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Epigenetic Regulation During Plant Development and the Capacity for Epigenetic Memory

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Abstract

The establishment, maintenance, and removal of epigenetic modifications provide an additional layer of regulation, beyond genetically encoded factors, by which plants can control developmental processes and adapt to the environment. Epigenetic inheritance, while historically referring to information not encoded in the DNA sequence that is inherited between generations, can also refer to epigenetic modifications that are maintained within an individual but are reset between generations. Both types of epigenetic inheritance occur in plants, and the functions and mechanisms distinguishing the two are of great interest to the field. Here, we discuss examples of epigenetic dynamics and maintenance during selected stages of growth and development and their functional consequences. Epigenetic states are also dynamic in response to stress, with consequences for transposable element regulation. How epigenetic resetting between generations occurs during normal development and in response to stress is an emerging area of research.

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INTRODUCTION

Epigenetic dynamics: differences in epigenetic state observed between generations, tissues, cells, or stages of development, depending on context

Epigenetic: heritable information not encoded in the DNA sequence

Intraindividual epigenetic inheritance: epigenetic modification is maintained for a period of development, but is reset between generations

Intergenerational epigenetic inheritance: stable passage of epigenetic modifications between generations

Plants undergo continuous development over their lifetime. This process involves the generation of new organs and tissues and key transitions from vegetative to reproductive growth. Most aspects of plant development, for example flowering and seed germination, are both genetically driven and highly responsive to environmental cues (23, 60). The extent and function of epigenetic dynamics throughout plant growth and development are major questions of interest.

The term epigenetic describes heritable information not encoded in DNA. By the strictest definition, heritability refers to information that is passed from parent to progeny through meiosis. However, in the context of epigenetic inheritance, the term has also been frequently adopted to refer to epigenetic information that is inherited from one cell division to the next within an individual. In this review, we consider both intraindividual and intergenerational epigenetic inheritance. For an epigenetic modification to be inherited across generations in plants (i.e., intergenerationally), it must be maintained through cell divisions in tissues from which a germline will be established. In this way, epigenetic information can be maintained over both developmental and generational timescales. Epigenetic information is also dynamic, with modifications being added and removed in distinct cell and tissue types in different developmental contexts, as well in response to certain environmental conditions. It has thus been useful to contrast static epigenetic states that are maintained faithfully over cell divisions with dynamic epigenetic modifications that are gained and lost in different conditions or at programmed points in development (88, 150). The ability of epigenetic information to be both static and dynamic on multiple timescales presents the possibility for the memory of a perceived stimulus, such as a stressor, to be passed on to progeny through heritable epigenetic modifications. Epigenetic transmission of a memory of a stressor to the next generation could benefit fitness if the next generation is likely to experience the same stress. However, such a memory could come with potential costs, for example, if the environment changes or a stress response is no longer needed. Therefore, a mechanism whereby a plant could

maintain an epigenetic modification through growth and development (i.e., intraindividually) but reset it between generations could be beneficial in some circumstances.

MECHANISMS FOR ESTABLISHING, MAINTAINING, AND REMOVING EPIGENETIC MODIFICATIONS

Multiple integrated, redundant, and self-reinforcing pathways and mechanisms contribute to a cell's or plant's epigenetic state. Different combinations of epigenetic pathway activity contribute to tissue-specific differences or dynamics. Here, we provide a brief overview of some of the key mechanisms that promote the stable maintenance of epigenetic states and those that affect epigenetic changes.

Transposable elements (TEs): DNA sequences that are or once were able to move around the genome; different categories of TEs are defined based on the mechanism of transposition

DNA Methylation

Addition of a methyl group to the 5′ carbon of cytosine, referred to as DNA methylation, is a common epigenetic feature of eukaryotic genomes. DNA methylation represses potentially harmful genomic elements, such as repeat sequences and transposable elements (TEs), and also transcriptionally regulates genes. DNA methylation in gene promoters typically represses gene expression, although notable exceptions exist (81, 151). In plants, 5-methylcytosine (5-mC) can be present in the CHH, CHG, and CG sequence contexts (where H is any base except a G). Co-occurrence of CG and non-CG methylation typically occurs in TEs and other repeat-derived or related sequences. CG-only methylation is found in some gene bodies, particularly those of longer genes with moderate expression (169). 5-mC is added de novo primarily through a small RNA (sRNA)-based pathway known as RNA-directed DNA methylation (RdDM) (32, 95). Canonically, this process begins by transcription of the target locus by the plant-specific RNA polymerase IV (Pol IV), producing RNAs that are converted to double-stranded RNAs (dsRNAs) by RNA-dependent RNA polymerase 2 (RDR2). These dsRNAs are cleaved primarily into 24-nucleotide (nt) sRNAs, but also 21- and 22-nt sRNAs, by Dicer-like proteins (DCL2 or DCL3) and loaded into an ARGONAUTE protein (AGO4 or AGO6) (32, 105). This complex then directs de novo DNA methylation by the methyltransferase DRM2 through association with transcripts being produced by another plant-specific RNA polymerase, Pol V. Variations on the RdDM pathway may contribute to unique targeting. The CLASSY family of chromatin remodelers facilitate Pol IV localization and have recently been shown to contribute to tissue-specific DNA methylation patterns (89, 166). RdDM is crucial for asymmetric CHH methylation establishment and maintenance, especially in euchromatic regions (160, 164). RNA polymerase II (Pol II)–dependent transcripts, such as those transcribed from inverted repeats, can direct DNA methylation through noncanonical RdDM as well (25). The maintenance methyltransferase *MET1* maintains DNA methylation in the symmetric CG context. MET1 is recruited to the DNA replication fork by VIM proteins and methylates the newly synthesized DNA strand based on the pattern of CG methylation on the parent strand (71, 153). *CMT2* and *CMT3* encode chromodomain-containing methyltransferases and contribute to the maintenance of DNA methylation in the CHH and CHG contexts (43, 85, 136). Genes involved in maintenance methylation are more highly expressed in cells that are dividing and are repressed in mature leaf tissue by *TCX5* and *TCX6*, which are components of the DREAM complex, thus preventing DNA hypermethylation in this tissue (100).

