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"To remember or forget: Insights into the mechanisms of epigenetic reprogramming and priming in early plant embryos"

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Abstract

Chromatin is dynamically modified throughout the plant life cycle to regulate gene expression in response to environmental and developmental cues. Although such epigenetic information can be inherited across generations in plants, chromatin features that regulate gene expression are typically reprogrammed during plant gametogenesis and directly after fertilization. Nevertheless, environmentally induced epigenetic marks on genes can be transmitted across generations. Moreover, epigenetic information installed on early embryonic chromatin can be stably inherited during subsequent growth and influence how plants respond to environmental conditions much later in development. Here, we review recent breakthroughs towards deciphering mechanisms underlying epigenetic reprogramming and transcriptional priming during early plant embryogenesis.

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Keywords

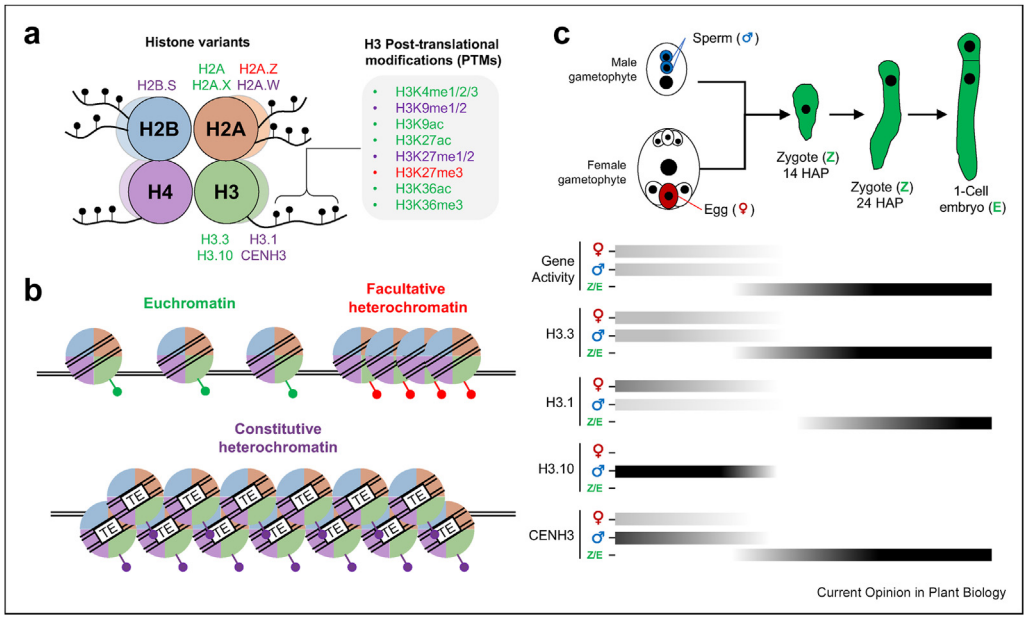
Chromatin, Epigenetics, Inheritance, Embryo, Arabidopsis, Plants, FLC.

Introduction

After fertilization, the transcriptional status of highly specialized gametes needs to be reprogrammed in zygotes to initiate gene expression programs necessary for embryogenesis [1]. At the same time, transposons must be silenced in the germline during reproduction to prevent their genome-destabilizing activities [2]. Epigenetic modifications of chromatin are dynamically regulated during these processes to balance the recapitulation of morphogenesis with the silencing of selfish genetic elements. Characterizing the mechanistic basis of epigenetic reprogramming during reproduction is therefore of central importance for understanding both inheritance and development.

Epigenetic states are characterized by a combination of factors that influence gene activity beyond what is encoded in their DNA sequences. In nucleosomes, DNA is wrapped around a histone octamer of four core histones: H2A, H2B, H3 and H4 (Figure 1a). In flowering plants, the H3, H2A and H2B histones have diversified into distinct variants, which in different combinations, confer a spectrum of gene-regulatory properties [3–5]. In addition, amino acids in the N-terminal tails of histones are chemically modified by various post-translational modifications (PTMs) which can affect gene activity [6]. Methylation of cytosines in DNA is an epigenetic mark that is associated with transposon-rich constitutive heterochromatin and functions together with histone-based mechanisms to ensure transposon silencing [7,8]. Chromatin can be broadly categorized into gene-rich euchromatic or transposon-rich heterochromatic domains (Figure 1b). Euchromatin is enriched for certain histone variants (e.g. H3.3, H2A and H2A.X) and PTMs (e.g. H3K4me1/2/3, H3K9ac, H3K27ac, H3K36ac, H3K36me3), which increase accessibility for transcription factors (TFs) and consequently increase transcriptional activities. In contrast, heterochromatin is associated with different epigenetic features (DNA methylation, H3.1, H2B.S. H2A.W, CENH3 variants and H3K9me1/2, H3K27me1/2 PTMs) which mark highly condensed chromatin that is inaccessible to TFs and thus transcriptionally silent [3,5] (Figure 1a&b). Chromatin states can transition between euchromatin and facultative heterochromatin to regulate gene expression in response to environmental and developmental signals. Facultative heterochromatin is often associated with H3K27me3 repressive marks (Figure 1a&b) that are established by Polycomb complexes and erased by histone demethylases [9]. Epigenetic reprogramming involves the

