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Bloom & Bust: Understanding the Nature and Regulation of the End-of-Flowering

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ABSTRACT

The reproduction of flowering plants is an incredibly important process, both ecologically and economically. A huge body of work has examined the mechanisms by which flowering plants correctly time their entry into the reproductive phase (the 'floral transition'). However, the corresponding mechanisms by which plants exit the reproductive phase remain relatively neglected. In this review, we identify four developmental processes that contribute to the end-of-flowering; floral arrest, inflorescence meristem arrest, inflorescence activation and 'vegetative transition'. We highlight that, due to the highly divergent nature of reproductive systems among flowering plants, these processes are differently important for end-of-flowering in different species. For each of these processes, we examine recent advances in understanding the regulatory mechanisms that govern the process, and how these mechanisms determine the timing of end-of-flowering.

KEYWORDS

Flowering, inflorescence meristems, shoot branching, floral transition, reproductive phase

MAIN TEXT

Introduction: a poorly understood but important process

In all plants, entry into the reproductive phase is carefully timed to maximize reproductive success. In flowering plants, it is essential that reproduction starts in the right season, allowing plants to utilize intermittent resources (e.g. water) and pollinator availability. After this 'floral transition', one or more vegetative shoot meristems becomes converted to an inflorescence meristem (IM), which initiates multiple floral meristems (FMs). The determinate FMs produce flowers with a (usually) fixed number of organs, which give rise to fruit and seed. Floral transition has been intensively studied as a classic model system for understanding the integration of environmental cues with growth and development [1,2].

In contrast, the conclusion of flowering plant reproduction ('end-of-flowering') has been largely neglected, and we know correspondingly little about its regulation. This is a curious oversight, since the duration of flowering directly determines the reproductive potential of a plant, and therefore the yield of many crops (including all cereals). Twenty-five years on from a seminal study of end-of-flowering in Arabidopsis [3], there has been a recent upturn in interest in this area. Although progress has been somewhat fragmentary, there has been a spate of exciting new data and models for end-of-flowering, often posing as many new questions as they answer [4,5,6]. In this review, we examine recent progress, and try to reconcile these data into a coherent framework.

What is the end of flowering?

To understand how the end-of-flowering is regulated, we need to define exactly when and where it occurs, but unlike the floral transition, this is not straightforward. As a phenomenon, the most obvious definition for end-of-flowering would be the time-point when no further flowers open (**'floral arrest'**). However, is flower-opening really a relevant regulatory checkpoint? Or do events earlier in the reproductive phase inevitably determine when end-of-flowering occurs? There is likely no single answer to this question, partly reflecting the stunning diversity of inflorescence architectures among flowering plants.

Inflorescence classification is complex, but from the perspective of end-of-flowering, the most important division is between inflorescences with invariant or variable developmental patterns. Variable inflorescences can produce a flexible number of flowers, depending on circumstances. Indeterminate inflorescences – those in which the IM does not form a terminal flower – are more likely to be variable, but this is not always the case. In variable inflorescences, flower opening/maturation might indeed be a relevant regulatory checkpoint, although ongoing IM activity

is likely to be important too – since once the IM ceases to produce FMs ('**IM arrest**') floral arrest becomes an inevitable consequence.

In invariant inflorescences, a very similar number of flowers are produced by each IM (though this is rarely completely lacking in variation). Invariant inflorescences will often also be determinate, with the IM converted to a terminal flower. In invariant inflorescences (and particularly determinate ones) the timing of floral arrest and IM arrest is also invariant, so these are unlikely to be relevant control points for flexible regulation of end-of-flowering in these species. In effect, plants with invariant inflorescences will only flower for as long as they continue to initiate new inflorescences, and this is likely to be the most important control point. It is worth noting that even variable inflorescences do not have unlimited developmental potential. Recent work in Arabidopsis has shown that each class of inflorescence is active for a characteristic length of time before arresting [6]; their development is thus time-limited, rather than number-limited. It is also notable that the timing of end-of-flowering in Arabidopsis also directly mirrors the timing of the end of inflorescence initiation [6]. Thus, even in plants with variable inflorescences, control of inflorescence initiation is likely to be an important control point for end-of-flowering. It is also worth noting that many species do not flower in a single coordinated burst, but may continue to initiate new inflorescences over a prolonged time, such that earlier inflorescences have completely arrested while new ones are still being initiated. In these species (of which domesticated tomato is a prime example), inflorescence initiation is certainly the major control point for end-of-flowering.

