



拟南芥花分生组织活性调控机制的研究进展

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摘要 在拟南芥发育过程中, 花分生组织(floral meristem, FM)活性的维持是花器官原基产生的前提. 在花发育的第6~7期, 雌蕊原基已经完成起始, 此时FM的活性须适时终止以确保雌蕊的正常发育. FM活性从动态维持向程序性抑制与终止状态的转变, 受到精细而复杂的调控. 本文以花发育的早期进程为顺序, 主要综述拟南芥FM活性调控的研究进展, 并重点介绍了FM终止的调控机制, 同时展望了拟南芥FM调控研究对农作物改良的参考意义.

关键词 花分生组织, 干细胞, 程序性终止, 表观遗传, 花发育

分生组织是高等植物产生与分化各种器官的基础. 茎尖分生组织(shoot apical meristem, SAM)是植物地上分生组织的重要组成部分, 它形成于胚胎时期^[1]; 进入胚胎后期, 其中的干细胞不断地增殖分化, 产生植物的地上部分, 包括茎、叶和花^[2]. 进入生殖生长阶段, SAM转变成花序分生组织(inflorescence meristem, IM), IM边缘产生花分生组织(floral meristem, FM)^[3~7]. 随着发育的进行, FM在特定的位置依次产生不同类型的花器官原基, 最终形成功能完全的花. 作为繁殖器官, 花的正常发育对被子植物的生殖过程和连续的生活史至关重要.

FM活性维持与终止机制的研究是植物学研究的重要课题. 本文主要综述模式植物拟南芥花芽中FM活性的建立与维持的分子机制和FM活性终止的程序性调控过程.

1 分生组织活性的建立与维持

1.1 CLAVATA(CLV)-WUSCHEL(WUS)反馈调节通路

在拟南芥中, 同源异型基因框WUS以一种非细胞自主的方式决定并维持FM的活性^[8,9]. 在wus-1功能缺失突变体中, FM活性无法正常建立和维持, 花的雄蕊仅1~2枚, 不产生心皮^[10,11]. SPLAYED(SYD)是一个SWI/SNF家族的染色质重塑因子, 它能利用ATP水解释放的能量改变其所结合的顺式作用元件的染色质状态, 促进下游基因的表达^[12]. 表型观察发现, SYD功能缺失的突变体显示多种发育缺陷, 包括植株矮小、生长缓慢、叶片极性缺陷、胚珠生长停滞和SAM活性的缺失. 研究显示, SYD被招募到WUS启动子, 在组织中心(organizing center, OC)细胞中直接激活WUS的表达, 从而建立和维持FM的活性^[13]; 该过程伴随着WUS

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染色质上组蛋白H3第4位赖氨酸残基的三甲基化(H3K4me3)活跃标记的形成^[14,15]。WUS蛋白是一种可移动的因子,它通过胞间连丝进入上方中心区(central zone, CZ)的干细胞中,并结合到*CLV3*启动子和3'UTR区域上的多个顺式作用元件上,特异性地识别“TAAT”序列,以直接激活干细胞的标志基因*CLV3*的表达^[8]。另外, WUS对*CLV3*顺式作用元件的结合具有阈值效应:低浓度和高浓度的WUS分别激活和抑制*CLV3*的表达^[16]。重要的是,干细胞区中WUS对分生组织活性的维持至关重要。研究显示,如果WUS通过胞间连丝向干细胞区的迁移被抑制,植株则无法产生功能完全的分生组织^[8,9],表明WUS在决定干细胞的属性与活性的维持上发挥着关键的作用。

