Minireview

**Floral induction and monocarpic versus polycarpic life histories**

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Abstract

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 offspring 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 described 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, *Argyroxyphium 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 senescence 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 polycarpic 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 polycarpic towards monocarpic

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]...
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 background 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 polycarpic species within the same plant family, and that their relationships indicate that transitions between monocarpic and polycarpic 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.

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