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## SI CHROMATIN AND DEVELOPMENT

# Transcriptional ‘memory’ of a stress: transient chromatin and memory (epigenetic) marks at stress-response genes

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## SUMMARY

Drought, salinity, extreme temperature variations, pathogen and herbivory attacks are recurring environmental stresses experienced by plants throughout their life. To survive repeated stresses, plants provide responses that may be different from their response during the first encounter with the stress. A different response to a similar stress represents the concept of ‘stress memory’. A coordinated reaction at the organismal, cellular and gene/genome levels is thought to increase survival chances by improving the plant’s tolerance/avoidance abilities. Ultimately, stress memory may provide a mechanism for acclimation and adaptation. At the molecular level, the concept of stress memory indicates that the mechanisms responsible for memory-type transcription during repeated stresses are not based on repetitive activation of the same response pathways activated by the first stress. Some recent advances in the search for transcription ‘memory factors’ are discussed with an emphasis on super-induced dehydration stress memory response genes in *Arabidopsis*.

**Keywords:** chromatin, epigenetics, transcriptional memory, memory genes, *Arabidopsis thaliana*, chromatin structure and transcription.

## INTRODUCTION

Eukaryotic genes function in the context of chromatin and it is the structure of chromatin that, ultimately, establishes permissive or restrictive conditions for the accessibility of transcribed sequences and passage of the transcriptional machinery. Altered chromatin structure often accompanies altered gene expression and it is thought that chromatin factors coordinately interact to establish optimal transcriptional output from the response genes. According to current models, chromatin remodelers, histone modifiers and DNA methylating/demethylating activities interact and influence each other’s performance, and their interactions are often mediated by both short and long non-coding RNAs (NcRNAs). These topics are actively researched and extensively reviewed (Castel and Martienssen, 2013; Baulcombe and Dean, 2014; Deinlein *et al.*, 2014; Han and Wagner, 2014; Zhao and Chen, 2014; Vriet *et al.*, 2015), and are not discussed in detail here. A comprehensive list of chromatin activities and the marks they establish at stress-response genes has been published (Van Oosten *et al.*, 2014).

Here, the focus is on the role of chromatin as a potential component in the ‘memory’ mechanism in responses to recurring stresses. The transcriptional behavior of genes that are induced by a dehydration stress but super-induced upon a subsequent stress is discussed as a model for ‘positive memory’ formation. Transcriptional stress memory has been actively studied in yeast and current models regarding the role of chromatin in yeast stress memory are also discussed briefly. Because chromatin structure provides an additional level of gene regulation, often referred to as ‘epigenetic’, the terms ‘epigenetics’ and ‘epigenetic marks’ are briefly discussed to clarify their use in our studies and applicability to stress-responding genes. The role of histone modifications in the initiation and elongation phases of transcription, as well as the similarities/differences between the priming of defense genes and the responses of a subset of dehydration stress-related genes to a repetitive stress, are discussed. Emerging evidence suggesting that the chromatin-modifying activities TrxG/H3K4me3 and PcG/H3K27me3, in particular, may play different roles at

stress-responding and developmentally regulated genes is presented. The duration of stress-induced memory as a potential factor in the adaptive mechanism of plants is also briefly considered.