*CLV3*多肽是*CLV3/ESR(CLE)*家族的成员^[17],由分生组织中CZ区的干细胞特异表达^[18],而后被分泌至细胞外行使配体功能,它被*CLV1*, *CLV2*, *CORYNE* (*CRN*)和*CLAVATA3 INSENSITIVE RECEPTOR KINASES(CIKs)*等组成的跨膜受体复合物所感知,并驱动受体胞内域的磷酸化,最终将信号传递到细胞核内以抑制*WUS*的表达^[11,18~21]。因此, *WUS*的表达上调会引起干细胞数目的增加和*CLV3*表达的增强,而*CLV3*表达增强又会导致*WUS*表达的抑制,这就形成了*CLV-WUS*负反馈调控通路。在花发育的早期阶段, *CLV-WUS*负反馈调控通路主要维持FM干细胞数目的稳态。

值得一提的是,在拟南芥的分生组织中,存在两种补偿机制,它们在维持干细胞数目的平衡上起着不可替代的作用。在*clv1*突变体中,受体激酶基因*BARELYANYMERISTEMS(BAMs)*的表达水平明显增加,部分行使*CLV*信号通路中受体的功能,以补偿因*CLV1*功能缺失对分生组织产生的影响,被称为“主动补偿”^[22,23]。此外,近期研究发现,在*clv3*突变体中, *CLV*信号通路的配体基因*CLEs*的表达水平并没有发生明显变化;相比*clv3*突变体, *clv3 cles*中分生组织活性明显增强,表明*CLV3*功能缺失后, *CLEs*在*CLV*信号通路中发挥配体的功能,以补偿受损的*CLV3*功能,从而参与干细胞稳态的维持^[24],这种旁系同源基因通过不改变自身表达水平来补偿遗传扰动的方式,被称为“被动补偿”^[22]。

1.2 多种因子参与干细胞活性的维持

细胞分裂素(cytokinin)在维持干细胞活性上发挥

关键作用,其功能由转录因子*MYB3R1*和*MYB3R4*介导^[25]。研究显示,外源cytokinin处理能明显促进干细胞的分裂;在*myb3r1 myb3r4*双重突变体中,相同的处理却无法促进细胞的分裂。*MYB3R1*存在于细胞核中;在有丝分裂开始前, *MYB3R4*存在于细胞质中,有丝分裂开始后则被转运至细胞核中,与*MYB3R1*形成二聚体,进而直接激活参与细胞分裂相关基因的表达,最终促进干细胞的分裂。另外, *MYB3R1*和*MYB3R4*广泛地存在于FM中,且调控*MYB3R4*核质穿梭的因子在FM中也存在较强的表达^[25],表明cytokinin可通过*MYB3R1*和*MYB3R4*维持花干细胞的分裂与活性的维持。

此外, cytokinin通过*CLV-WUS*途径维持干细胞活性。根据之前的报道, cytokinin处理野生型植株,可诱导出类似*clv*功能缺失突变体的表型,导致*WUS*的表达增强^[26]; cytokinin连续处理*clv3*突变体,也能导致*WUS*的表达水平明显增加,说明cytokinin诱导的*WUSCHEL*表达通过*CLAVATA*依赖和*CLAVATA*非依赖两条途径进行^[26,27]。type-B型ARABIDOPSIS RESPONSE REGULATORS(B型-ARRs)是cytokinin信号响应基因的调控因子^[28]。研究发现, *WUS*是B型-ARR因子的直接靶基因; *arr1/10/12*三重突变体产生的SAM较小,这也意味着cytokinin对干细胞的维持很重要^[29]。*LONELY GUY (LOG)*是一种cytokinin合成基因,水稻中其功能的丧失会导致SAM提前终止^[30]。