Figure 1



Chromatin states and epigenetic reprogramming during the gamete to zygote transition. (**a**) Schematic representation of a nucleosome. DNA is wrapped around a histone core octamer composed of histones H2A, H2B, H3 and H4. These histones are further subjected to a series of post-translational modifications (PTMs). Distinct histone variants and PTMs are color-coded based on their association with (**b**) euchromatic (green), facultative heterochromatin (red) and constitutive heterochromatin (purple). Here, we focus on the variants and modifications described in the main text. For an extended review on histone variants, see Refs. [3,5]. (**c**) Changes in gene activity and distribution of H3 variants (H3.3, H3.1, H3.10 and CENH3) in egg (red), sperm (blue), zygotes and one-cell embryos are shown. The bulk of H3 variants are replaced in the zygote, thus limiting the inheritance of the parental epigenetic state [20,21].

integrated actions of enzymes related to histone turnover, histone PTMs and DNA methylation [10,11].

In contrast to what is found in animals, plant germlines are not set aside early during development but are derived from sporophytic cells after long periods of growth and exposure to environmental stress. Relative to animals, plants therefore have a greater capacity to record environmental information in their epigenetic codes and transmit it to the next generation to improve offspring fitness [12]. Although the transmission of gene-regulatory epigenetic information across generations has been documented in plants, the underlying mechanisms are largely unknown [13]. In this review, we highlight recent insights into how epigenetic states are reprogrammed during early embryogenesis or installed in embryonic chromatin to regulate growth and physiology much later in development.

Epigenomic reprogramming during the gamete-to-zygote transition

Egg and sperm have distinct chromatin features and transcriptional programs that must be reprogrammed upon fertilization to generate a transcriptionally active and totipotent zygote. Consistent with large-scale reprogramming of zygotic chromatin, the vast majority of maternally and paternally derived alleles appear to be equally expressed in zygotes [14–16]. Although maternal biases have been reported in zygotes [15,17–19], this is at least partially due to carry over of transcripts from the egg cytoplasm. Thus, there are currently different interpretations regarding the transcriptional activities of maternal and paternal alleles in zygotes. Regardless, H3 variants appear to be completely removed from chromatin in zygotes within a few hours after fertilization and subsequently replaced by newly produced H3 proteins before the first zygotic division [20–22]. Because H3 variants and their epigenetic marks regulate gene expression, the reprogramming of H3 proteins before the first zygotic division limits the transmission of epigenetic information from parents to offspring (Figure 1c).

Precisely how parentally derived histones are replaced by their zygotic counterparts remains to be determined. Rather than passive dilution across cell divisions, active mechanisms of histone replacement are expected since the majority of H3 proteins are replaced before the first zygotic division. Mutants in the HIRA chaperone complex do not have defects during reproduction suggesting that other mechanisms, such as SWI/SNF-based chromatin remodeling, are involved in zygotic chromatin reprogramming [21]. Surprisingly, complete removal of H3.3 variants did not cause seed defects [23], whereas H3.3 was shown to be required for pollen viability [24] and the floral transition [25]. The lack of seed defects in

H3.3-deficient mutants may be due to compensation by H3.1 variants, which are abundant in rapidly dividing early embryos. Newly deposited histones in zygotes must be decorated by chemical modifications to confer their gene regulatory properties. For example, H3K36me3 levels are markedly increased during the egg-to-zygote transition [26], which is consistent with large-scale transcriptional activation of plant zygotes [14,15,18,27]. Future research employing advanced microscopy and genomics approaches are expected to reveal where histone variants are deposited in zygotic chromatin, as well as how their epigenetic codes are modified to regulate initial waves of gene expression.