### 'REMEMBERING' STRESS

Pre-exposing plants to various abiotic stresses, i.e. high-salt, mild or high temperature, cold, or water withdrawal, may cause altered responses to future stresses (Mittler *et al.*, 2012; Stief *et al.*, 2014; To and Kim, 2014; Wang *et al.*, 2014b). *Arabidopsis thaliana* and *Zea mays* (maize) plants that have been subjected to several dehydration/rehydration cycles displayed improved retention of water compared to plants experiencing a first stress (Ding *et al.*, 2012a, 2014; Virilouvet and Fromm, 2015). Pre-treatment with stress-signaling molecules [jasmonic acid (JA), salicylic acid (SA), or abscisic acid (ABA)] or pre-exposure to pathogens or herbivory resulted in an increased Systemic acquired resistance (SAR) and resistance to subsequent biotic stresses (Goh *et al.*, 2003; Jakab *et al.*, 2005; Conrath *et al.*, 2006; Conrad 2011; Bruce *et al.*, 2007; Rasmann *et al.*, 2012; Slaughter *et al.*, 2012; Bruce, 2014). Resistance to abiotic stresses was also improved after treatment with SA or  $\beta$ -aminobutyric acid (Zimmerli *et al.*, 2000; Jakab *et al.*, 2001).  $\beta$ -aminobutyric acid-treated *Arabidopsis* or SA-treated wheat plants (*Triticum aestivum*) displayed increased resistance to drought and high salinity (Shakirova *et al.*, 2003; Ton and Mauch-Mani, 2004; Jakab *et al.*, 2005); SA treatment differentially affected the chilling tolerance of maize, cucumber (*Cucumis sativus*) and rice (*Oryza sativa*) seedlings (Kang and Saltveit, 2002) and improved thermotolerance in *Arabidopsis* and in mustard (*Sinapis alba*) plants (Dat *et al.*, 1998; Clarke *et al.*, 2004). Mobilizing physiological, biochemical and molecular mechanisms to provide faster and/or stronger responses is thought to ensure enhanced protection without the costs associated with constitutive expression of stress-related genes (Van Hulten *et al.*, 2006). However, repeated stresses may result in increased sensitivity to deleterious effects (Soja *et al.*, 1997), down-regulated photosynthesis, or perturbed growth and development (Skirycz and Inzé, 2010).

Collectively, available evidence suggests that, after experiencing a stress, plants may modify their responses to a future stress, leading to the concept of 'stress memory' (Bruce *et al.*, 2007; Gális *et al.*, 2009; Ding *et al.*, 2012a). Stress memory may increase resistance to stress factors, as an adaptive mechanism, but may also compromise aspects of the plant's overall performance (Skirycz and Inzé, 2010).

In addition to memory responses at the organismal/cellular levels, referred to as 'physiological memory' (Ding *et al.*, 2014; Virilouvet and Fromm, 2015), dramatic changes in gene expression patterns may occur, illustrating the concept of 'transcriptional stress memory' and revealing

the existence of dehydration stress 'memory' genes (Ding *et al.*, 2012a).

### TRANSCRIPTIONAL MEMORY OF DEHYDRATION STRESS MEMORY GENES

The operational criterion used for transcriptional memory is that transcript levels from response genes in subsequent stresses (S2 and S3), after a recovery period from the first stress (R1), must be significantly different from the levels of transcripts produced during S1, despite a similar level and duration of the stress. Recovery is determined by the restoration of metabolic, transcriptional or protein levels to their pre-stress levels. Memory genes altered transcriptional responses to a subsequent stress, while 'non-memory' genes respond similarly to each stress. At the chromatin level, non-memory genes displayed dynamically changing H3K4me3 patterns correlating with the degree of transcription, while memory genes maintained increased H3K4me3 during the recovery phase, when transcription was low (Ding *et al.*, 2012a). Most importantly, these memory marks contributed significantly to future transcriptional responses.

Whole-genome transcriptome analysis of multiply stressed *Arabidopsis thaliana* plants revealed the existence of an unsuspected diversity of transcriptional response patterns (Ding *et al.*, 2013). Depending on the level of transcripts produced in subsequent stresses (S2/S3) compared to the levels in the first stress (S1), four distinct memory response patterns were recognized, suggesting a whole new level of complexity of transcription regulatory mechanisms. More than 2000 *Arabidopsis* genes showed memory responses. The existence of distinct transcriptional response types raised the question of whether memory patterns have biological relevance. Gene ontology (GO) analysis indicated a biased distribution of the memory types with respect to cellular/organismal functions, associated with four general strategies used by plants to improve stress tolerance and/or survival: (1) increased synthesis of protective, damage-repairing and detoxifying functions, (2) coordinating growth and photosynthesis under repetitive stress, (3) re-adjusting osmotic and ionic equilibrium to maintain homeostasis, and (4) re-adjusting interactions between dehydration and other stress-regulated pathways (Ding *et al.*, 2013).

Repeatedly stressed maize (*Zea mays*) plants displayed transcription memory responses similar to those of *A. thaliana* (Ding *et al.*, 2014). These results are important as they indicate evolutionary conservation of dehydration stress memory in eudicot and monocot plants. Evolutionarily conserved stress memory function was also suggested for *miR156*, which is implicated in memory responses to recurring heat stress in *Arabidopsis* (Stief *et al.*, 2014).