*Knotted 1-like homeobox(KNOX)*基因,包括*SHOOT MERISTEMLESS(STM)*, *BREVIPEDICELLUS (BP)/Knotted 1-like from Arabidopsis thaliana 1 (KNAT1)*, *KNAT2*和*KNAT6*,它们对维持分生组织活性十分重要,且存在功能冗余^[31~33]。*STM*属于同源型蛋白家族的成员,并广泛表达于SAM和早期FM中^[34],其对干细胞活性的建立及维持发挥重要的作用^[35,36]。在*STM*功能缺失的植株中,花器官数目明显减少^[37]。*STM*主要通过三条途径调控干细胞活性。(i) *STM*通过促进cytokinin的合成维持分生组织的活性^[38,39];(ii) *STM*通过与转录因子FLOWERING LOCUS T(FT)-FLOWERING LOCUS D (FD)复合体互相作用,激活花身份基因*LEAFY(LFY)*和*APETALA1(API)*,并维持干细胞活性^[40];(iii) *STM*通过促进*CLV3*的表达,维持FM活性^[34]。其中, *STM*能特异识别并结合*CLV3*启动子上的“TGTC”基序,并发挥转录激活因子的作用;另外,

STM能直接与WUS蛋白互作, 并增强WUS对*CLV3*基因座的结合^[34].

Polycomb-group(PcG)复合体的PRC1亚复合体通过调控KNOX家族基因的表达维持分生组织干细胞数目的稳态. *AtRING1a*和*AtRING1b*是PRC1的成员, 在*atring1a atring1b*双重突变体中, 出现异位的分生组织^[38]. 研究发现, *AtRING1a*, *AtRING1b*和LIKE HETEROCHROMATIN PROTEIN1(LHP1)形成复合物, 其与催化染色质组蛋白H3第27位赖氨酸三甲基化(H3K27me3)修饰的PRC2核心成员CURLY LEAF (CLF)结合, 从而导致*KNOX*基因的沉默^[41].

HAIRY MERISTEM(HAMs)蛋白是GRAS结构域家族的成员, 在分生组织中与*WUS*的表达区域存在重叠, 且*WUS*-HAMs之间的相互作用参与分生组织活性的维持^[42-44]. 在*hams*突变体中, SAM和FM都无法形成. *WUS*蛋白的HAMs结合域(HAMs binding domain, HBD)参与*WUS*-HAMs的互相作用, HBD缺失的*WUS*蛋白无法挽救*wus*突变体分生组织缺陷的表型^[44]. 另外, 在分生组织中, HAMs决定*CLV3*正确的表达模式^[45]. 在SAM中, HAMs和*CLV3*表达的区域呈互补分布. 在野生型植株中, *CLV3*表达于分生组织OC区上方的干细胞区; 而在*hams*突变体中, *CLV3*则异位表达在OC区; 相比*hams*突变体, 在*wus-1 hams*突变体的SAM中, 检测不到*CLV3*的信号. 因此, HAM和*WUS*协同调控*CLV3*的时空表达: *WUS*在干细胞中激活*CLV3*的表达, 而HAMs则在顶端-基部轴向上限定*CLV3*的表达区域^[45].

HD-ZIP III家族基因*PHABULOSA(PHB)*, *PHAVOLILA(PHV)*和*CORONA(CNA)*在分生组织活性调控中也起着重要的作用^[46,47]. 表型观察发现, *PHB/PHV/CNA*的突变会导致植株分生组织活性增强, 花器官数目明显增加; 相比*wus-1*单突变体, *wus-1 phb phv cna*四重突变体可产生有功能的分生组织, 说明*PHB/PHV/CNA*以独立于*WUS*的方式调控干细胞的活性^[47]. miRNA在拟南芥花发育过程中起着十分重要的作用. 当miRNA前体被加工为成熟的miRNA后, 它们被组装成含有ARGONAUTE(AGO)的RNA诱导沉默复合物(RNA-induced silencing complex, RISC), 并以序列特异性的方式抑制靶基因的表达^[48]. AGO蛋白家族的两个成员AGO1和AGO10是RISC复合物的关键组成部分, 它们参与miR165/166调控植物生长发育的过程^[49]. 研

究发现, 在分生组织中, miR165/166通过靶向*PHB/PHV/CNA*, 调控分生组织活性^[46]. 另外, miR394也参与分生组织中干细胞身份的确立^[50]. 在分生组织中, 表皮层细胞特异地产生miR394, 它能移动至下层细胞中, 在AGO1的介导下, 靶向*LEAF CURLING RESPONSIVENESS(LCR)*并抑制其表达^[50], 这对*WUS*建立和维持干细胞活性的功能是必须的.