Active DNA demethylation in plants is accomplished by a family of 5-mC DNA glycosylases that remove 5-mC by initiating base excision repair. The four *Arabidopsis* DNA demethylases are DME, ROS1, DML2, and DML3. While the genes are partially redundant, they have distinct target loci and expression patterns (85, 104, 110, 130, 149). *DME* expression in the central cell

of the female gametophyte is essential during reproductive development for seed viability (21). In rice, four DNA glycosylases have been identified, three of which have been shown to demethylate DNA at partially distinct target sites (72, 167). Maize homologs of *DME—ZmROS1a* and *ZmROS1b/MDR1*—have been shown to prevent DNA hypermethylation at specific target sites in endosperm (42, 157). Maize plants that are mutant for both *DNG102*, another maize *DME* homolog, and *ZmROS1b/MDR1* fail to make viable seed (42). DNA glycosylases are typically thought to identify and excise DNA lesions by scanning the DNA (61). Several studies in *Arabidopsis* have suggested a role for histone interactions in targeting 5-mC DNA glycosylases. The histone acetyltransferase IDM1 promotes ROS1-mediated DNA demethylation at many loci, and correspondingly many TEs targeted by ROS1 are enriched in H3K18Ac and related histone modifications (118, 139). DME activity is facilitated by association with histone H1 as well as chromatin remodeling by the FACT complex (54, 122). Shared target sites of DME and FACT are enriched in heterochromatin markers H3K9me2 and H3K27me1 (38).

Heterochromatin: silenced regions of DNA, molecularly characterized by the prevalence of repressive histone marks and DNA methylation

Histone Modifications and Variants

In eukaryotes, DNA is wrapped around histone protein octamers, and interactions between histones and other proteins package the DNA into chromatin. Histone tails can be modified, which alters chromatin state and can make DNA more or less accessible for transcription and other processes. There are many enzymes that add and remove histone modifications; more comprehensive reviews can be found elsewhere (18, 27, 112). Here, we focus primarily on histone 3 lysine residue methylation.

Methylation of H3K9 and H3K27 is associated with gene silencing and constitutive and facultative heterochromatin. The histone methyltransferase KRYPTONITE (KYP) methylates histone 3 lysine 9 (H3K9) and recognizes non-CG DNA methylation as target sites for methylating histones (29). The CMT2 and CMT3 maintenance methyltransferases in turn bind H3K9 and use it as a mark for where to methylate DNA. This feedback loop helps maintain the heterochromatic state, especially in pericentromeric regions (29, 30). Expression of the H3K9 demethylase *IBM1* is regulated epigenetically, by both H3K9me2 and DNA methylation within an intron of the gene (124). One of the plant PcG protein complexes, Polycomb Repressive Complex 2 (PRC2), catalyzes H3 lysine 27 methylation. In *Arabidopsis*, CLF, SWN, and MEA are H3K27 methyltransferases that are included in distinct PRC2 complexes and play important roles in regulating key developmental transitions, as has been reviewed extensively elsewhere (51). Additionally, PRC1 catalyzes monoubiquitination of histone H2A, contributing to transcriptional silencing both independently and at PRC2 target sites (51). H3K27 methylation is removed by Jumonji-type histone demethylases. In this review, we refer specifically to ELF6 and REF6, two of the H3K27 demethylases that have been shown to affect *Arabidopsis* development (24, 90). Binding of the H3K27me3 demethylase REF6 to DNA is inhibited by DNA methylation (119).

Briefly, there are other modifications that can affect transcription and chromatin state. H3K4 and H3K36 methylation has been shown to either promote or repress transcription, depending on how many methyl groups are added (156, 161). Histone acetylation, deposited by histone acetyltransferases, usually marks active chromatin and is found near transcribed regions (112).

Variants of the core histones can be found in different regions of chromatin, different stages of development, and cells that are in different stages of the cell cycle (137). Some H3 variants cannot be efficiently modified by PRC2, contributing to the remodeling of chromatin state during specific stages of development (8, 158). Histone variant H1 restricts RdDM activity from heterochromatic regions, highlighting another example of the interwovenness of different epigenetic pathways (20).

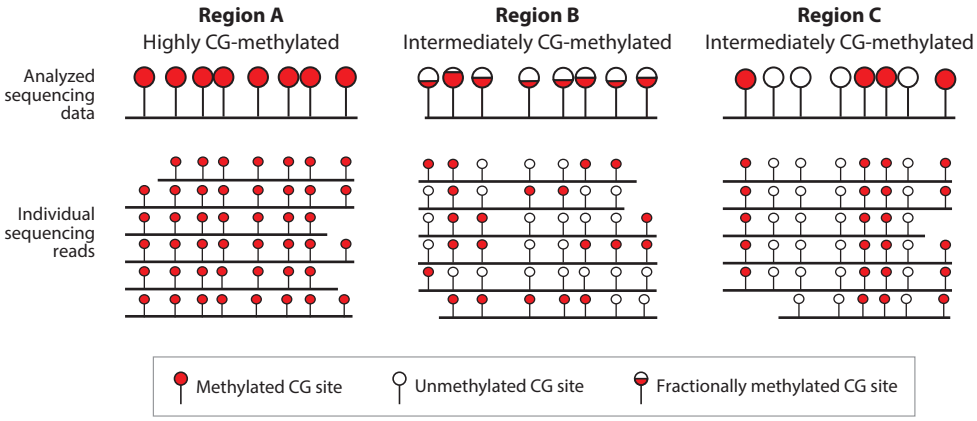

Figure 1

Interpreting intermediately CG-methylated regions. The stability of DNA methylation patterning shows some dependency on the surrounding genomic and epigenomic context. Depending on the parameters of the study, genomic regions are defined and levels of cytosine methylation are averaged across each region. All cytosines in region A are methylated across all sequencing reads; this region is highly methylated. In region B, all cytosines are fractionally methylated: In some reads the cytosine is methylated, and in others it is not. This region would likely be designated as intermediately methylated, depending on how intermediate is defined. Cytosines in region C are either completely methylated or completely unmethylated, as indicated by the supporting sequencing data. If a strict average of methylation across this region was taken, this region would also be defined as intermediately methylated.

