

中科院团队揭秘植物春季开花的新机制

Scientists from Chinese Academy of Sciences discovered the New Mechanism for Plants Blooming in Spring



11月2日,《Nature》期刊最新发表了中国科学院上海植物逆境生物学研究中心何跃辉课题组完成的题为“Embryonic epigenetic reprogramming by a pioneer transcription factor in plants”的研究论文。

何跃辉课题组于2016年12月在Nature Genetics报道了模式开花植物拟南芥在营养生长阶段,如何利用长期低温(冬季)沉默开花抑制关键基因FLC的表达并随后在常温维持其沉默(‘低温记忆’)的表观遗传机制,这一机理使植物具备了在春季开花的能力(春化作用)。在本研究中,该课题组进一步揭示了开花后的胚胎发育早期擦除‘低温记忆’,激活FLC基因使下一代又需经历冬季低温才能在春季开花的分子机制。

春化作用是指某些植物必须经历一段时间的持续低温才能由营养生长阶段转入生殖阶段生长的现象。植物如何响应并记忆长时间的低温处理即春化作用一直是表观遗传学和发育生物学的研究热点。解析植物如何响应冬季低温,并在春季气温上升后能“记住”其冬季低温经

历、以适时开花的分子机制，具有重要的理论和实际应用价值。

本研究揭示了开花后的胚胎发育早期擦除‘低温记忆’，重新激活 FLC 基因的分子机制；此外、研究发现了植物营养生长期的‘胚胎记忆’现象及调控这一现象的表观遗传机理。营养生长阶段的植株经历春化作用后，FLC 位点的组蛋白携带大量的 H3K27me3 等标记，一直处于沉默状态，直至受精作用完成。在胚胎发育早期（原胚）FLC 被一个种子特有的‘先驱’转录因子（Pioneer transcription factor）重新激活，组蛋白上的抑制转录的 H3K27me3 被逐步消除，而促进 FLC 表达的组蛋白修饰被大量地添加上去，FLC 的激活表达持续整个胚胎发育时期；并且此激活状态在种子发芽出苗后，因组蛋白标记在细胞分裂中的传递得以维持，形成了苗期的‘胚胎 FLC 表达记忆’。从而防止植物在过冬前或过冬时开花。

综上，本研究揭示了植物早期胚胎染色质状态重编程的崭新分子机制，同时也阐述了胚胎中的基因激活如何传递到发育后期的表观遗传机理，是开花调控分子与遗传机制的重要突破。该研究不仅具有重要的理论意义，同时也为其在作物花期调控的生产应用提供了新的作用靶点。



Embryonic epigenetic reprogramming by a pioneer transcription factor in plants

植物基因在胚胎发育早期的从头激活的分子与表观遗传机理

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2017年11月2日

doi:10.1038/nature24300

Epigenetic modifications, including chromatin modifications and DNA methylation, have a central role in the regulation of gene expression in plants and animals. The transmission of epigenetic marks is crucial for certain genes to retain cell lineage-specific expression patterns and maintain cell fate. However, the marks that have accumulated at regulatory loci during growth and development or in response to environmental stimuli need to be deleted in gametes or embryos, particularly in organisms such as plants that do not set aside a germ line, to ensure the proper development of offspring. In *Arabidopsis thaliana*, prolonged exposure to cold temperatures (winter cold), in a process known as vernalization, triggers the mitotically stable epigenetic silencing of the potent floral repressor *FLOWERING LOCUS C* (*FLC*), and renders plants competent to flower in the spring; however, this silencing is reset during each generation. Here we show that the seed-specific transcription factor LEAFY COTYLEDON1 (*LEC1*) promotes the initial establishment of an active chromatin state at *FLC* and activates its expression *de novo* in the pro-embryo, thus reversing the silenced state inherited from gametes. This active chromatin state is passed on from the pro-embryo to post-embryonic life, and leads to transmission of the embryonic memory of *FLC* activation to post-embryonic stages. Our findings reveal a mechanism for the reprogramming of embryonic chromatin states in plants, and provide insights into the epigenetic memory of embryonic active gene expression in post-embryonic phases, through which an embryonic factor acts to ‘control’ post-embryonic development processes that are distinct from embryogenesis in plants.