除此之外, 还存在其他因子通过调控*WUS*的表达来维持FM的稳定性. 根据之前的报道, 与DNA修复有关的因子BRCA1-associated RING domain 1(BARD1), 其突变体植物表现出严重的分生组织缺陷, 而*BARD1*超表达会导致*WUS*转录水平的降低, 产生类似于*wus-1*的表型; 研究者发现其通过将*WUS*的表达限定在OC区来影响干细胞的维持^[51]. *ULTRAPETALA1(ULT1)*属于trithorax Group (trxG)家族的成员, 编码一个含有SAND结构域的蛋白. *ult1*突变体具有增大的IM和额外的花器官. 研究表明, *ULT1*负调控FM中*WUS*的表达, 且这种抑制可能与*AG-WUS*通路有关^[51]. *STIMPY(STIP)*编码同源异型结构域的蛋白^[52], 其突变体茎顶存在缺陷. 遗传学分析显示, *STIP*基因功能的缺失可减弱*clv3*突变体的表型; 超表达*STIP*基因则会增强*clv3*的表型; *STIP*可能通过正调控*WUS*来影响FM的维持. 另外, 在SAM中, 生长素(auxin)的局部积累能触发分化, 尽管干细胞在植物整个生命周期中都能被维持. 研究者发现, *WUS*通过广泛的转录调控来限制干细胞中的auxin信号, 同时允许低水平的信号输出, 从而平衡干细胞的分化和长期的维持^[53].

综上所述, 在早期花芽中, *CLV-WUS*通路是干细胞增殖与分化的关键“调节器”, STM, HAMs, miRNAs, 染色质重塑因子以及植物激素也发挥不可替代的作用, 共同调控FM活性与干细胞数目的稳态(图1).

2 花分生组织活性的抑制与终止

随着花发育的进行, 不同类型的花器官原基以时间顺序起始, 并根据其器官发生的不同形态特征被定义为不同的花发育时期^[54]. 为了防止干细胞过度增殖影响花器官发育, 从花发育第3期开始, FM活性被适度抑制; 在花发育的第6~7期, 此时雌蕊原基已完成起始, 须在这个短暂的时间窗内适时终止FM活性, 以保证花器官数目的确定性.

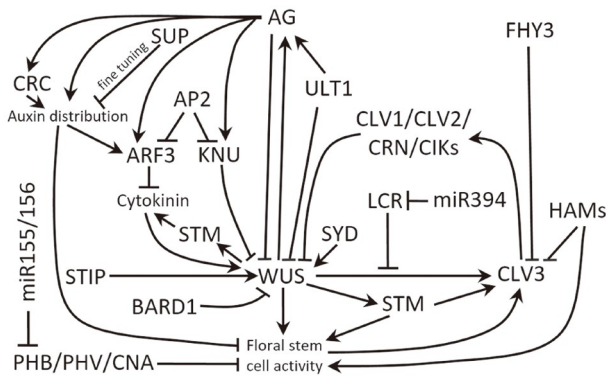


图 1 多种通路和因子调控FM活性的维持与适时终止

Figure 1 Multiple pathways and factors involved in the maintenance and timed termination of floral meristem activity