Genomic Context and DNA Methylation Stability

Methylation in the CG context is most abundant in plant genomes relative to methylation in other sequence contexts and, as previously discussed, is maintained by MET1. However, recent work has added complexity to our understanding of the inheritance of CG methylation in plants. For most bulk methylation sequencing studies, the methylation status of a given cytosine is calculated as the percentage of total sequencing reads that indicate that the cytosine is methylated. Averaging these values across a defined number of base pairs represents the methylation level of a given region, although the exact methods vary depending on the study. If most CG pairs in a region are highly methylated, that region would be considered highly CG methylated (**Figure 1**, region A). If most CGs in a region are intermediately methylated (**Figure 1**, region B) or there is a mixture of more highly methylated and more lowly methylated CG pairs in a region (**Figure 1**, region C), this region would be classified as intermediately methylated, depending on the parameters of the study. Note that the two types of intermediate methylation have distinct underlying features. Recent work suggests that intergenerational changes observed at regions of intermediate to low levels of CG methylation reflect the integrated activity of different DNA methylation pathways.

Methylation of individual CGs within TEs and heterochromatic regions, which are usually highly methylated and accompanied by non-CG methylation, is maintained with greater fidelity over generations than CG methylation of protein-coding genes (6, 128, 145). Furthermore, changes in the CG methylation status of a region accumulate at a higher rate over generations than non-CG methylation status changes (28). Correlating changes in region-level CG methylation over generations with available chromatin state annotation data demonstrated that the stability of CG methylation status is correlated with genome annotation and observed chromatin structure of the same region in other experiments (28, 47). Hotspots for dynamic CG methylation

status are found most prevalently in regions with gene body methylation (gbM) and also contain regions that are intermediately CG methylated, here defined as regions with an average CG methylation level between 20% and 80% (47). In recombinant-inbred lines (RILs) between ecotypes of *Arabidopsis* that differ in their levels of gbM, sites that lost or gained methylation were more likely to occur in genic regions with intermediate to low levels of CG methylation and low levels of non-CG methylation in the parent (113).

In mutants of all four *Arabidopsis* DNA demethylases (*drdd*), where *DME* expression is supplied in the central cell to avoid seed lethality, hypermethylation in leaves occurs in multiple sequence contexts (149). Most hypermethylation occurs in putative RdDM targets, but there are many additional targets that are hypermethylated in only the CG context. In wild type, these regions are largely intermediately methylated, defined as 5–50% methylation (149). Regions defined by Hazarika et al. (47) as hotspots for dynamic CG methylation also correlate fairly strongly with regions that are CG hypermethylated in a *drdd* mutant background, suggesting a role for DNA demethylases in the dynamic CG methylation status observed in these regions of the genome.

Recent work has suggested that when maintenance methylation activity is inhibited, such as in a double mutant of histone *H1* and the chromatin remodeler *DDM1*, de novo CG methylation by the RdDM pathway is important for intergenerational maintenance of resulting intermediate CG methylation levels in heterochromatic regions. These findings propose a previously unknown role of the RdDM pathway in preventing the degradation of CG methylation patterning in heterochromatin under some conditions (91).

Based on the available evidence, regions with intermediate levels of CG methylation as measured by bulk sequencing methods represent regions of dynamic DNA methylation and demethylation over generations. These regions are enriched in protein-coding gene bodies and depleted in TEs and heterochromatin, where additional pathways may be stabilizing CG methylation. How the opposing effects of DNA methylation and demethylation pathways can affect the stability of CG methylation at these regions during development and over generational time will be an interesting future area of research.

EPIGENETIC REGULATION IN THE CONTEXT OF PLANT DEVELOPMENT

The extent and function of epigenetic dynamics throughout development and during developmental transitions have been perennial questions of interest. DNA methylation patterning and chromatin state are highly heritable between generations, suggesting that epigenetic state is maintained through the population of cells that contribute to reproduction, perhaps in combination with genetic encoding of targeting mechanisms. Wild-type siblings segregated from some DNA methylation mutants do not regain the original methylation state, implying the need for methylation state to be inherited rather than being strictly genetically targeted (36, 66, 123, 141). Distinct epigenomic patterns in different tissues and at different stages point to the occurrence of epigenetic dynamics through growth and development even when direct developmental phenotypes are not observed in epigenetic mutants (88). Understanding the mechanisms that underlie epigenetic dynamics as well as those that support intraindividual epigenetic memory will provide a more complete understanding of any roles of epigenetics in development and how targeting of epigenetic modifications occurs. In this section, we highlight some examples of epigenetic dynamics through development, focusing on tissues that would promote intergenerational epigenetic memory. Examples of epigenetic regulation of developmental processes are also discussed.

Potential for Transmission of Epigenetic Memory Through Stem Cell Populations

Pluripotent: the ability of a cell or tissue to form many different cell types through differentiation

Stem cells are defined by their ability to both replenish stem cell populations and generate other cell types, contributing to differentiated tissue development. In plants, stem cells reside in meristems. Primary apical meristems arise during embryogenesis and contain the initial stem cell populations in the root and shoot, providing differentiated cells for upward and downward growth of the plant. All reproductive cell types of the plant arise from shoot apical meristem (SAM)-derived tissues. Under the proper environmental and developmental conditions, the vegetative SAM will transition to an inflorescence meristem, which will then give rise to floral meristems. The current understanding of molecular mechanisms establishing and maintaining the SAM is mostly from work in *Arabidopsis*, where it is maintained by feedback loops with the transcription factor WUSCHEL (WUS) and the regulatory peptide CLAVATA3 (CLV3) (33, 144). The central zone of the meristem contains the pluripotent stem cell niche. Surrounding regions regulate the proliferation of stem cells into peripheral tissues. Spatially and temporally restricted gene expression is critical for proper SAM and floral meristem development, as well as downstream cell patterning. Important regulators of flowering time and flower development are targets of the PRC2 complex in *Arabidopsis* seedlings (12, 22, 23, 155). Often this regulation involves transcription factors. For example, chromatin remodeling ATPases, directed by the pioneer transcription factor LEAFY, counteract silencing by the CLF-PRC2 complex and promote proper floral patterning (64, 155).