Transcriptional stress memory is therefore a biologically relevant mechanism that is conserved during evolution of land plants and regulates different responses to a single stress versus recurring stresses. The concept of transcriptional memory implies that there is a mechanism for storing the 'information' from a previous stress. Studies in animal and yeast systems have suggested that altered chromatin structure, integrating the effects of a signaling pathway with transcriptional responses, may provide such a mechanism (Suganuma and Workman, 2012, 2013; Badeaux and Shi, 2013; Johnson and Dent, 2013).

### CHROMATIN AND EPIGENETICS

Some authors have suggested that heritability of chromatin modifications during mitotic and meiotic divisions is a necessary prerequisite for defining an event as epigenetic (Eichten *et al.*, 2014). However, the Roadmap Epigenomics Project defined epigenetics as '...both heritable changes in gene activity and expression (in the progeny of cells or of individuals) and also stable, long-term alterations in the transcriptional potential of a cell that are not necessarily heritable' (<http://www.roadmapepigenomics.org/overview>). Within this broader definition, the terms 'epigenetics' and 'epigenetic marks' may be legitimately used when studying stress responses, as they often occur during vegetative periods and in tissues, such as leaves, that have ceased cell division.

While providing a useful conceptual framework for understanding transcriptional responses to stress, this broader definition blurs the distinction between the 'proactive' and 'responsive' roles of chromatin in the transcription of stress-related genes. The tight correlation between gene expression and chromatin structure forms the backbone of the current concept of epigenetics (Henikoff and Grosveld, 2013), but whether changes in chromatin structure induce, or simply reflect, established transcriptional states is still debatable (Henikoff and Shilatifard, 2011). Earlier, Bird (2007) suggested that '... epigenetic systems would not, under normal circumstances, initiate a change of state at a particular locus but would register a change already imposed by other events'. Therefore, epigenetic marks are viewed as 'responsive', not 'proactive', reflecting 'structural adaptation of chromosomal regions so as to register, signal, or perpetuate altered activity states' (Bird, 2007). It may be misleading, then, to refer to changes in chromatin structure occurring at transcriptionally active or silent sites as 'epigenetic', or as evidence that chromatin structure induces or represses transcription, in cases where causality is not established (Henikoff and Shilatifard, 2011).

### CHROMATIN (HISTONE) AND EPIGENETIC MARKS ARE NOT SYNONYMOUS

A gene's active/silent transcriptional state usually correlates with altered DNA cytosine methylation patterns, histone

modification levels, or a changed structure of associated nucleosomes. However, although linked, chromatin and epigenetic marks are not equivalent (Ding *et al.*, 2012a; Eichten *et al.*, 2014). We have suggested that a major distinction between 'chromatin' and 'epigenetic' marks is that chromatin marks reflect modifications that are dynamically associated with the state of a gene's transcription but are removed at its conclusion, while epigenetic marks persist after the initial stimulus that caused the chromatin mark is no longer present. Therefore, as an operational definition, a memory mark's duration does not have to be permanent but must exceed that of the original stimulus that established the mark. Most importantly, an epigenetic mark must have a significant effect on a future gene's transcriptional performance. Thus, histone modifications (i.e. H3K4me3) that are increased by stress-triggered transcription but then decrease when the signal is removed and transcription is restored to its baseline level, are transient chromatin marks; histone modifications retained at altered levels after removal of the signal are consistent with a function as 'epigenetic marks'. However, to define a modification as an epigenetic mark, it is necessary that the marks contribute to the subsequent transcription. Consequently, H3K4me3 accumulated at super-induced dehydration stress-response genes (Ding *et al.*, 2012a) or at primed defense-related genes before actual transcription (Jaskiewicz *et al.*, 2011) functions as an epigenetic mark. However, the low H3K4me3 levels remaining at a few dehydration stress-responding genes after a stress that did not affect their subsequent transcription (Kim *et al.*, 2012b) do not satisfy the criterion for an epigenetic mark.

Therefore, chromatin marks that are established or removed by a stimulus-activated gene network are defined as epigenetic if they remain after the stimulus is removed and influence the future transcriptional behavior of associated genes. Thus epigenetic systems may act as the conduit for environmental cues initiating short- or long-term changes in gene expression in response to stress.