2.1 在花发育的早期阶段FM被多种因子抑制

同源异型基因*AGAMOUS*(*AG*)是花发育的C类基因,控制着雄蕊和心皮的发育^[55]。从花发育的第3期开始,*WUS*与*LFY*等多种因子共同诱导*AG*表达^[56-60]。研究显示,*LFY*通过招募*SYD*和*BRAHMA*到*AG*第二个内含子上,以去除*AG*染色质上的H3K27me3标记来诱导*AG*的表达^[61]。*ULT1*与*trxG*家族蛋白*ARABIDOPSIS HOMOLOG OF TRITHORAX1*(*ATX1*)相互作用,共同负责形成*AG*染色质上的H3K4me3标记,并以不依赖于*LFY*的方式激活*AG*的表达^[58]。在*AG*功能缺失的突变体*ag-1*中,花器官表现出(萼片-花瓣-花瓣)重复的FM不确定的表型^[3],表明*AG*能够抑制FM的活性。在分子水平上,*AG*通过两种方式抑制*WUS*的表达。其一,*AG*直接结合到*WUS*的基因座上,并通过蛋白互作的方式募集PcG的关键因子*TERMINAL FLOWER 2*(*TFL2*),引起*WUS*位点上H3K27me3标记的生成,从而抑制*WUS*表达^[62]。其二,*TFL2*以不依赖于*AG*的方式结合在*WUS*基因座上,通过*TFL2-AG*蛋白互作,形成染色质环,这阻止了RNA聚合酶II(RNA polymerase II)结合到*WUS*的启动子区,从而抑制*WUS*的表达^[63]。

C2H2型锌指蛋白是植物中一类重要的转录因子,在植物的开花、花器官形成以及花粉和雌蕊的形成上都发挥关键的作用^[64]。在拟南芥中,*SUPERMAN*(*SUP*)编码C2H2型锌指蛋白,从花发育的第3期开始,表达在第3轮与第4轮花器官原基之间的边界区域^[65,66]。在*sup*突变体中,雄蕊数目明显增加,表明*SUP*调控花干细胞的活性及FM的确定性发育过程^[66,67]。在*sup ag clv3*三

重突变体中,FM的直径显著大于*ag clv3*^[68],说明*SUP*可能以一种平行于*AG*和*CLV3*的方式抑制FM活性。遗传学分析发现,*sup wus-1*花的雄蕊1枚,心皮缺失,与*wus-1*的表型类似^[69],说明*wus*突变上位于*sup*,暗示*sup*中雄蕊数目的增加依赖于*WUS*。另外,在花发育的第4~5期,*sup*的FM中*CLV3*和*STM*的表达区域都明显增加^[69]。在花发育的过程中,*SUP*的表达区域与*CLV3*和*WUS*都不重叠,表明*SUP*以非细胞自主的方式调控FM的活性^[69]。相关研究发现,*SUP*与PcG复合体的成员*CLF*和*TFL2*形成抑制复合体,在第3轮与第4轮花器官原基的边界区域负调控auxin合成基因*YUCCA1/4*(*YUC1/4*)的表达,从而调控FM活性^[69]。

组蛋白乙酰化是组蛋白乙酰基转移酶(histone acetyltransferase, HAT)将乙酰基从乙酰辅酶-A上转移至组蛋白的赖氨酸残基上,从而影响转录因子与DNA结合,最终调控靶基因表达^[70]。*GENERAL CONTROL NONDEREPRESSIBLE 5*(*GCN5*)是一类重要的乙酰基转移酶^[70,71]。研究发现,在*gcn5*突变体中,花序的末端形成顶端花,且发生萼片向雄蕊、花瓣向类心皮的同源异型转变,这些花芽中*AG*和*WUS*的表达区域明显扩大,表明*GCN5*可能通过影响*AG-WUS*的表达调控FM活性^[72]。因此,从花发育的第3期起,*AG*和*SUP*等因子开始适度地抑制FM活性,防止干细胞过度增殖,确保了雄蕊和雌蕊原基数目的稳定和正确的起始。

2.2 FM活性在花发育第6~7期终止

之前的研究显示,在35S:*AG*植株中,花的数目减少,且IM最终形成末端花(terminal flower),但这些花仍能形成雌蕊及发育异常的雄蕊^[73],表明超表达*AG*并不能明显抑制FM的活性表明除了*AG*外,还存在其他因子抑制花干细胞活性。*AG*的两个直接靶基因*KNUCKLES*(*KNU*)和*CRABS CLAW*(*CRC*),在FM活性的终止上都发挥关键的作用^[15,74,75],且二者的激活是在*AG*与花的同源异型因子*SEPALLATA3*(*SEP3*)形成的异源四聚体的参与下实现的^[76]。