The SAM includes cells that will ultimately be incorporated into the plant germline, meaning that the genomic integrity of these proliferating cells is likely critical. Additionally, this means that for both intraindividual and intergenerational epigenetic memory to exist, epigenetic information should be maintained through the SAM (**Figure 2**).

Data from several plant species suggest that genes involved in epigenetic silencing pathways are more highly expressed in shoot meristem tissue relative to various nonmeristem tissues (5, 103, 127, 152). This may in part simply reflect the fact that the meristem is enriched in dividing cells compared to nonmeristematic tissue. Despite the upregulation of epigenetic silencing pathways in the meristem, TEs are expressed and even upregulated in shoot meristems of maize, *Arabidopsis*, and rice (45, 87, 103, 138). In maize, most TEs are repressed by RdDM activity in the SAM, but not all, and some TEs even display RdDM-dependent expression (63, 103). One hypothesis to explain these seemingly opposing findings is that TE expression in some cells promotes silencing of TEs in cells that are fated for germline incorporation (94). Another nonexclusive hypothesis is that expression of TEs in the SAM is a consequence of host–TE dynamics. TEs may have evolved mechanisms to evade silencing in the meristem and thereby promote their transmission to the next generation.

Stem cells in the *Arabidopsis* SAM express *CLV3*, while nonstem cells do not. Gutzat et al. (45) took advantage of this specificity to observe epigenetic and transcriptional differences between stem cells (*CLV3*+) and nonstem cells (*CLV3*−) in the *Arabidopsis* SAM at vegetative and reproductive stages of development. TE expression is increased in *CLV3*+ cells relative to *CLV3*− cells, which correlates with DNA methylation in the CHG context during early vegetative development. These data do not support the model that TE silencing in stem cells is required for genome integrity. In fact, TE expression, whether indicative of host programming or TE escape of host silencing, is occurring in stem cells. In rice whole-SAM samples, CHH methylation at the ends of genes and TEs is slightly higher in the reproductive SAM than in the vegetative SAM or leaf tissue (50). Altogether, DNA methylation is dynamic in shoot meristems, but the effect of DNA methylation on TE and gene expression in this tissue is unclear. Future work with techniques such as single-cell methylome profiling in the SAM could clarify (*a*) the role, if any, of dynamic

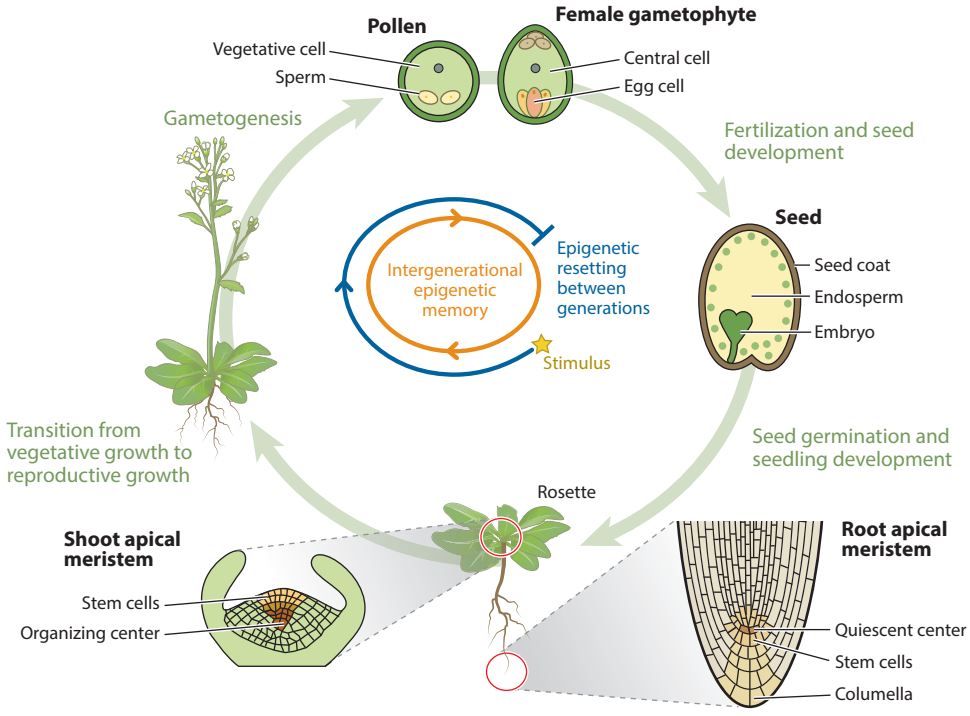

Figure 2

Epigenetic memory on different timescales. For epigenetic states to be maintained through generations, the relevant information needs to be transmitted through all relevant developmental phases, depicted here. Resetting of an epigenetic state between generations occurs in most instances of epigenetic response to a stimulus, an example being temperature stimulus and *FLOWERING LOCUS C* (*FLC*) silencing. Figure prepared by Jen Cook-Chrysos.

DNA methylation in the SAM; (*b*) if DNA methylation patterning in the differentiating cells of the SAM is reflective of the fated mature cell types; and (*c*) how epigenetic information is maintained intraindividually through developmental trajectories in the SAM, as well as in reproductive meristems.

Epigenetic Dynamics Through the Germline

For epigenetic information to be transmitted intergenerationally, cells that are incorporated into the germline should maintain the relevant information. In plants, there are losses and gains of epigenetic marks throughout reproductive development, but there is no global erasure of DNA methylation patterning as in animals (40). Additionally, the germline in plants is not established during early development. Instead, cells are specified from ovule primordium and inside anthers to undergo meiosis after the transition to flowering occurs. Ultimately, these cells derive from the SAM (**Figure 2**).