In contrast to gene-rich regions of the genome, heterochromatic regions harboring transposons must be constitutively silenced in gametes and zygotes to protect genome integrity. Topologically associated domains mark regions of the genome that colocalize in three-dimensional space and have similar gene activities [28]. Remarkably, topologically associated domains are rearranged in rice zygotes after fertilization to form compact silencing centers, which are not present in sperm and appear to conceal genes that are silenced in zygotes [29]. DNA methylation associated with heterochromatin is reprogrammed in rice zygotes during fertilization with important contributions from the OsCMT3a/b methyltransferases [19,30,31]. Together with CMT2, 24-nt sRNAs that are produced in embryos after fertilization progressively establish high levels of methylation on transposons during later stages of embryogenesis and seed development [32–38]. Decondensation of chromatin during early embryogenesis appears to promote the production of 24-nt siRNAs, which subsequently help methylate the transposons they are derived from [32]. These sRNAs may include those that associate with AGO4/6/9 to help establish H3K9me2-marked heterochromatin [39].

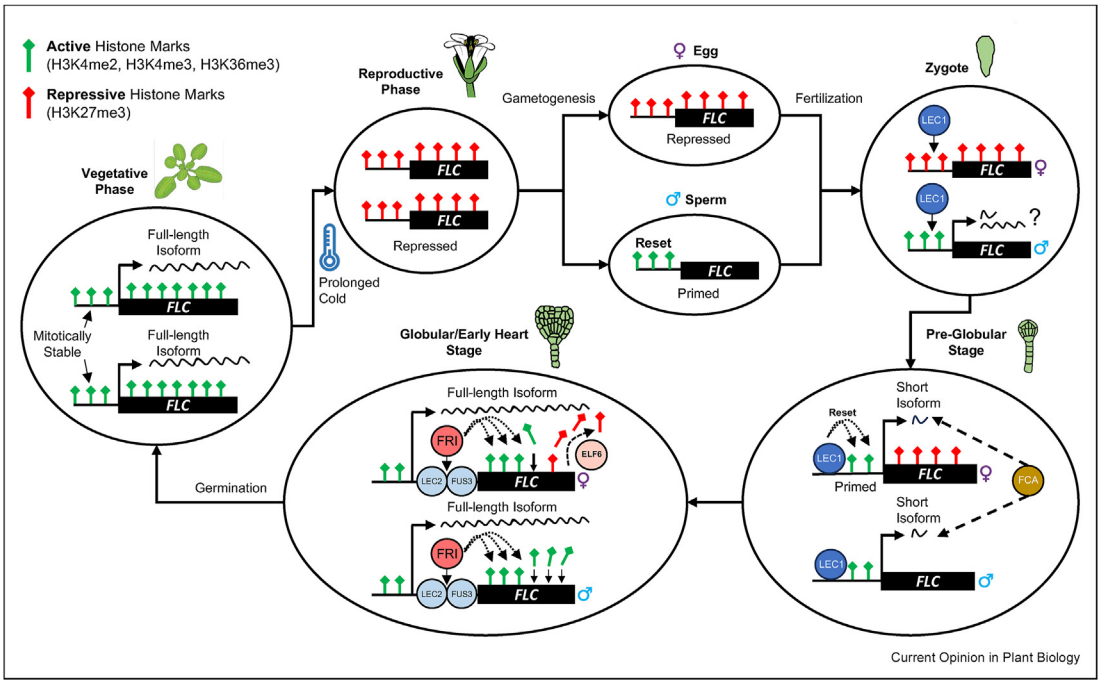
Epigenetic inheritance during the gamete-to-zygote transition and beyond

Although large-scale epigenetic reprogramming appears to limit the inheritance of epigenetic information across generations, recent research indicates how environmentally-induced epigenetic marks acquired in parental generations can be transmitted to their progeny. Repressive H3K27me3 and active H3K4me3 marks were recently shown to co-exist on genes in sperm [40]. In addition to such bivalently marked genes, which may influence gene activities post-fertilization, another class of genes lost H3K27me3 and gained H3K4me3 during sperm formation [40,41]. Consistent with addition of H3K4me3 priming gene activity after fertilization, H3K4me3-marked genes had increased chromatin accessibility in sperm and were more likely to be expressed in early zygotes [40–42].