In annuals and monocarpic perennials, flowering marks the effective end of the life-cycle, and the plant commits all remaining resources to the production of inflorescences, flowers, fruit and seed. However, in polycarpic perennials, the commitment to reproduction is less dramatic, and these plants will produce new vegetative growth after flowering. The most notable example of this is in flowering trees, which alternate reproductive and vegetative life phases for many years, often separated by dormancy episodes [2]. These woody perennials presumably have a more complex regulation of flowering time, often undergoing floral transition in autumn ahead of a spring bloom [7]. By the time these trees blossom, they are usually back in a vegetative phase of growth, and thus the relevant regulatory end-of-flowering actually likely occurred in autumn, before a single flower opened. The complexity found among polycarpic species highlights that merely ending the initiation of inflorescences is not sufficient to explain end-of-flowering in many perennials; rather, the plant must actively return to the vegetative phase ('**vegetative transition**'). Thus, there seem to be at least four biological processes that could act as regulatory control points to bring about end-of-flowering (Figure 1). It is likely that all four are utilised among different species, to different degrees, to provide an appropriately coordinated end-of-flowering. It is also likely that each of these processes is highly sensitive to environmental conditions, allowing end-of-flowering to be flexibly altered in response to changing conditions across the reproductive phase. Inflorescence

initiation is certainly tightly regulated by environmental conditions [7], and it is likely that both IM and floral arrest can be flexibly altered to the prevailing conditions. For instance, there is significant variation in both the lifetime of inflorescences, and the number of flowers produced per inflorescence, between different experiments in Arabidopsis [6].

The vegetative transition

The current consensus model for initiation of flowering is that a variety of environmental and developmental cues regulate the production of 'florigen', a mobile signal that triggers the floral transition in shoot meristems [8,9,10]. Over 200 genes regulating floral initiation have been identified [11] and much of the underlying genetic network has been characterized [12,13,14,15], but the relative contribution of these genes can differ between species. Of particular note in Brassicaceae is FLOWERING LOCUS C (FLC), a MADS-box transcription factor that represses flowering until vernalization has occurred. Sensitivity of FLC to other environmental cues such as ambient temperature and light intensity make it a key regulator of floral transition [16,17]. The mobile protein FLOWERING LOCUS T (FT) (or equivalent orthologues) is another central regulator of flowering [9]. Favourable environmental cues such as long days or high temperature up-regulate FT expression in leaves [18], and the resulting mobile pool of FT protein in turn moves to shoot meristems and triggers their conversion to IMs [19]. Whilst FT is often viewed as florigen, the main signal mediating floral transition [9], a growing body of research highlights the importance of FT-independent mechanisms under certain conditions or in some species [20]. Again, different molecular networks might be more or less important for inducing flowering depending on the species or the environmental conditions.

It is logical to assume that vegetative transition involves withdrawal of the floral stimulus, perhaps including a decline in FT levels or an increase in FLC, at least in Brassicaceae. Evidence in terms of FT is rather mixed; FT levels are much higher after floral transition, especially in reproductive tissues [21,22], and FT overexpression leads to uniformly short vegetative and reproductive phases [9]. Conversely, it has been found that FT and FLC expression follow an inverse trend at the end of the reproductive period, with FLC peaking and FT levels declining [5]. Consistent with this, there is clear evidence for the involvement of FLC in the vegetative transition. In the polycarpic perennial *Arabis alpina* (a close relative of Arabidopsis), vegetative growth is resumed after each reproductive phase, while previously formed inflorescences die back [23]. In the subsequent reproductive phase, a new floral stimulus triggers floral transition in new meristems, which grow out to form inflorescences [24,25]. However, in mutants lacking *PERPETUAL FLOWERING1*, the FLC orthologue, the first flowering episode never ends, with new shoot meristems continually converted to IMs [24]. However, individual inflorescences continue to arrest normally, showing this effect is mediated at the level of inflorescence initiation. It is also worth noting that two of the top upregulated genes in arrested Arabidopsis inflorescences are FLC and