The female germline begins to form when the megaspore mother cell (MMC) undergoes meiosis. Heterochromatin is decondensed during specification of the MMC (131, 132). In *Arabidopsis*, CHH methylation decreases prior to MMC specification but is subsequently restored (56). During male germline formation, the microspore mother cell (MiMC) has high levels of CG and CHG methylation and low CHH methylation, along with unusual regions of local CHH

hypermethylation in genes (146). A mature pollen grain contains two haploid sperm nuclei and a diploid vegetative cell, which will develop into a pollen tube to reach the ovule for fertilization. These sister nuclei have distinct epigenetic states. *DME* and *ROS1* are expressed and active in the vegetative nucleus (VN) of the male gametophyte (pollen grain) but are not detected in sperm, and *DDM1* and *MET1* are expressed in the sperm but not the VN (10, 129). Together these enzymes are responsible for the relative CG hypomethylation of the VN (15, 53). Sperm are CHH hypomethylated relative to the VN (52). Similar methylation dynamics between sperm and the VN have also been observed in rice, suggesting conservation of mechanism (72). Loss of H3K9me2 occurs in the VN, including in pericentromeric regions, and many of these regions of open chromatin are also targets of DME, specifically near protein-coding genes (9). While there is currently no evidence for global erasure of DNA methylation during germline formation, and in fact DNA methylation is reinforced in the male germline (141), there are data suggesting that histone modifications are partially reprogrammed. During sperm differentiation, H3K27me3 is both removed through active demethylation and lost through replacement of histone H3 with the sperm-specific variant H3.10, which is not effectively modified by PRC2 (8).

The movement of noncoding sRNAs between cell types during gametogenesis, and any resulting effects on DNA methylation and transcription, has been debated. Evidence is emerging that sRNAs expressed in the tapetum, a secretory tissue of the anther that supports pollen development, affect DNA methylation in meiocytes. In *Arabidopsis*, Pol IV–derived 24-nt sRNAs are expressed from transposons in the tapetal cells. These small interfering RNAs (siRNAs) accumulate in the meiocytes, directing DNA methylation at homologous TEs as well as at genes with similar sequences (89). Supporting this model, deletion of the TEs from which the siRNAs are expressed results in loss of DNA methylation at the corresponding genes in male meiocytes (89). A similar mechanism seems to be occurring in maize, where Pol II–derived 24-nt sRNAs produced in maize tapetal cells accumulate in meiotic cells, suggesting that transport between cells may be occurring (168).

Epigenetic Dynamics in Seed Development

Seeds are the product of plant reproduction, from which progeny will continue to develop. A typical angiosperm seed is composed of an embryo and a nutritive tissue called the endosperm, both of which are encased in the maternally derived seed coat (**Figure 2**). Both the embryo and endosperm are products of fertilization. The diploid central cell is fertilized by a sperm cell to ultimately form the triploid endosperm common to most flowering plants, while the other sperm fertilizes the egg cell to form the zygote, which develops through embryogenesis.

Embryogenesis. Embryogenesis is a critical stage in the life cycle when the SAM and root apical meristem are established. Early stages of embryogenesis are characterized by rapid cell division; thus, rapid maintenance or reinforcement of DNA methylation states requires active methyltransferase pathways. DNA methylation, although dynamic during embryogenesis, is highly heritable from one generation to the next. CHG and CHH methylation levels increase over early embryogenesis, while CG methylation levels are overall stably maintained over this same period but increased in pericentromeric heterochromatic TEs (11, 67, 83, 107). The RdDM pathway is active during seed development in *Arabidopsis*, soybean, and chickpeas, and likely contributes to these dynamics (1, 11, 67, 121). *Arabidopsis* embryos produce high levels of 24-nt sRNAs (31, 106) that reinforce TE methylation and silencing (106). As an example of RdDM activity during embryogenesis, de novo methylation of an unmethylated, naive *FWA* promoter transgene, which contains direct repeats, occurs in early embryogenesis and is also dependent on *MET1* (65). Indeed, *MET1*, accessory proteins, and *CMT3* are highly expressed in early embryos (106, 107). CHG methylation

is increased in flower buds and embryos compared to leaves but decreases in late embryogenesis, subsequent to miRNA-driven cleavage of CMT3 transcripts (107, 117, 149). If repression of CMT3 in mid-embryogenesis is prevented, protein-coding genes become ectopically hypermethylated. This embryonic hypermethylation persists to at least 3 weeks after seed germination, highlighting the capacity for embryonic events, once initiated, to be maintained at later stages of the life cycle and the need to tightly control the expression of methylation machinery (107). After seed germination, CHH methylation levels in particular decrease (11, 67).

Endosperm and gene imprinting. The other product of fertilization, the endosperm, is a nutritive tissue that provides resources for the embryo and regulates seed germination, among other functions. The endosperm is the site of gene imprinting, or biased expression of either the maternal allele [a maternally expressed imprinted gene (MEG)] or the paternal allele [a paternally expressed imprinted gene (PEG)] (40). Understanding the mechanisms of gene imprinting is informative for understanding how developmentally programmed epigenetic modifications are implemented and maintained. Gene imprinting is established, at least in part, by differential epigenetic patterning initiated in the gametes before fertilization, some of which has already been described. The 5-mC DNA glycosylase DME is active in the central cell, and a subset of imprinted genes is associated with DME-dependent hypomethylation on maternally inherited alleles in the endosperm (115). Homologs of *DME* in rice and maize have also been implicated in gene imprinting (108, 125, 157). Both MEGs and PEGs are associated with DME-dependent hypomethylation of the maternal allele. Thus, for PEGs, DNA methylation actually promotes transcription (93, 116). Maternal allele silencing of PEGs is maintained by PRC2-directed H3K27 methylation and CHG methylation on the maternal allele (75, 99). Members of the PRC2 complex are themselves targets of DME and are MEGs in *Arabidopsis*, rice, and maize (19, 26, 41). There are now several examples of imprinted genes regulating other imprinted genes, highlighting the dynamic, layered transcriptional network created by epigenetic regulation of transcription factors and epigenetic regulators (4). A recent study has shown that imprinting is heterogeneous among different endosperm domains, suggesting that distinct epigenetic dynamics may occur among domains after fertilization (114). Future research in both *Arabidopsis* and other species could uncover how region-specific and timepoint-specific mechanisms of imprinting in the endosperm are established and maintained. Additionally, understanding how genes become imprinted de novo could provide insight into mechanisms of stable but developmentally transient epigenetic changes.

