SAMs remain vegetative and that *A. alpina* is polycarpic.

In *Arabidopsis*, the stability of *FLC* repression is associated with repressive modifications to *FLC* chromatin, such as increased trimethylation of histone 3 at lysine 27 (H3K27triMe) and lysine 9 (H3K9triMe). These modifications are initiated during a vernalizing cold exposure, and the levels of these modifications increase after plants experience warm temperatures (see, for example, [6-10]). In *A. alpina*, H3K27triMe levels in *PEP1* chromatin increase during cold, but then decrease when plants are returned to warm temperatures [4]. It will be interesting to explore the molecular basis of *PEP1* expression and histone modification reversibility in *A. alpina*. For example, is reversibility inherent in the *PEP1* locus (for example, might *PEP1* lack certain *cis*-regulatory elements that are required for stable repression)? If this is the case, then *PEP1* might exhibit a similar transient repression even when introduced into *Arabidopsis*. There are precedents for such '*cis* effects'. Deletion of a region of the first intron of *Arabidopsis FLC* known as the 'vernalization response element (VRE)' creates a 'PEP1-like' allele for which cold repression is not maintained [8], and vernalization-mediated repression of cabbage *FLC* may not be maintained when the gene is introduced into *Arabidopsis* [11]. Alternatively, *PEP1* reversibility may be due to differences in the chromatin-modifying complexes in *A. alpina* compared with *Arabidopsis*; if this were the case, *Arabidopsis FLC* might be only transiently repressed in *A. alpina*. There are also precedents in *Arabidopsis* for this alternative. The reversible, cold-specific repression of *PEP1* in *A. alpina* is similar to that observed for *FLC* in certain *Arabidopsis* mutants such as *lhp1, vrn1* and *vrn2* [6-8,12-14].

Regardless of the mechanism of *PEP1* repression, it is clear that an important difference in the monocarpic versus polycarpic life histories of *Arabidopsis* versus *A. alpina* is, respectively, the permanent versus transient repression of *FLC/PEP1* by vernalization. This is not the complete story, however. As Wang *et al.* [4] discuss, *pep1* mutants do not phenocopy the monocarpic habit of *Arabidopsis*; some SAMs remain vegetative, and the *pep1* mutant continues to grow indefinitely after flowering. This indicates that additional genes are responsible for the monocarpic habit. Perhaps a

further round of mutagenesis in the *pep1* mutant back-ground might result in monocarpic lines, and thus reveal additional genes that are involved in life-history traits.

From monocarpy towards polycarpy

Looking at the question from another angle, Melzer *et al.* [15] reported in a paper in *Nature Genetics* last year that loss of two genes, *SUPPRESSOR OF CONSTANS 1* (*SOC1*) and *FRUITFULL* (*FUL*), causes *Arabidopsis* to assume a polycarpic habit. As discussed earlier, the monocarpic habit in *Arabidopsis* is caused, at least in part, by conversion of all active SAMs into inflorescence meristems, which eventually stop growing (although they do not terminally differentiate, as implied in [15]). In wild-type *Arabidopsis*, once a SAM becomes floral it never reverts to vegetative growth because a positive feedback loop of floral promoters locks in the flowering state [16-18]. Melzer *et al.* [15] show that *SOC1* and *FUL* are required for this lock-in. In *soc1/ful* double mutants, some inflorescence meristems revert to vegetative growth and other SAMs do not flower. The resulting double-mutant plants do not completely senesce after flowering because the vegetative SAMs keep growing.

Polycarpy requires not only the preservation of vegetative SAMs for future growth cycles, but the ability to produce new vascular tissue (secondary growth) to maintain the connection between shoots and the root system. In *soc1/ful* double mutants, there is enhanced secondary growth, and Melzer *et al.* suggest that 'loss of SOC1 and FUL function rather than the increased life span of the plants was responsible for the observed secondary growth' [15], but it is also possible that the enhanced secondary growth is an indirect effect of the presence of active vegetative SAMs in plants that are flowering. Vegetative SAMs on a flowering stem might, for example, alter phytohormone levels and fluxes such that secondary growth is favored.