One such H3K4me3-primed gene is the flowering repressor *FLC* [40,41]. Polycomb-dependent H3K27me3 marks deposited on *FLC* chromatin in response to prolonged cold (i.e. vernalization) are maintained during vegetative growth under warm conditions and enable the vegetative-to-reproductive transition in many Brassicaceae species including *Arabidopsis thaliana* (Arabidopsis) [43]. Indeed, *FLC-GUS* reporters were previously shown to be preferentially expressed from paternal alleles in zygotes and differential chromatin marks on parental *FLC* alleles had been proposed to cause parentally biased expression after fertilization [44,45]. Moreover, the Polycomb-repressed state of *FLC* was shown to be inherited across meiosis and mitosis during egg formation, as well as transmitted to zygotes and subsequent mitotic divisions in early embryos [46]. Remarkably, the duration of cold exposure in maternal vegetative tissues was proportional to how long it took to express *FLC-GUS* in early embryos of the next generation [46]. Therefore, environmental exposure can not only be recorded as epigenetic marks in parents and transmitted across generations, but also appears to quantitatively regulate gene expression in the next generation.

In the following, we describe current models for how *FLC* epigenetic marks are dynamically regulated during embryogenesis to re-establish the vernalization requirement for flowering in the next generation and subsequently prime *FLC* transcriptional activity for after embryogenesis (Figure 2). Cold-induced H3K27me3 repressive marks established during the vegetative phase of parents are either erased in sperm or maintained in eggs [41,46,47]. Directly after fertilization, H3K27me3 is retained on maternal *FLC* alleles while paternal alleles have reduced H3K27me3 and increased H3K4me3 active marks. Beginning in zygotes, the LEC1 TF binds to the *FLC* promoter to remodel chromatin to an active state (i.e. increased H3K36me3 and H3K4me3, and decreased H3K27me3) [48]. Paternal *FLC* alleles may be more transcriptionally active in zygotes due to their reduced H3K27me3 and increased H3K4me3 relative to maternal alleles. From the zygote to globular stages, LEC1-mediated *FLC* activation progressively reinforces this active transcriptional state until maternal and paternal alleles are equally active. However, FCA antagonizes LEC1 activity in early embryos by co-transcriptionally promoting the production of truncated versions of FLC transcripts that encode non-functional proteins [47]. Eventually, LEC1-mediated activation of *FLC* facilitates the recruitment of FUS3 and LEC2 TFs to the cold memory element within the first intron of *FLC* [49]. Subsequently, FUS3/LEC2 recruit FRI at the globular and early heart stages to further activate *FLC* transcription and counteract FCA to produce full-length FLC transcript isoforms [47,49]. The FRI-FUS3/LEC2 complex mediates changes in the chromatin state of FLC by facilitating the accumulation of active histone marks

Figure 2



Model of FLC reprogramming and priming during gametogenesis and embryogenesis. During vegetative growth, the mitotically stable and active epigenetic state of *FLC* favors the transcription of the full-length transcript isoform to repress the transition to the reproductive phase. Prolonged cold establishes the epigenetic repressive state of *FLC* mainly through the accumulation of H3K27me3 marks across the locus [43]. During egg formation, the cold-induced repressive state is meiotically and mitotically stable [46]. In contrast, the repressive epigenetic state of *FLC* is lost in sperm [40,41]. Moreover, active H3K4me3 marks accumulate around the transcription start site of *FLC* in sperm [41]. Upon fertilization, the paternal *FLC* allele becomes transcriptionally more active than the maternal allele likely due its primed epigenetic state (i.e. gain of H3K4me3 and loss of H3K27me3 on paternal allele compared to retained H3K27me3 on maternal allele) [44,45]. Beginning in zygotes, LEC1 binds upstream of FLC and promotes the removal of H3K27me3 marks and accumulation of active H3K4me3 and H3K36me3 marks [48]. However, FCA counteracts LEC1 activity in preglobular embryos by promoting the production of non-functional truncated versions of FLC transcripts [47]. LEC1 progressively increases chromatin accessibility, which eventually allows binding of B3-type transcription factors (LEC2 and FUS3) [49]. LEC2 and FUS3 then recruit FRI [49], which promotes co-transcriptional activities that antagonize FCA and enable the production of full-length FLC transcripts [47]. The sequential and combined activities of LEC1, FUS3/LEC2, FRI and the H3K27me3 demethylase ELF6 [50] establish an epigenetically active state that is mitotically stable for the remainder of embryogenesis, germination and vegetative growth [47,49,51,52].

along the FLC gene [49]. Together with the H3K27me3 demethylase ELF6, this process establishes an active chromatin state that promotes the transcription of the full-length FLC transcript isoform [50]. The combined activities of FUS3, LEC2, FRI and ELF6 at the globular and heart stages, together with contributions from the ABI3 TF during later stages, establish an epigenetically active state that is competent to produce full-length FLC isoforms. This epigenetic state of *FLC* is then mitotically stable for the remainder of embryogenesis, germination and weeks thereafter to repress flowering [47,49,51,52].