### DEFENSE PRIMING AND TRANSCRIPTIONAL STRESS MEMORY: SIMILARITIES AND DIFFERENCES

Defense-related genes pre-treated with jasmonic acid, salicylic acid (SA) or its analog (benzo-(1,2,3)-thiadiazole-7-carbothioic acid S-methyl ester (BTH) display higher transcription upon subsequent attack (Conrath, 2011; Jaskiewicz *et al.*, 2011). This phenomenon, known as 'defense priming', is consistent with stress memory. Mechanistically, however, there are differences between the regulation of 'primed' defense genes and that of dehydration stress memory genes in multiply stressed plants. Based on the few available examples, one apparent difference is that, although pre-treatment with jasmonic acid/SA/BTH significantly increased transcription from some defense genes during subsequent attacks, the signals did not

directly induce, or only weakly activated, transcription from defense-related genes before the attack (Jaskiewicz *et al.*, 2011). In contrast, super-induced transcription of dehydration stress memory genes in a subsequent stress occurs only after active transcription during a previous exposure; moreover, the magnitude of the transcriptional response in the second stress depends strongly upon the degree of transcription that occurred in the first (Ding *et al.*, 2012a). H3K4me3 is a signature feature that functions as an epigenetic mark for both primed and dehydration stress memory genes (Jaskiewicz *et al.*, 2011; Ding *et al.*, 2012a). An important difference, however, is that elevated H3K4me3 is retained at dehydration stress memory genes during their low-transcription periods (i.e. during recovery after a stress) as a 'memory' of their previous active transcription; in contrast, the accumulation of H3K4me3 at the promoters of primed genes does not reflect a memory of earlier activity (Jaskiewicz *et al.*, 2011). The molecular mechanism that results in H3K4me3 accumulation in the absence of transcription at primed genes is unknown. In addition, a RNA polymerase II phosphorylated at serine 5 of its tail domain (Ser5P Pol II) retained (stalled) at dehydration stress memory genes during watered recovery functions also as an epigenetic (memory) mark as it is associated with super-induced transcription (Ding *et al.*, 2011a,b, 2012a). These data provided the first evidence of a stalled RNA polymerase in plants, and identified it as a factor in the memory transcription of dehydration stress-response space interval genes. Whether Ser5P Pol II accumulates at primed defense genes before their transcription is unknown.

Therefore, although both primed and dehydration stress memory genes 'remember' previous treatments and modify their responses to a subsequent stress, the molecular mechanisms regulating their 'memory transcription' may be different. To reveal these mechanisms and the players involved is a challenging task.

### MOLECULAR MECHANISMS OF TRANSCRIPTIONAL MEMORY

Sustained/accumulated levels of key signaling metabolites, plant hormones and proteins involved in their synthesis, or transcription factors and the kinases/phosphatases regulating their activity, have been considered potential 'memory factors' (Bruce 2014; Conrath, 2011; Santos *et al.*, 2011; Kinoshita and Seki, 2014; Vriet *et al.*, 2015).

A model whereby higher ABA levels retained from a previous stress may be responsible for the transcription memory is not supported by our data: endogenous ABA levels increase to the same extent during each dehydration stress but, nonetheless, memory-type genes produce different transcript amounts in the first stress and in subsequent stresses (Ding *et al.*, 2012a, 2013; Liu *et al.*, 2014a,b). Furthermore, although critically required for transcription of

the memory gene *RD29B*, ABA alone was insufficient to super-induce transcription in subsequent stresses (Virilvet *et al.*, 2014). Apparently, a dehydration-dependent ABA-independent factor of unknown nature that was not activated by ABA alone is required.

Transcription factors and kinases regulating their activity contribute to memory responses. The SPL transcription factors are critical for heat stress memory (Stief *et al.*, 2014), and heat shock factor HsfB1 was associated with SAR (Pick *et al.*, 2012); accumulation of inactive mitogen-activated protein kinases MPK3 and MPK6 and their mRNAs after SA/BTH treatments has been associated with the priming of defense-related genes (Beckers *et al.*, 2009). The transcription factor MYC2 was identified as the critical component that determines the memory behavior of a specific subset of MYC2-dependent genes (Liu *et al.*, 2014a). However, the transcription patterns of a transcription factor do not necessarily correlate with the memory patterns of dependent genes, even of directly regulated genes (Liu *et al.*, 2014a). For example, the MYC2 marker gene *RD22* (Yamaguchi-Shinozaki and Shinozaki, 1993; Boter *et al.*, 2004) depends on MYC2 for its transcription during the first stress but does not require MYC2 in S2 (Liu *et al.*, 2014a). Therefore, the expression of a transcription factor cannot predict the memory behavior of its targets. This further supports the idea that different molecular mechanisms (including different transcription factors) are involved in transcriptional responses during a single stress and when encountering repeated exposures to the stress.

