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MINI-REVIEW

Control of floral stem cell activity in Arabidopsis

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ABSTRACT

In Arabidopsis, the floral meristem is essential for the production of floral organs. The floral meristem is initially maintained to contribute cells for floral organ formation. However, this stem cell activity needs be completely terminated at a certain floral developmental stage to ensure the proper development of floral reproductive organs. Here, we have reviewed recent findings on the complex regulation of floral meristem activities, which involve signaling cascades, transcriptional regulation, epigenetic mechanisms and hormonal control for floral meristem determinacy in *Arabidopsis*.

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Introduction

In Arabidopsis, a fixed number of floral organs are generated by floral meristems. Floral meristems are established by the homeobox gene *WUSCHEL* (*WUS*) expressed in the cells of the organizing center (OC). The WUS protein specifies stem cell identity in a non-cell autonomous manner by migrating from the OC into the central zone (CZ).[1] In the CZ, WUS binds directly to the *CLAVATA3* (*CLV3*) locus and activates *CLV3*, which is uniquely expressed in aerial stem cells.[1] The CLV3 peptide diffuses to the OC region, can be recognized by the CLAVATA (CLV) receptor system, including CLV1, CLV2, CORYNE (CRN), BARELY ANY MERISTEMS (BAMs) and CLAVATA3 INSENSITIVE RECEPTOR KINASE (CIKs), and restricts *WUS* expression.[2–6] In early stages of flower development, the *CLV-WUS* signaling pathway maintains stem cell homeostasis, thus giving rise to proper flower formation.[7] However, stem cell activity is terminated at floral stage 6[8] to ensure initiation and proper development of carpels.

In this review, we mainly summarize the mechanisms of floral stem cell termination. These mechanisms involve transcriptional and epigenetic regulation of multiple factors. Phytohormones also play an indispensable role in the intricate and precise control of floral meristem determinacy.

AGAMOUS (AG) and SUPERMAN (SUP) repress WUS from early floral stages

AG directly represses WUS *from floral stage 3*

The floral homeotic gene *AG* alone specifies carpel identity in Arabidopsis.[9] In addition, AG is a critical transcription factor involved in the direct repression of *WUS* in the floral meristem. AG activity is induced at the center of stage 3 flower buds by WUS and LEAFY (LFY).[10,11] In turn, AG represses *WUS* expression by directly binding to the locus from floral stage 3 onward.[12] Polycomb group protein (PcG) complex, which can introduce the repressive mark, histone H3 Lys 27 trimethylation (H3K27me3) to silence genes,[13] is also involved in *WUS* regulation by AG. Polycomb repressive complex 1 (PRC1) factor TERMINAL FLOWER 2 (TFL2) is recruited by AG to the *WUS* locus for direct repression. The PRC2 factor, CURLY LEAF (CLF), is also required for the regulation of AG-mediated FM determinacy. A recent study has shown that there is a chromatin loop on *WUS* that is formed by the interaction between AG and TFL2 at two specific regions on the *WUS* locus.[14] The recruitment of RNA polymerase II is blocked by this chromatin loop on *WUS*, and *WUS* expression is thereby repressed by the loop. However, *WUS* is only mildly repressed by AG from floral stage 3 onward. Although the null *ag-1* mutant shows loss of floral meristem determinacy with the homeotic transformation of flower phenotype to sepal-petal-petal reiteration,[15] transgenic lines of *35S:AG*, in which *AG* is overexpressed, still produce flowers with normal carpels.[16] This finding suggests that floral meristem termination requires factors other than the AG protein alone.

SUPERMAN represses WUS from early stages of flower development

SUPERMAN (*SUP*) encodes a C2H2-type zinc finger transcription factor, and the loss of *SUP* function leads to floral meristem indeterminacy and over-production of stamens at the expense of proper carpel formation.[17] A recent paper found that extra stamens in *sup* mutants originate from cells in both third and fourth whorls and undergo fate changes from carpels to stamens.[18] In loss-of-function *sup*, *ag* and *clv3* triple mutant flowers, the floral meristem becomes much larger than the meristem in *ag clv3* double mutants, suggesting SUP may regulate floral stem cell activity in parallel with AG and CLV3.[19] At floral stage 3, *SUP* expression is observed on both sides of the