its close homologue FLM [26], consistent with the idea that even monocarpic plants may undergo a cryptic vegetative transition at end-of-flowering (Figure 1).

Regulation of inflorescence number

As specialised shoot branches, activation of inflorescences is regulated comparably to vegetative branch activation. Indeed, since they are the only true branches Arabidopsis produces, a significant part of our knowledge of branching is derived from inflorescences. The current 'hybrid' model for shoot branching suggests that resource availability, communicated through hormonal signals including sugars, dictates the number of branches produced, with each shoot apex making a local 'priming' decision, and competition between apices determining which primed apices ultimately grow [7]. Such a model neatly explains the regulation of inflorescence number in species like Arabidopsis, where inflorescences are produced as a coherent, terminal event: the plant initiates as many inflorescences as it can 'afford'. But what about species that produces inflorescences alongside continued pseudo-vegetative growth (pea, tomato), or polycarps that do not completely commit to reproduction? Presumably the total number of shoot apices is still dictated by resource availability, but how does the plant decide how many inflorescences to initiate? The answers to this are currently unclear, but careful control of developmental phasing is likely to be key.

Arabis alpina again offers insights into this process. Shoot meristems initiated at different phases of the *A. alpina* life-cycle have strongly divergent fates; new (but inactive) meristems formed before winter will remain inactive; those initiated during winter will form vegetative branches and those formed after vernalization will form inflorescences [23]. This suggests the identity of inflorescences can be 'locked-in', and that FT produced in inductive conditions can only act in 'receptive' meristems. One possibility is the members of the BRANCHED1 (BRC1) family of TCP transcription factors, well-established regulators of shoot branching, may play a key role in determining this 'receptivity'. In Arabidopsis, BRC1 activity prevents FT from inducing inflorescence fate in dormant meristems – only when BRC1 levels fall during activation of the meristem is IM fate acquired [27]. A comparable situation has been demonstrated in wheat, in which TEOSINTE BRANCHED1 (TB1) activity delays the initiation of spikelets (infloresences) within the developing ear, by opposing FT1A activity [28]. A plausible model would be that very high levels of BRC1 (and homologs) inhibit meristem activity completely, while intermediate levels allow vegetative (and pseudo-vegetative) meristem activity, and very low levels allow specification of inflorescences (Figure 2). Thus, precise regulation of BRC1 activity in meristems might determine the level of inflorescence initiation.

Regulation of IM arrest

As discussed above, once an inflorescence has been specified and activated, it may initiate an invariant or variable number of FMs. In this section, we specifically focus on the regulation of IM

activity in variable IMs. One of the clear advantages of variable IMs is their ability to compensate for changes in status during flowering, and to produce more or less flowers depending on environmental conditions or past developmental events. Recent work in Arabidopsis and other Brassicaceae strongly suggests that inflorescences can exert feedback on one another, since approximately 50% of all flowers are found on secondary inflorescences irrespective of the number of inflorescences [29]. This suggests that individual IM activity is homeostatically regulated by global IM activity.