Intraindividual Maintenance of Silencing

Detailed studies on the control of flowering time in *Arabidopsis* have illustrated the need for a balance between intraindividual epigenetic memory and intergenerational epigenetic erasure (23, 96). A critical decision point in plant development is the transition from vegetative to reproductive growth. Flowering time regulation in *Arabidopsis* is centered around the activity of the MADS-box transcription factor *FLOWERING LOCUS C* (*FLC*), whose expression inhibits flowering. In vernalization-sensitive strains of *Arabidopsis*, prolonged cold exposure or, more precisely, the absence of warmth, represses *FLC* expression (162). *FLC* remains stably repressed upon the return to warmth, and plants are licensed to flower. *FLC* silencing occurs in multiple phases: The initial silencing of *FLC* is achieved through different mechanisms than the stable maintenance of the silent state. *FLC* is initially transcriptionally silenced through the activity of sense and antisense noncoding RNAs at *FLC* that are induced by cold temperatures (163). Epigenetic switching is then driven by H3K27me3 deposition at a short 3-nucleosome region downstream of the transcriptional start site, a process referred to as nucleation. The fraction of *FLC* copies that are nucleated is dependent on the length of cold exposure (7, 96). Nucleation requires the transcriptional repressor VAL1,

which interacts with protein complexes ASAP and PRC1 (97, 120). After the return to warmth, VELs, which are Polycomb accessory proteins, facilitate H3K27me3 deposition by PRC2 beyond the nucleation site. This causes long-term stable *FLC* silencing, which is maintained over cell divisions through the reproductive life of the plant (69, 159). *FLC* regulation is an example of a digital epigenetic switch: The switch from transcriptionally active to silent happens for each allele in each cell in a stochastic, all-or-nothing, cell-autonomous manner after the initial signal (2, 7).

Dedifferentiation: increasing the pluripotency of a cell or tissue

Because each generation of plant must flower at the right time given its environmental conditions, the epigenetic status of *FLC* must be reset, or forgotten, each generation. There is evidence that this occurs during embryogenesis and is mediated by the H3K27me3 demethylase ELF6 and the embryonic transcription factor LEC1, although this process is less understood than the mechanisms that establish and maintain silencing (23, 140).

The *FLC* example provides an interesting model for how a gene could be stably silenced epigenetically through a low-probability off switch but reset between generations (**Figure 2**). Other examples of PRC2 targets that are silenced through a similar mechanism have been identified (96). Future work applying what has been learned through the study of the *FLC* epigenetic switch to other genes will be valuable in understanding why some epigenetic modifications become heritable from parent to progeny while others are reset.

Epigenetic Dynamics Through Callus and Regeneration

Plants have a significant capacity to regenerate tissue. After wounding or in vitro, mature tissue can be induced to develop a pluripotent tissue known as callus (33, 35, 55). Agricultural research and transgenics rely heavily on the capacity of mature plant tissue to form callus, which can be induced to generate root and shoot tissue using different ratios of plant hormones. Additionally, cultured plant tissue can be induced to undergo somatic embryogenesis (35).

Alterations to histone modifications have been shown to play important roles in callus formation and regeneration in response to wounding as well as in culture. Many genes that are induced in response to wounding are marked by histone acetylation, and disruption of histone acetyltransferase activity inhibits callus formation after wounding (126). Decreased levels of H3K27me3 caused by the displacement of histone H3 have been observed in response to wounding in roots and hypocotyls, promoting callus formation (158). Mutation of PRC2 components results in the dedifferentiation of seedling tissue and abnormal developmental phenotypes (12, 17, 74). In vitro, PRC2 is required for callus production from leaf. H3K27me3 levels at a subset of transcription factors and metabolism-related genes are altered in leaf-derived callus relative to leaf tissue (48). Histone demethylases have also been shown to regulate callus formation and shoot regeneration. For example, JMJ30 promotes callus formation from leaf by removing H3K9me3 from genes that drive root primordia identity (79). *LDL3* is expressed in meristems and lateral root primordia and promotes shoot regeneration from callus in *Arabidopsis* by removing H3K4me2 from some shoot-promoting genes, priming those genes for later activation (57).

DNA methylation changes have been observed as a result of culturing plant tissue. The tissue used to generate the culture, as well as culturing methods, likely has an effect on the epigenome (147). In maize and rice callus, CHH methylation levels are generally increased, while CG and CHG methylation levels are generally reduced, relative to leaf tissue (46, 135). In rice, this loss of DNA methylation is maintained in the regenerated plant and is largely heritable through sexual reproduction over multiple generations (135). CHH hypermethylation also occurs during somatic embryogenesis from immature cotyledons in soybean, likely through the upregulation of RdDM components (62). In *Arabidopsis*, changes in DNA methylation generated through somatic embryogenesis are also heritably transmitted through subsequent generations of sexual reproduction (147).

Global DNA methylation changes impact tissue dedifferentiation and shoot regeneration in *Arabidopsis*. Callus with a mutation in *MET1* undergoes uninduced shoot formation more rapidly (82). Molecular evidence indicates that *MET1* is a negative regulator of *WUS* expression in callus, acting to inhibit shoot regeneration (82). Roots derived from *drm1 drm2 cmt3* triple mutants (*ddc*) and *cmt3* mutants are able to regenerate shoots directly from lateral root meristems when placed on shoot-inducing media (133). Loss of *DME* promotes callus formation, but suppresses de novo shoot formation from callus (73). Overall, the available data suggest that DNA methylation maintenance pathways may repress de novo shoot regeneration from callus while DNA demethylation induces de novo shoot regeneration.