(1) *AG-KNU-WUS*通路。*KNU*编码一个C2H2型的锌指蛋白。在*knu*突变体中,雌蕊中着生异位的雄蕊和雌蕊,FM表现出较强的不确定性,这是由*WUS*异位表达引起的^[77,78]。在35S:*KNU-GR*植株中通过激素处理诱导*KNU*超表达,FM活性被强烈的抑制,花的雌蕊缺失^[78],表型类似于*wus-1*。从花发育的第6期开始,*KNU*

表达在FM的中心区域, 覆盖着CZ和OC区^[77,78], 作为转录抑制因子, KNU在*WUS*的适时抑制与沉默过程中发挥了关键的作用^[15,78,79]。

在FM中, AG直接诱导KNU的表达^[78]。在花发育的第3期之前, PcG复合体成员EMBRYONIC FLOWER 2 (EMF2)和FERTILIZATION INDEPENDENT ENDOSPERM(FIE)结合在KNU启动子上的多梳蛋白响应元件(polycomb responsive element, PRE)上, 引起抑制性标记H3K27me3在KNU基因座上富集, 使KNU表达被抑制。当花发育至第3期, AG蛋白直接结合在KNU启动子的CArG框上。由于PRE与CArG框在序列上重叠, 导致AG结合于KNU启动子之后直接竞争性地驱逐了PcG蛋白复合体, 进而导致H3K27me3无法被继续维持。从花发育第3~6期, 经过1~2次细胞分裂, KNU基因座上的H3K27me3水平被逐渐稀释, 使得KNU的表达被激活。KNU开始表达的时间点对FM的适时终止至关重要。研究显示, KNU表达的提前或延迟分别产生无心皮的花或异位增生的花器官的表型^[80]。

作为干细胞的决定基因, *WUS*表达的抑制与沉默是FM活性终止的前提。在花发育的第6~7期, KNU介导的程序性调控过程抑制并终止*WUS*的表达^[15]。首先, KNU结合到*WUS*启动子的近端, 导致表观激活因子SYD被驱逐, 进而降低*WUS*染色质的可接近性^[15]。在*WUS*出现转录抑制的4 h后, *WUS*染色质上的活跃标记H3K4me3和组蛋白H3乙酰化的水平都出现明显的下降^[15]。

在SYD被驱逐的过程中, KNU也参与了组蛋白去乙酰化复合体(HDAC)介导的*WUS*转录抑制^[15,79]。首先, 锚定蛋白MINI ZINC FINGER2(MIF2)直接结合到*WUS*第一个内含子上, 而后通过蛋白互作的方式, 招募KNU, TOPLESS(TPL)和HISTONE DEACETYLASE19(HDA19), 形成转录抑制复合体, 抑制*WUS*的表达^[79]。*hda19*突变体中*WUS*的表达变强; 组蛋白去乙酰化酶活性的抑制剂曲古抑菌素A(TSA)处理导致野生型植株中*WUS*的表达强烈增加, 并且在MIF2超表达的植株中将*WUS*水平回复到野生型状态, 此研究表明*WUS*的抑制涉及了HDAC介导的组蛋白去乙酰化过程^[79]。

KNU单独介导的*WUS*转录抑制可能不是一个长效的抑制机制, 因为在诱导KNU超表达后的4 h, *WUS*的mRNA水平先出现显著下调, 随后逐渐回升^[15], 说明