Arabidopsis inflorescences typically arrest at a consistent time after initiation, irrespective of the number of flowers initiated [6]. Recent work suggests that the timing of IM arrest in Arabidopsis is influenced by the developmental age, which is perceived through the ratio of certain *miRNAs* [30]. As plant age increases and floral transition occurs, *miR156* levels decrease, triggering a concomitant rise in expression of *SQUAMOSA-PROMOTER BINDING PROTEIN-LIKE* (*SPL*) family proteins normally targeted by *mi156* [13]. The SPL genes promote expression of *miR172* and the FM identity gene *FRUITFULL* (*FUL*), which both act to repress the floral identity gene *APETALA2* (*AP2*) and its homologues [30]. Since AP2 normally promotes expression of the meristem maintenance gene *WUSCHEL* (*WUS*), this age-related inhibition of *AP2* leads to IM arrest [30,31] (Figure 3). Conversely, mutations in *FUL*, or in the *miR172* binding site in *AP2*, result in an extension of IM activity [30], delaying end-of-flowering (Figure 3). It is currently unclear how generalizable this pathway is, but it is certainly worth noting that the causative lesion in the *perpetual flowering2* mutant of *Arabis alpina* is in an *AP2* homologue [24].

Even though their arrest is normally precisely timed, it is clear that IM arrest is a flexible state, which is sensitive to fruit production; if global fruit levels are too low, IM activity may be extended in response [3,6]. Indeed, IM arrest is a reversible state, and if fruit are removed after arrest, the IM can reinitiate activity and produce more fruit [3]. Arabidopsis IM arrest appears to be a classically dormant state, and consistent with this, the transcriptome of arrested IMs closely resembles that of dormant axillary meristems, including expression of bud dormancy markers AtDRM1 and AtDRM2 [26]. This strongly contrasts with determinate inflorescences, where the IM is irretrievably differentiated as a flower.

Regulation of flower maturation

While there has been much research into the initiation of flowers, we know less about regulation of their maturation, especially with regard to the numbers that are ultimately opened. Arabidopsis inflorescences typically arrest with a cluster of unopened buds (Figure 4A), so clearly the initiation of FMs does not result in their inevitable maturation into flowers. Thus, while IM activity regulates

the number of flowers initiated, IM arrest is not sufficient to explain the timing of floral arrest in Arabidopsis. We thus believe that there must be an additional mechanism controlling the maturation of flowers. Auxin export from fruit located proximal to the IM has been shown to be a requirement for floral arrest in Arabidopsis [6], but the site of auxin action is unclear. A distinct possibility is that, rather than acting via the IM, the export of auxin from older fruits directly inhibits the maturation of younger FMs, leading to undeveloped flowers, and the characteristic bud cluster seen in Arabidopsis at the end-of-flowering (Figure 4B).

Regulation of floral maturation as a developmental control point does not seem to be exclusive to Brassicaceae – indeed, similar phenomena are seen in multiple species, with floret degradation in wheat being one example. Wheat initiates up to twelve florets per spikelet (inflorescence), but typically only four will fully develop and set seeds; prior to physiological maturity, the other florets are aborted, determining the number of remaining fertile florets [32]. Different wheat cultivars can show considerable variation in the number of seeds set per spikelet [33], so floret abortion seems to be a flexible process, which might be analogous or homologous to floral arrest in Arabidopsis.

Conclusions

This brief journey through the end-of-flowering highlights the diverse processes that contribute to bringing the reproductive phase to an end, and particularly the flexible manner in which these processes can be regulated to adjust the duration of flowering in response to environmental conditions. Deeper understanding of the regulatory mechanisms that underpin the end-of-flowering will open up the prospect of precise manipulation of end-of-flowering to “push” yields, or conversely to reduce flowering to promote early and more synchronous harvest-readiness in crop species. Interest in the end of reproduction has lagged far behind interest in its start, but just as floral arrest must necessarily happen after floral transition, perhaps it is apt the end-of-flowering is only now reaching the forefront of plant developmental biology research.