Roles of DNA Methylation During Development

Different tissues have unique DNA methylation and transcriptional profiles, and different combinations of epigenetic modifiers and pathways likely contribute to some of these observed differences. Given the inextricable role of gene expression in the differentiation of cell types, it is reasonable to hypothesize that DNA methylation could indirectly contribute to cell differentiation by impacting gene expression. Some examples of this have already been discussed. Overall, there are few examples of morphological phenotypes associated with loss of a single DNA methylation or demethylation gene or pathway in *Arabidopsis*. Notable exceptions include *MET1* and *DME*, which have consequences for plant development and fertility (36, 66, 70, 73, 129). Recently, researchers have generated *Arabidopsis* mutants where effectively all DNA methylation or demethylation activity is eliminated. In a *met1 drm1 drm2 cmt3 cmt2* (*mddcc*) quintuple mutant, all DNA methylation is lost (49), leading to aberrant gene and TE expression. Mutant *mddcc* seedlings are much smaller than wild type and fail to flower, although whether this is caused by a dysregulation of flowering pathways or a complete failure of the shoot meristematic tissue is unclear. Root development is impacted as well, with root apical meristem cells appearing disorganized and root growth severely stunted (49). The quadruple mutant of all four DNA demethylases, *drdd*, flowers early (149). This is likely due to a decrease in *FLC* expression in the *drdd* background at the seedling stage and is correlated with hypermethylation of tandem repeats upstream of *FLC* (149).

While there are well characterized DNA methylation differences between cell or tissue types, loss or gain of DNA methylation at a scale less drastic than that induced by mutations in all methyltransferases or demethylases does not have clear consequences for cell and tissue differentiation. For example, through fluorescence-activated cell sorting (FACS) of the root tip and bisulfite sequencing of DNA from different cell types, researchers discovered that columella cells are DNA hypermethylated, especially in the CHH context, relative to both surrounding root cells and all other profiled cell types in *Arabidopsis* (68). The increased CHH methylation is likely established through RdDM and found predominantly in genes overlapping with TEs (68). The role of this remarkable CHH hypermethylation in columella is unclear, and columella identity does not depend on RdDM.

EVIDENCE (OR LACK OF EVIDENCE) FOR EPIGENETIC RESPONSE TO ABIOTIC STRESSORS ON MULTIPLE TIMESCALES

The inherent potential for plasticity of an epigenome has brought up questions of whether and how the epigenome can allow for more rapid response to environmental stressors and ultimately even adaptation on a shorter timescale than genetic mutation may allow (88).

Evidence is accumulating for epigenetic and regulatory RNA-mediated mechanisms promoting male fertility in flowering plants, particularly under unfavorable conditions. In maize and rice,

pathways that promote the production of phased, secondary small interfering RNAs (phasiRNAs) appear to buffer male fertility in the face of restrictive temperatures, as well as lengthening the photoperiod in rice (34, 80, 142, 165). Cleavage of phasiRNA precursors is dependent on miRNAs, and loss of miR2118, a miRNA that is responsible for cleavage of the phasiRNA precursor in rice that prevents photoperiod-restrictive male sterility, also results in the phenotype (3). Additional investigation indicates that miR2118-mediated 24-nt phasiRNA production promotes cell wall and anther wall development (3). In cotton, chemically induced loss of DNA methylation causes microspore sterility under high-heat conditions (92). Epigenetic strategies employed by plants to support fertility under high-heat conditions are of considerable interest in the face of climate change.

Changes to the transcriptome and epigenome that are induced by stress conditions are known (76, 98, 102, 148); however, the timescale on which these changes happen is an important consideration for future research. What molecularly distinguishes an intragenerational memory of a repeated stress that may allow an individual plant to tolerate this stress more robustly versus a response that is inherited through limited generations?

The initial response of a plant to increased temperature is regulated by many different pathways, both epigenetic and through transcription factors (102, 111). The transcriptional response to heat stress has been well characterized. Heat shock factors are critical for transcriptional changes in response to heat stress, with key factors being the HSFA1s and HSFA2 (102). The expression of some loci is maintained at a high level for hours or even days after the initial stress event, allowing for a more robust response to future stress events in that individual plant (102). *FORGETTER1* (*FGT1*) has been identified as a key gene for maintaining somatic memory of heat stress by associating with chromatin remodeling complexes and promoting open chromatin at other genes required for heat shock memory (13). The heat shock factor HSFA2 forms a heteromeric protein complex with HSFA3, and together they are critical for promoting sustained transcription of other heat shock genes through direct activation and by directing deposition of the activating mark H3K4 methylation (37, 77). *FGT1* is required for sustained transcription of the HSFA2 target HSA32, suggesting cooperation between these pathways (13). HSFA2 also activates the H3K27 demethylase REF6, which then further promotes HSFA2 expression in a positive feedback loop after heat shock stimulus (86).

Despite known epigenetic dynamics under stress conditions, evidence for intergenerational memory of stress in the absence of the stressor is limited (88). If so-called transgenerational epigenetic memory of heat stress is mostly nonexistent, why is this so? What mechanisms prevent epigenetic changes induced by heat response from being stably inherited indefinitely? After heat stress exposure, many TEs and repetitive regions are transcriptionally upregulated (78, 109, 143). The regulation of heat-activated TEs could inform future studies of epigenetic regulation under heat stress conditions. Expression of the long terminal repeat (LTR) retrotransposon *ONSEN* is initiated by heat stress and counteracted by Pol IV and RdDM activity (16, 58) (**Figure 3**). Insertion of *ONSEN* can relay heat sensitivity to nearby genes (58). Importantly, the loss of Pol IV siRNAs and DNA methylation is not sufficient for *ONSEN* expression, but loss of CHH methylation seems to enhance the expression of *ONSEN* under heat stress (16, 58). During flower development, likely before gametogenesis has been initiated, transposition of *ONSEN* is restricted by Pol IV. This prevents the propagation of new *ONSEN* insertions in the progeny of heat-stressed plants (39, 58). *CMT3* promotes *ONSEN* expression under heat stress in young seedlings, while *CMT2* restricts *ONSEN* expression (101) (**Figure 3**). The body and ends of *ONSEN* are highly CHH methylated, and this methylation is primarily *CMT2* dependent, although RdDM is likely contributing at the ends. In a *CMT3* mutant, *ONSEN* is CHH hypermethylated, H3K9me2 is enriched, and tagged CMT2 protein is enriched at *ONSEN* (101). These studies raise interesting