Given that there are typically both monocarpic and poly-carpic species within the same plant family, and that their relationships indicate that transitions between monocarpy and polycarpy are common, perhaps the genetic differences between monocarpic and polycarpic species in a particular family are not extensive. These recent studies are an exciting start towards understanding the genetic basis of the difference between monocarpic and polycarpic habits in the Brassicaceae.

References
1. Charlesworth B: *Evolution in Age-Structured Populations*. Cambridge, UK: Cambridge University Press; 1980.
2. Humphries S, Stevens DJ: **Reproductive biology: Out with a bang.** *Nature* 2001, **410:**758-759.
3. Bleecker AB, Patterson SE: **Last exit: Senescence, abscission, and meristem arrest in *Arabidopsis*.** *Plant Cell* 1997, **9:**1169-1179.
4. Wang R, Farrona S, Vincent C, Joecker A, Schoof H, Turck F, Alonso-Blanco C, Coupland G, Albani MC: ***PEP1* regulates perennial flowering in *Arabis alpina*.** *Nature* 2009, **459:**423-427.
5. Amasino R: **Vernalization, competence, and the epigenetic memory of winter.** *Plant Cell* 2004, **16:**2553-2559.
6. Bastow R: **Vernalization requires epigenetic silencing of FLC by histone methylation.** *Nature* 2004, **427:**164-167.
7. Sung S, Amasino RM: **Vernalization in *Arabidopsis thaliana* is mediated by the PHD finger protein VIN3.** *Nature* 2004, **427:**159-164.
8. Sung S, He Y, Eshoo TW, Tamada Y, Johnson L, Nakahigashi K, Goto K, Jacobsen SE, Amasino R: **Epigenetic maintenance of the vernalized state in *Arabidopsis thaliana* requires *LIKE HETEROCHROMATIN PROTEIN 1*.** *Nat Genet* 2006, **38:**706-710.
9. Finnegan EJ, Dennis ES: **Vernalization-induced trimethylation of histone H3 lysine 27 at FLC is not maintained in mitotically quiescent cells.** *Curr Biol* 2007, **17:**1978-1983.
10. Sung SY, Schmitz RJ, Amasino RM: **A PHD finger protein involved in both the vernalization and photoperiod pathways in *Arabidopsis*.** *Genes Dev* 2006, **20:**3244-3248.
11. Lin SI, Wang JG, Poon SY, Su CI, Wang SS, Chiou TJ: **Differential regulation of *FLOWERING LOCUS C* expression by vernalization in cabbage and *Arabidopsis*.** *Plant Physiol* 2005, **137:**1037-1048.
12. Mylne JS, Barrett L, Tessadori F, Mesnage S, Johnson L, Bernatavichute YV, Jacobsen SE, Fransz P, Dean C: ***LHP1*, the *Arabidopsis* homologue of *HETEROCHROMATIN PROTEIN1*, is required for epigenetic silencing of *FLC*.** *Proc Natl Acad Sci USA* 2006, **103:**5012-5017.
13. Gendall AR, Levy YY, Wilson A, Dean C: **The *VERNALIZATION 2* gene mediates the epigenetic regulation of vernalization in *Arabidopsis*.** *Cell* 2001, **107:**525-535.
14. Levy YY, Mesnage S, Mylne JS, Gendall AR, Dean C: **Multiple roles of *Arabidopsis VRN1* in vernalization and flowering time control.** *Science* 2002, **297:**243-246.
15. Melzer S, Lens F, Gennen J, Vanneste S, Rohde A, Beeckman T: **Flowering-time genes modulate meristem determinacy and growth form in *Arabidopsis thaliana*.** *Nat Genet* 2008, **40:**1489-1492.
16. Tooke F, Ordlidge M, Chiurugwi T, Battey N: **Mechanisms and function of flower and inflorescence reversion.** *J Exp Bot* 2005, **56:**2587-2599.
17. Hempel FD, Welch DR, Feldman LJ: **Floral induction and determination: where is flowering controlled?** *Trends Plant Sci* 2000, **5:**17-21.
18. Ferrandiz C, Gu Q, Martienssen R, Yanofsky M: **Redundant regulation of meristem identity and plant architecture by *FRUITFULL*, *APETALA1* and *CAULIFLOWER*.** *Development* 127:**725-734.