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FIGURES

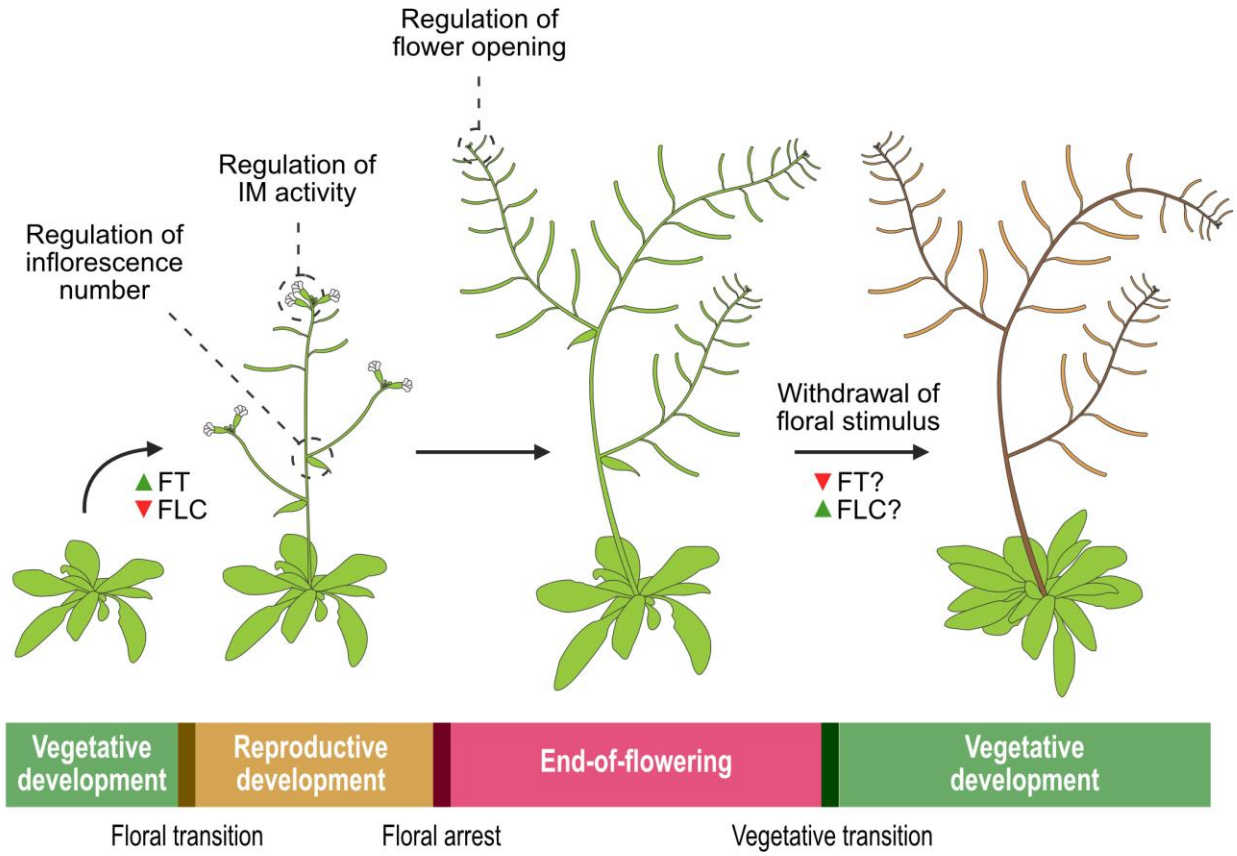

Figure 1. The life cycle of plants and the end of flowering.

Cartoon of life-cycle transitions in a generic Brassica. Upon perceiving inductive conditions, plants undergo the floral transition and switch from vegetative development to reproductive development. Some shoot meristems are converted to inflorescence meristems (IMs) and activate to generate flower-bearing inflorescences. A number of different molecular signals have been proposed as the 'floral stimulus' that drives this transition, such as an increase in the promotive FLOWERING LOCUS T (FT) and a reduction in the repressive FLOWERING LOCUS C (FLC) (or homologues), among others. Plants eventually exit reproduction in an end-of-flowering developmental phase. We have identified at least four control points that contribute to end-of-flowering (indicated in the diagram). Polycarpic perennials may reinitiate vegetative development through a 'vegetative transition'. We propose that this requires withdrawal of the floral stimulus, and that this vegetative transition may also occur cryptically in annual/monocarpic species.