The *FLC* example described above supports the notion that epigenetic reprogramming of hundreds of genes in the male germline can prime expression from paternal alleles in zygotes directly after fertilization. Nonetheless, based on the inspection of parental transcript contributions of other candidate primed genes in zygotes [15], the gain of H3K4me3 and loss of H3K27me3 does not appear to be sufficient for preferential expression from paternal alleles in zygotes. This likely depends on whether the TFs that activate expression are present in zygotes and affected by such epigenetic marks, as well as the epigenetic status of maternal alleles. Further investigation is required to test and refine this model. For example, profiling epigenetic marks in the female germline would give insights into which parent is more likely to transmit different epigenetic states to their progeny. Similar to what was described for the transmission of cold memory, stress-induced DNA methylation of specific loci is inherited mainly from mothers due to the activities of DNA demethylases in the male germline [53]. Additionally, thermomemory is transmitted maternally through H3K27me3 demethylation [54]. We speculate that transmission of environmentally induced epigenetic marks through mothers may be

beneficial because plants are more likely to grow in conditions that are similar to their mothers relative to their fathers. Moreover, the egg contributes more cytoplasmic contents to the zygote relative to the sperm. Hypothetically, the mother can therefore transmit more trans-acting factors that re-establish epigenetic marks in zygotes after their apparent widespread erasure due to histone replacement during the gamete-to-zygote transition.

Temperate grasses such as barley and wheat also require prolonged cold exposure of vegetative tissues to switch to reproductive modes of development [55]. In vegetative tissues of common wheat, cold exposure activates the floral promoter *TaVRN1* and represses the floral repressor *TaVRN2* TFs. These transcriptional states are mitotically stable during post-cold growth and induce flowering. Furthermore, the epigenetic states of *TaVRN1* and *TaVRN2* were recently found to be maintained during subsequent egg and sperm formation, and transmitted across generations into zygotes and early embryos [56]. During embryo development, H3K4me3 and H3K36me3 are lost on *TaVRN1* while H3K27me3 gradually increases until it is fully installed in seedlings to establish the vernalization requirement for flowering in the next generation [56]. In contrast to *VRN1*, the epigenetic state of *VRN2* is not reset during embryogenesis but rather upon germination in response to light [56]. Results from a study that profiled H3K27me3, and multiple other epigenetic features, in early seeds and embryos of wheat further indicate that embryonic epigenomes are dynamically reprogrammed after fertilization [57].

Conclusions and future perspectives

The stable inheritance of DNA methylation helps re-establish constitutive heterochromatin to ensure silencing of mutagenic transposons [58,59]. In contrast, epigenetic marks induced by developmental or environmental cues to regulate gene expression appear to be mostly reprogrammed during reproduction to allow for proper development and physiology in the next generation. Nevertheless, it is becoming increasingly recognized that environmentally-induced epigenetic marks acquired in parents can be transmitted to their offspring and may increase their fitness [12,60,61]. Recent research, exemplified by the detailed mechanistic studies of *FLC* in Arabidopsis, have shed new light on the mechanistic basis of epigenetic inheritance across generations, as well as how the establishment of epigenetic marks in early embryos can have long-lasting consequences on gene expression after embryogenesis.

Future investigations employing emerging technologies to profile epigenetic features genome-wide in diverse species under various conditions will yield insights into the mechanistic basis and physiological consequences of epigenetic inheritance in plants. The field is now ripe for the discovery of additional mechanisms that regulate both the inheritance of environmentally-induced epigenetic features and their installation in embryonic chromatin to prime future gene-regulatory events.

Declaration of competing interest

None.

Data availability

No data was used for the research described in the article.

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Similar to what was reported in reference 41, the authors found that H3K27me3 was reduced in sperm during gametogenesis. By using a more sensitive method to profile H3K27me3, they were also able to identify genes that had gained both H3K4me3 active and H3K27me3 repressive marks. These bivalently marked genes were further categorized into whether they were expressed or not in sperm, which correlated with their transcriptional activities before or after fertilization. The authors also identified genes in which H3K27me3 was lost in sperm such as FLC, and suggested that reduced H3K27me3 reduces the barrier for their transcriptional activation after fertilization.

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The authors found that the FLC locus mostly produces non-functional and truncated transcript isoforms during early Arabidopsis embryogenesis likely through the co-transcriptional activities of FCA. FRI counteracts FCA's activities to progressively promote the production of full-length transcripts during embryogenesis. Moreover, the establishment of transcriptionally active and mitotically stable epigenetic states forecast FLC's expression state much later in development.

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