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boundary between whorl 3 stamens and whorl 4 carpels.[17,18] SUP forms a repressor complex with PcG factors CURLY LEAF (CLF) and TFL2 and fine-tunes local auxin signaling by negatively regulating the expression of the auxin synthesis genes *YUCCA1/4* (*YUC1/4*).[20] In *sup* mutant flowers, auxin production is increased in the whorl 3/4 boundary region, while auxin accumulation is reduced in the center of the flowers.[20] Repression of auxin biosynthesis by SUP is pivotal for floral meristem determinacy from floral stage 3 onward; at this stage the stem cell marker CLV3-GFP begins to indicate there is an increase in stem cell numbers in *sup* mutant flowers compared to wild type flowers.[20,21]

AG induces KNUCKLES (KNU) and CRABS CLAW (CRC) to repress WUS at floral stage 6

AG induces KNU to terminate floral stem cells

WUS expression is directly repressed by AG from floral stage 3 onward.[12] However, this inhibition is moderate and insufficient to terminate stem cell activity. At floral stage 6, AG directly induces *KNUCKLES* (*KNU*), which encodes a C2H2-type zinc finger protein. Both weak mutant *knu-1* and null mutant *knu-2* display indeterminate floral phenotypes with ectopic reiterative stamens and carpels formed within gynecium due to prolonged *WUS* activity.[22,23] In contrast, transgenic plants with an over-expression of *KNU* produce flowers resembling the loss-of-function mutant *wus-1* phenotype; this observation suggests that KNU plays a decisive role for *WUS* termination at stage 6.[8,23,24]

From floral stage 3, AG directly binds to the *KNU* promoter.[23] However, *KNU* expression is not activated immediately after AG binding. There is a characteristic 153 bp Polycomb Responsive Element (PRE) on the *KNU* promoter; PRE formation coincides with AG binding to CArG boxes.[24] The PRC2 factors, including EMBRYONIC FLOWER 2 (EMF2) and FERTILIZATION INDEPENDENT ENDOSPERM (FIE), associated with *KNU* PRE, are displaced by AG from floral stage 3; this action leads to the failure of the maintenance of repressive marker H3K27me3 on *KNU* chromatin.[24] Through 1 ~ 2 rounds of cell division, which takes approximately 2 days,[24] H3K27me3 repression on *KNU* is diluted. Therefore, *KNU* expression is activated at floral stage 6. The timing of *KNU* induction is pivotal for normal floral development. Delayed or early *KNU* expression leads to indeterminate floral organs or to flowers without carpels, respectively.[7]

The termination of floral stem cells is characterized by the silencing of *WUS* activity at floral stage 6, which is a multistep process mediated by the function of *KNU*.[8] As a repressor, KNU directly binds to the *WUS* proximal promoter region and co-localizes to the SPLAYED (SYD) binding site. SYD is a SWI/SNF chromatin remodeling factor and functions as a key activator of *WUS*.[25] KNU binding causes SYD eviction, reduces DNA accessibility of the *WUS* locus, and decreases the levels of active H3K4me3 histone markers and H3 acetylation on *WUS* chromatin. These events are associated with the repression of *WUS* mRNA within 4 hours of KNU activation.[8]

The deacetylation of histones is reported to be required for *WUS* repression.[8,26] As an initial step, the adaptor protein MINI ZINC FINGER2 (MIF2) binds to the first *WUS* intron and recruits KNU, TOPLESS (TPL) and HISTONE DEACETYLASE19 (HDA19) to form a transcriptional repressor complex that represses *WUS*.[26] This activity possibly occurs simultaneous with the eviction of SYD.

Next, KNU interacts with FIE and recruits PRC2 to *WUS* in a KNU-dependent manner; this recruitment leads to increased H3K27me3 levels on *WUS* chromatin approximately 8 hours after *KNU* activity is induced.[8] The deposition of H3K27me3 is only detected several hours later than *WUS* transcriptional repression and reduction of active markers on *WUS* chromatin. The deposition of H3K27me3 may be a prerequisite step for PcG-mediated silencing of *WUS*. In the transgenic co-suppression line *35S:GFP-FIE*, which has mostly silenced FIE activity,[27] ectopic carpelloid tissue is found inside the gynecium; this is the result of prolonged *WUS* activity.[8] On the other hand, over-expression of *KNU* in *tfl2* and *clf* mutants still gives rise to normal carpels, unlike *KNU* over-expression in wild types in which carpel formation is fully abolished. All these findings suggest that PcG activity is required to silence *WUS*. Hence, KNU integrates transcriptional repression and epigenetic silencing of *WUS* through multiple steps for floral stem cell termination.