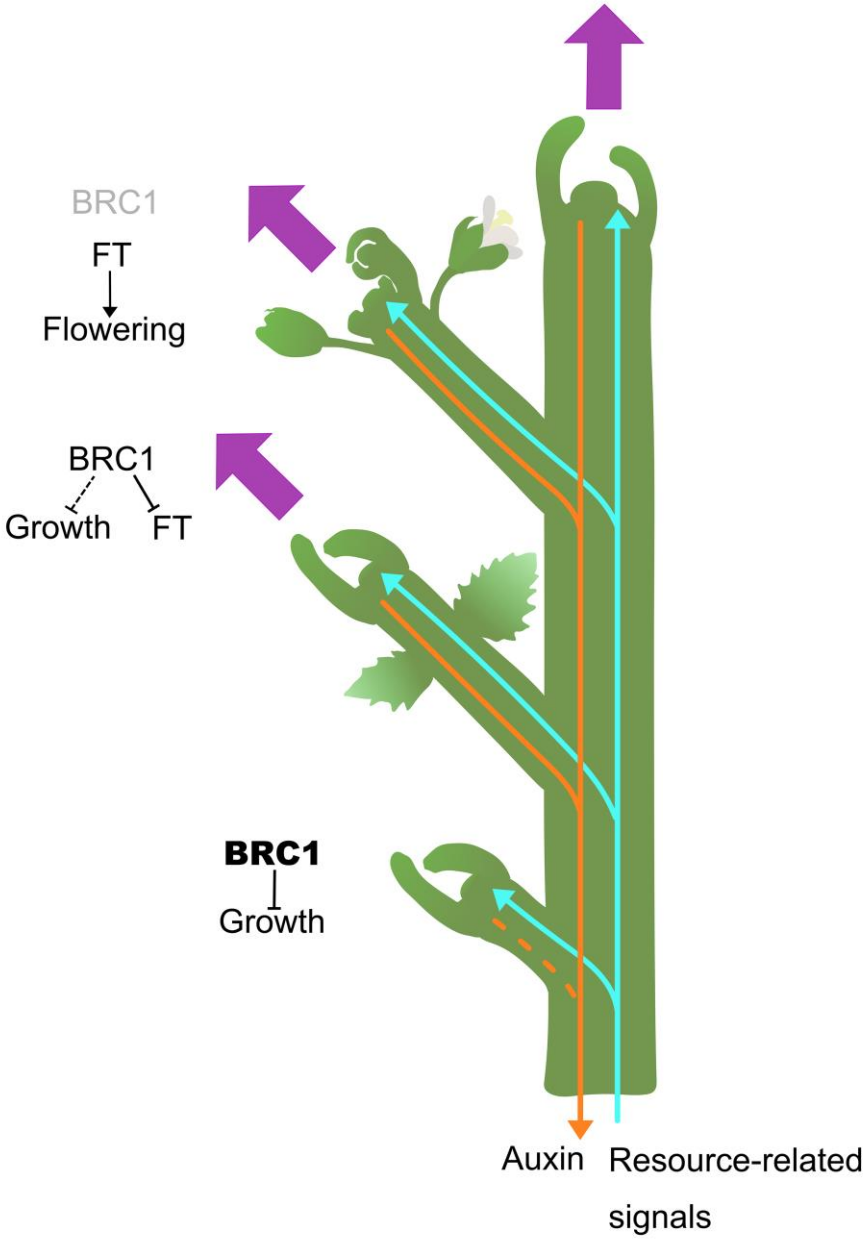

Figure 2. Regulation of inflorescence activation.