还需要其他的因子参与*WUS*的稳定沉默。研究显示, KNU结合*WUS*启动子后, 通过与PcG复合体中关键因子FIE互作, 募集PcG复合体到*WUS*染色质上, 导致抑制性标记H3K27me3在*WUS*基因座上富集^[15], 这对*WUS*的沉默至关重要。在FIE基因沉默的植株中, *WUS*表达延长, 且雌蕊中着生异位的类心皮的组织。另外, 在*tf12*和*clf*突变体中超表达KNU, 植株仍能产生雌蕊正常的花^[15]。这表明在花发育的第6~7期, KNU通过整合转录抑制和表观沉默机制来终止*WUS*的表达, 从而终止FM的活性。

PRC1能够特异识别H3K27me3, 并维持靶基因沉默的过程^[81]。研究表明, 具有BAH(Bromo adjacent homology)结构域的PRC1新组分蛋白SHORT LIFE(SHL)和EARLY BOLTING IN SHORT DAYS(EBS)能够与PRC1的组分EMF1和BMI1互作, 形成BAH-EMF1抑制复合体与TFL2一起参与到大量基因的调控中^[81]。因此EBS和SHL蛋白可能也参与了FM的调控。

(2) AUXIN RESPONSE FACTOR 3(ARF3)参与FM活性的终止调控。Auxin响应因子ARF3参与FM活性的终止。在花发育的第1~7期, ARF3在FM中有着很强的表达^[82]。研究显示, 在花发育的3~4期, AG在FM中抑制ARF3的表达, 导致ARF3的靶基因ISOPENTENYL-TRANSFERASEs(IPTs)被解除抑制。IPTs是cytokinin的合成基因, 因此FM中细胞分裂能力增强, 干细胞活性得以维持。进入花发育的第5~6期, auxin和AG正调控ARF3的表达, ARF3能抑制cytokinin合成相关基因和cytokinin受体基因ARABIDOPSIS HISTIDINE KINASE4(AHK4)的表达, 从而间接抑制*WUS*^[83]。在AG的参与下, ARF3也能直接抑制*WUS*的表达, 调控FM的活性^[82]。

(3) CRABS CLAW(CRC)参与FM活性的终止调控。在花发育的第5~6期, AG在FM中直接激活YABBY家族的转录因子CRC的表达^[74,84,85]。遗传学分析显示, 相比*knu-1*, *crc-1 knu-1*中FM表现出更强的不确定性^[74]。通过RNA-seq分析, 研究人员发现CRC通过调控auxin分布来抑制FM活性^[74]; 利用auxin转运抑制剂处理*crc-1 knu-1*, 其雌蕊的强烈不确定表型可回复至*knu-1*的状态, 表明CRC通过调控auxin参与FM活性的抑制。进一步研究发现, 在花发育的第6期, CRC通过两种途径调控auxin在FM中心区域的积累来抑制FM活性: 其一, CRC和AG协同地促进植物auxin合成基因

*YUC4*的表达^[75], 其二, CRC通过结合auxin负调控因子编码基因*TORNADO2*(*TRN2*)启动子上的YABBY结合位点, 负调控*TRN2*的表达^[86-89]. 通过以上方式, CRC促进FM中心区域auxin极大值的形成, 进而抑制*WUS*的表达和促使FM活性的终止^[74].

(4) FAR-RED ELONGATED HYPOCOTYL3 (*FHY3*)和*APETALA2*(*AP2*)调控FM活性. 研究发现, 相比*ag-10*, *fhy3 ag-10*中FM的缺陷程度明显增加; 超表达*SEPALLATA2*(*SEP2*)能挽救*fhy3 ag-10*中FM缺陷的表型. 此外, 作为转录因子, 光敏色素A信号通路中的正调控因子*FHY3*可直接结合*CLV3*和*SEP2*的启动子序列, 分别抑制*CLV3*和促进*SEP2*的表达, 进而调控花确定性发育的过程^[90].