AG induces CRC for floral meristem termination through fine-tuning auxin homeostasis

CRC encodes a YABBY family transcription factor, which is directly activated by AG at floral stage 5 ~ 6.[28–30] Single mutants of *crc* have no obvious floral meristem defects; however, stronger floral meristem indeterminacy is observed in *crc-1 knu-1* double mutants compared to *knu-1* mutants, indicating that *CRC* is also involved in the regulation of floral meristem determinacy.[31,32] At floral stage 6, CRC and AG synergistically activate *YUC4* leading to auxin accumulation in the floral meristem.[33] CRC can also directly repress *TORNADO2* (*TRN2*), which encodes a transmembrane protein involved in the negative regulation of auxin signaling.[34–37] To establish the auxin maxima needed to direct floral organ differentiation in the floral meristem, CRC finely modulates auxin homeostasis by both promoting *YUC4* and repressing *TRN2*.[30]

AG activity is modulated by SEPALLATA3 (SEP3) and APETALA2 (AP2)

AG plays dual roles in floral organ identity control, and floral meristem regulation, but it is still unknown whether AG participates in different complexes due to its distinct functions. It has recently been reported that the MADS-domain protein complex formed by AG and SEPALLATA3 (SPE3) tetramers is required to activate AG's direct downstream genes *KNU* and *CRC* for floral meristem determinacy.[38]

Interestingly, the role of AG in floral stem cell regulation is antagonized by the floral homeotic protein APETALA2 (AP2), which represses *KNU* expression at floral stage 6.[39] Therefore, AP2 could serve as a brake in the feed-forward regulatory loop consisting of *AG*, *KNU* and *WUS*.

Other factors involved in floral meristem regulation

In flower development, auxin and cytokinin interact to co-regulate floral meristem determinacy. Promoted by auxin, *AUXIN RESPONSE FACTOR3* (*ARF3*) functions in two ways to inhibit floral stem cell activity. ARF3 can directly bind to the *WUS* promoter to repress its expression.[40] ARF3 can also repress cytokinin activity by repressing the *ISOPENTENYLTRANSFERASE* (*IPT*) and *LONELY GUY* (*LOG*) family genes that encode enzymes for cytokinin biosynthesis.[41] Furthermore, ARF3 directly represses the expression of *ARABIDOPSIS HISTIDINE KINASE4* (*AHK4*), which encodes a cytokinin receptor; this repression leads to a decrease in cytokinin activity and *WUS* repression.[41] In flower development, both *AG* and *AP2* can dynamically regulate *ARF3* expression,[40,42] thereby linking *ARF3* transcription factor activities with phytohormones for the regulation of floral stem cells.

At floral stage 3, *AG* expression is induced at the center of flower buds by LFY and WUS.[10,11] ULTRAPETALA1 (ULT1), a trxG protein, regulates floral stem cell activity by inducing AG expression in a LFY-independent manner.[43] PERIANTHIA (PAN) is a bZIP transcription factor, and the expression region of *PAN* overlaps with *AG*; PAN can also directly activate *AG* expression.[44,45] A reduced *AG* expression level and an increased number of floral organs are observed in *pan* mutant flowers.[45] In addition, other genes, including *SQUINT* (*SQN*) and *REBELOTE* (*RBL*), function redundantly upstream of *AG* and maintain *AG* expression from floral stage 3 onward.[32]

In shoot apical meristems and floral meristems, a *WUS*-independent pathway can also control stem cell activity.[46] The stem cell activity is repressed by HD-zip III transcription factors, including PHABULOSA (PHB), PHAVOLUTA (PHV) and CORONA (CNA). Premature termination of the floral meristem is partially rescued in *wus phb phv cna* quadruple mutants. *PHB, PHV, CNA* are the targets of miR165/166 that has been repressed by ARGONAUTE10 (AGO10).[47] Another factor, REVOLUTA (REV) is required for floral meristem specification; REV antagonizes the function of PHB/PHV/CAN.[48,49] In addition, *WUS* expression is repressed by an *ERECTA* (*ER*)- and *JABBA* (*JAB*)-mediated signaling pathway, independent of the *CLV* pathway.[50] It is reported that *WUS* expression levels significantly increase in *jba-1D/+ er-20* compared to *jba-1D/+*.[50]