Model for the regulation of inflorescence activation in a generic perennial. Two hormonal systems determine which shoot apices are active. Long-distance root-to-shoot resource-related signals including cytokinins and strigolactones determine the number of active apices, while canalization-dependent competition for auxin export between shoot apices determines which apices remain active. The level of *BRANCHED1* (*BRC1*) expression in apices contributes to meristem fate in a rheostatic manner; very high *BRC1* helps to prevent activation (bottom meristem) and generates a dormant bud bank; moderate *BRC1* does not prevent or reverse activation but sequesters FT and maintains vegetative meristem identity (middle meristem), while low *BRC1* allows FT to promote the conversion to inflorescence meristem fate (top meristem).

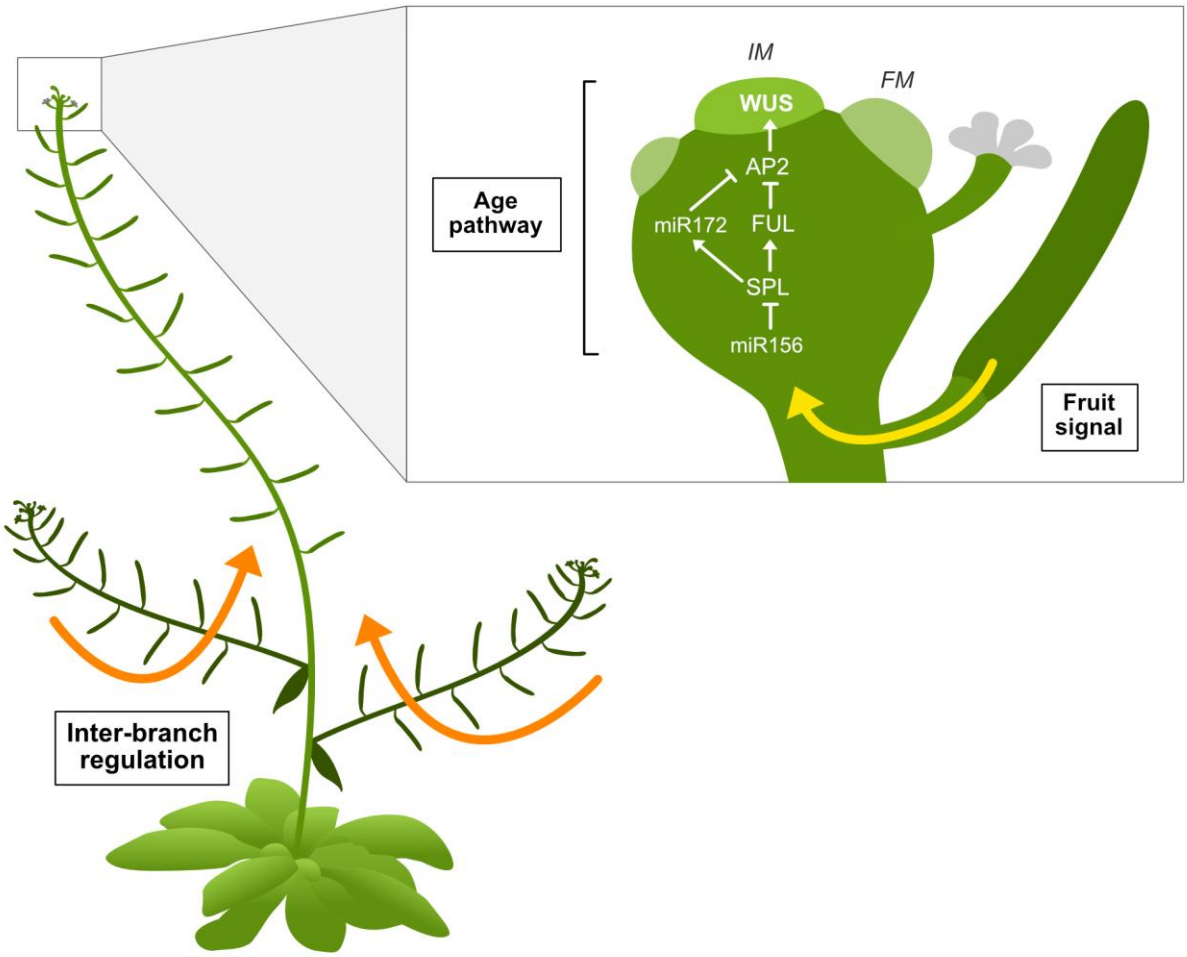

Figure 3. Pathways controlling IM activity.