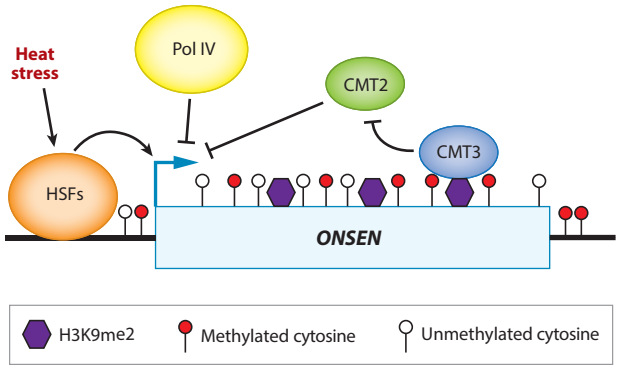

Figure 3

Model of *ONSEN* regulation in response to heat stress in *Arabidopsis*. *ONSEN* is a long terminal repeat (LTR) transcription factor family whose expression is induced by heat stress through heat shock transcription factors (HSFs). Expression and transposition of *ONSEN* are dampened under heat stress by RNA polymerase IV (Pol IV) (16, 58). CHH methylation of *ONSEN* is primarily *CMT2*-dependent and represses *ONSEN* expression (16). CMT3 recognizes H3K9me2 and CHG sites at *ONSEN* and noncanonically acts to promote expression by limiting CMT2 occupancy (101).

questions about potential noncanonical roles of *CMT3* and the mechanism by which, during development, *ONSEN* transposition is restricted by Pol IV activity to prevent inheritance of active *ONSEN* elements in heat stress conditions (**Figure 3**).

Mutator transposons in maize are well-characterized MULE superfamily transposons (84). Recent work on this transposon family has provided insight into the interplay between different epigenetic modifications under normal and stress growth conditions. Transposition of nonautonomous *Mu* elements can only occur in the presence of an active autonomous element, *MuDR*. *MuDR* encodes two genes, *mudrA* and *mudrB*; both are required for transposition. The terminal inverted repeats (TIRs) at the ends of *MuDR* act as promoters for these genes (84). *MuDR* is stably silenced in the presence of the *Mu killer* locus, which has sequence homology to *mudrA* (134). In a silenced *MuDR* background, the TIR for *mudrA* (TIRA) is methylated, and different regions of TIRA have been shown to become methylated through distinct small RNA-mediated mechanisms (14). Loss of *MOP1*, an *RDR2* homolog and key component of the maize RdDM pathway, can lead to the gradual loss of *mudrA* silencing over generations (154). This initial maintenance of *mudrA* silencing in a *mop1* mutant is not dependent on DNA methylation levels at TIRA (44). However, loss of *MOP1* does result in increased levels of H3K9me2 and H3K27me2 at TIRA and H3K27me3 at TIRB (44), suggesting an interaction between the loss of RdDM activity and gain of repressive histone modifications at the TIRs. Subjecting young *mop1* mutant seedlings that contain silenced *MuDR* elements to heat stress reactivates *mudrA* and *mudrB* transcription, corresponding with the loss of repressive histone methylation abundance at the TIRs. The heat-activated status of *MuDR* was heritable both intraindividually and to progeny over several generations in the absence of heat or the *mop1* mutation (44). These data suggest that histone methylation at the TIRs, supported in some way by MOP1 activity, is responsible for the silencing of *MuDR* under heat stress and restricting transmission of active *MuDR*.

These studies of TE activation and inheritance under heat stress highlight another potential benefit to resetting epigenetic responses to stress: Restricting the inheritance of heat-induced epigenetic changes may limit the risk of passing on potentially harmful active TEs to the progeny. In another study, chromatin remodelers *DDM1* and *MOM1* were observed to interact genetically

to prevent transgenerational memory of heat stress at a heat-responsive reporter transgene (59), further hinting at underlying mechanisms of heat response resetting.

SUMMARY POINTS

1. Patterning of epigenetic modifications can be heritable intergenerationally, but there are many instances of epigenetic modifications that are maintained intraindividually but reset between generations. How these two outcomes are mechanistically distinguished is not always clear.
2. For an epigenetic mark to be intergenerationally heritable, some form of memory of that mark needs to be maintained through stem cells of the shoot apical meristem, gametogenesis, and embryogenesis, as plants do not form a germline during early development. Intraindividual inheritance of epigenetic marks requires memory of the information through cell division and development but also a resetting of epigenetic information to prevent inheritance between generations.
3. The role of epigenetic regulation in development has been made clear for some regulators, such as Polycomb Repressive Complex 2 (PRC2), but not all epigenetic dynamics have or are likely to have developmental consequences.
4. Regions of intermediate CG methylation may represent regions that are enriched for DNA demethylation and de novo methylation activity and thus are subject to higher levels of epigenetic change over generations. These regions are enriched in protein-coding gene bodies but may reflect a larger phenomenon of mechanisms that buffer against stochastic loss of DNA methylation intergenerationally.
5. Heat-activated transposable elements (TEs) provide a valuable platform to study the role of epigenetic regulation in repressing TEs as well as mechanisms for preventing intergenerational memory of environmental stressors.

FUTURE ISSUES

1. When and where during development are epigenetic modifications that will be inherited between generations established?
2. What distinguishes an epigenetic mark that is established and reset within one individual plant from a heritable change to the epigenome?
3. What mechanisms underlie the epigenetic regulation of the maintenance of differentiated states during development? Are these mechanisms utilized or reversed in response to wounding?
4. How can we use and manipulate epigenetic processes to increase plant resiliency in the face of changing climates and the resulting loss of resources?

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