In most events, the key for floral meristem determinacy is the repression and silencing of *WUS*, a central player in the establishment and maintenance of stem cell activity. Meanwhile, the repression of another key gene, *CLV3*, may also be necessary for floral meristem determinacy. It was reported that *CLV3* expression is transcriptionally repressed by FAR-RED ELONGATED HYPOCOTYL3 (FHY3) in flower development.[51] Notably, repression of *CLV3* is observed within 4 hours and is independent of cycloheximide activity when *KNU* is expressed; this result indicates that KNU may directly repress both *CLV3* and *WUS* at floral stage 6, thus ensuring the termination of robust floral stem cell activities within a narrow window of time.[8]

Conclusions and future perspectives

Floral meristem determinacy is governed by a complex regulatory network (Figure 1). In this network, AG-mediated downstream regulatory pathways play a central role. The activation of *KNU* to silence *WUS* plays a pivotal role for floral meristem termination. This *AG-KNU-WUS* pathway also involves epigenetic events including the dynamic eviction

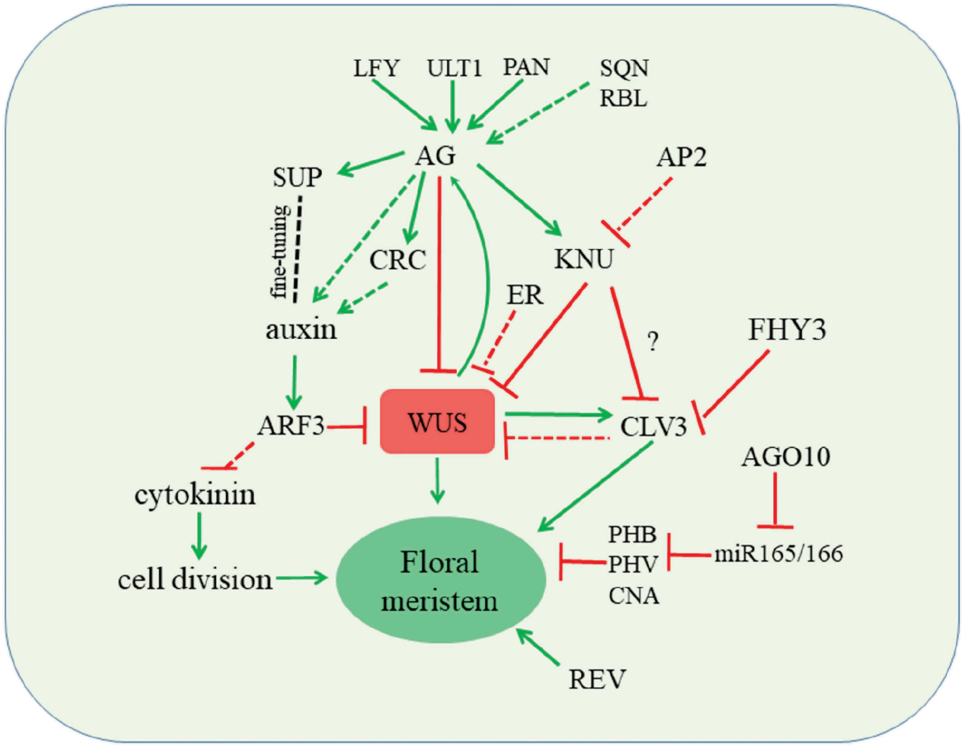

Figure 1. Termination of floral stem cell activity is controlled by multiple pathways. Red and green lines indicate repressions and activations, respectively. Solid and dashed lines indicate direct and indirect regulation, respectively.

of PcG from *KNU* and the recruitment of PcG to *WUS*. In addition, plant hormones regulated by SUP and CRC play important roles in balancing floral stem cell proliferation and differentiation. Because of the complex nature of floral stem cell regulation, many other factors are yet to be discovered. Whether these regulatory mechanisms are conserved in other plant species are intriguing questions whose answers will shed light on the enhancement of future crop yields.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

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