*AP2*和*AG*分别是花发育的A类和C类基因, 并分别决定第1~2轮和第3~4轮花器官的发育^[55,91,92], 且二者在功能上相互拮抗^[93]. 此外, *AP2*也参与FM活性的调控. 一方面, *AP2*通过直接抑制*ARF3*, 导致*WUS*的去抑制来维持分生组织的活性^[82]; 另一方面, 在花发育的第6期, *AP2*负调控*KNU*的表达^[93]. 所以, 在FM活性的调控上, *AP2*可能发挥“制动器”的作用, 防止FM过早终止以保证雌蕊原基适时的产生.

综上所述, 已有越来越多的因子被发现参与FM的调控^[80,94](图1). 这些因子协同作用于激素调控、信号转导和转录调控途径来精细调控花干细胞活性并确保FM的适时终止.

3 展望

分生组织的调控对于禾本科作物的分蘖数、穗和籽粒数目具有直接的决定作用, 并决定了作物的产量. 当前和今后一段时期, 粮食问题依然是人类面临的重大挑战.

因此, 如何将FM调控的分子机制应用到农作物的增产上, 是一个亟待解决的问题.

植物中FM调控的机制相对保守^[24,95,96], 尤其是*CLV-WUS*核心调控通路^[24]. 研究显示, *CLV-WUS*信号通路中相关因子的突变可潜在提高作物的产量^[95,97-99]. 近年来, 在水稻(*Oryza sativa*)、玉米(*Zea mays*)和番茄(*Solanum lycopersicum*)等作物中, 通过突变体筛选、数量性状基因座分析和全基因组关联分析, 已鉴定出FM调控相关基因, 并对其进行了相应的研究^[100]. 通过CRISPR/Cas9手段, 编辑玉米中*CLV3*的同源基因, 研究人员得到了高产的基因型^[95], 为分子育种提供了重要的理论依据和新的思路. 另外, 番茄中*CLV3*同源基因的突变导致干细胞的过度增殖, 产生更大的果实^[97]; 在番茄中, 研究人员通过筛选T-DNA插入的突变体, 获得了*AP2/ERF*转录因子家族的*EXCESSIVE NUMBER OF FLORAL ORGANS*(*ENO*)基因突变的植株^[98]. 与野生型植物相比, *eno*突变体产生更多的花器官和更大的果实^[98]. 研究显示, *eno*突变体中*SIWUS*表达区域扩展, 表明*eno*的表型跟*SIWUS*和*SICLV3*这两个分生组织核心基因有关^[98]. 以上研究都是基于对FM中*CLV3-WUS*信号通路的调节来增加果实的大小. 此外, 番茄中的*SIKNU*基因突变也会造成果实的明显增大^[79], 表明*KNU-WUS*调控通路在番茄和拟南芥中相对保守. 因此, 在作物中, 通过进化分析和实验手段, 寻找并研究与拟南芥中同源并参与FM调控相关的因子, 将为增加作物产量提供新的可能^[99]. 随着越来越多新的分生组织调控因子被发现和CRISPR/Cas9等基因编辑工具的出现, 研究者可以通过改造作物中调控FM活性的基因用于产量优化^[99]. 另外, 借助现代分子生物学手段, FM调控的基础研究也将为培育经济花卉新品种提供潜在的理论参考.

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Research advances on the regulatory mechanisms of floral meristem activity in *Arabidopsis*

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In *Arabidopsis*, maintaining the floral meristem (FM) activity is a prerequisite for initiating floral organ primordia. Studies showed that during the floral stages 6–7, the carpel primordia arose and FM activity was terminated to ensure the proper development of carpels. Hence, from dynamic balance to timed termination, FM activity is controlled via a fine-tuned complex regulatory network. Herein, we mainly introduced the progress in the research on FM activity regulation in *Arabidopsis* during early floral development and focused on the regulatory mechanism of FM determinacy control. Further, we provided insights on the potential benefits of research on *Arabidopsis* FM in enhancing crop yields.

floral meristem, stem cell, programmed termination, epigenetics, flower development

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