Inflorescence meristem (IM) activity is ultimately controlled by core meristem maintenance genes including WUSCHEL (WUS). WUS levels in IMs are maintained by APETALA2 (AP2) activity, but AP2 is gradually repressed by an age-dependent pathway that involves FRUITFUL (FUL) and *miR172*. Fertile fruits and/or seeds also exert a regulatory role on IM activity, although the molecular mechanisms are not completely understood. Fruit on other branches within the same plant also act to inhibit IM activity through uncharacterised long-distance signalling.

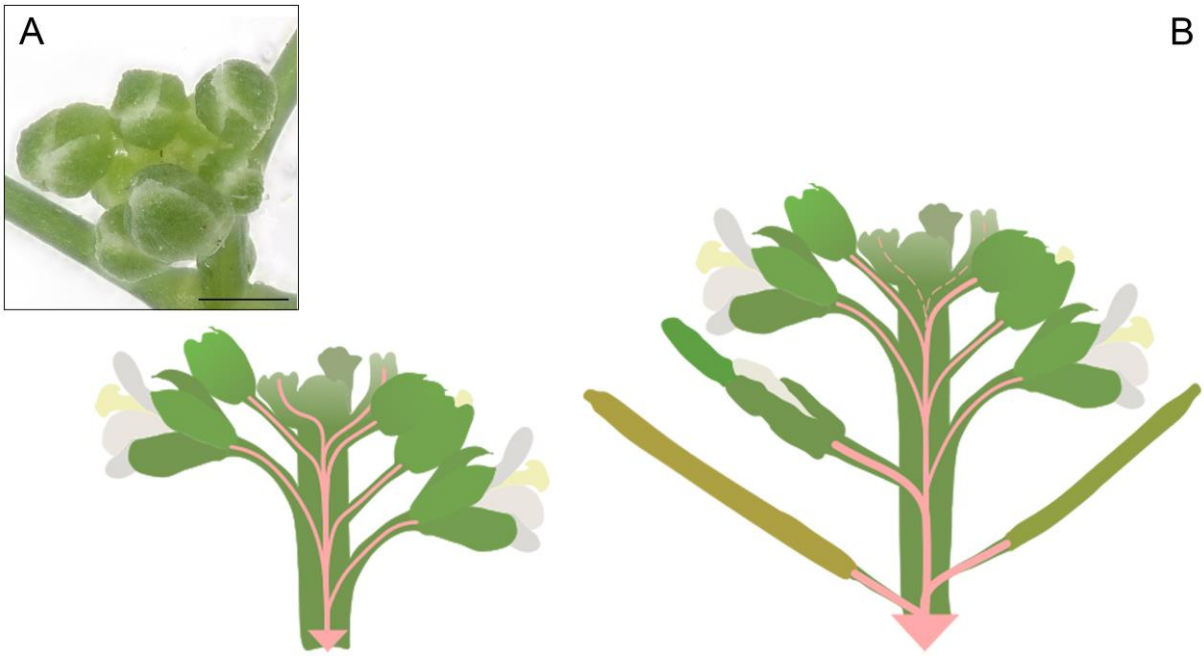

Figure 4. Regulation of floral maturation

(A) Image showing the bud cluster of an arrested wild type (Col-0) Arabidopsis inflorescence. Unopened buds and floral primordia surround the meristem. Bar = 500µm. (B) A proposed model for inhibition of floral maturation. Initially, maturing floral primordia can freely canalize to the polar auxin transport stream (PATS, pink). Production of sufficient mature fruits saturates the PATS, preventing canalization from the younger floral primordia. This inhibits floral maturation, resulting in a cluster of buds at the arrested inflorescence apex.

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