Furthermore, the protein levels of three ABRE-binding factors (AREB1, AREB2 and ARF3), which regulate a large number of dehydration stress-response genes including the memory genes *RD29B* and *RAB18* (Yoshida *et al.*, 2010), did not change significantly during repeated dehydration stress exposures (Virilvet *et al.*, 2014). Nonetheless, transcription from their direct targets (*RD29B* and *RAB18*) dramatically increased in S2, excluding the possibility that accumulated ABRE-binding factors provide the mechanism for super-induced transcription. Moreover, the transcriptional memory was still functional in the absence of ABRE-binding factors despite strongly decreased transcription from *RD29B* and *RAB18* (to <1%) in a triple loss-of-function *areb1/areb2/arf3* mutant background. However, depletion of the kinases SnRK2.2/3/6 that activate the ABRE-binding factors (Fujita *et al.*, 2013) completely abrogated transcription in both S1 and S2, suggesting that an ABA-independent component (of unknown nature) works together with the ABA/SnRK2-dependent pathway in the memory response (Ding *et al.*, 2012a; Virilvet *et al.*, 2014).

### CHROMATIN STRUCTURE AS A POTENTIAL MEMORY FACTOR

The ability of chromatin to undergo both dynamic and stable changes in its structure in response to stress has been

considered a mechanism for stress memory propagation (Van Oosten *et al.*, 2014). Recent models propose that proteins involved in signaling pathways, i.e. mitogen-activated protein kinases, may transfer signals to chromatin/nucleosome structure through chromatin-modifying enzymes. Consequently, chromatin may act as memory 'storage' where 'signal transduction pathways converge upon sequence-specific DNA binding factors to reprogram gene expression' (Suganuma and Workman, 2012, 2013; Badeaux and Shi, 2013; Johnson and Dent, 2013).

However, chromatin-based mechanisms may be developmental stage-, tissue-, gene- and signal-specific (Bratzel *et al.*, 2010; Farrona *et al.*, 2011; Ismail *et al.*, 2014; Wang *et al.*, 2014b). Thus, different factors regulate site-specific DNA methylation (Stroud *et al.*, 2013), and histone modifications may play different roles in events that require a rapid gene-specific response to external stimuli compared to those at genes regulated by long-term developmental programs (Liu *et al.*, 2014b). Furthermore, histone-modifying enzymes may have non-histone substrates: the acetyltransferase activity of the elongator complex acetylates also  $\alpha$ -tubulin (Creppe and Buschbeck, 2011) and the SET domain of Arabidopsis Trithorax 1 (ATX1) specifically methylates Elongation Factor 1A, dramatically affecting cytoskeletal actin (Ndamukong *et al.*, 2011). The results suggest that the process involves signaling both to and from chromatin (Creppe and Buschbeck, 2011; Badeaux and Shi, 2013; Johnson and Dent, 2013). In addition, the signaling lipid phosphoinositide 5-phosphate, which accumulates in response to dehydration stress, affects the nuclear/cytoplasm distribution of the histone modifier ATX1. Phosphoinositide 5-phosphate and ATX1 co-regulate an overlapping set of genes that act as components of a pathway that translates an environmental signal into altered chromatin structure and expression of ATX1-dependent genes (Alvarez-Venegas *et al.*, 2006a,b; Ding *et al.*, 2009; Ndamukong *et al.*, 2010).

Collectively, available results suggest the roles of chromatin in transcriptional responses to stress are complex, and apparently gene-, stress signal- and species-specific, as briefly discussed below.

#### **CHROMATIN-MODIFYING ACTIVITIES MAY HAVE DIFFERENT ROLES AT STRESS-RESPONDING AND DEVELOPMENTALLY REGULATED GENES**

Chromatin factors are implicated in the convergence of stress-responding and developmentally regulated pathways (Kim *et al.*, 2012a; Jung *et al.*, 2013; Perrella *et al.*, 2013; Stief *et al.*, 2014; Wang *et al.*, 2014b). However, chromatin modifiers and the marks they establish may function differently at genes involved in responses to environmental or developmental cues (Weake and Workman, 2010; Liu *et al.*, 2014b). Thus, priming of C4 photosynthesis genes in maize for enhanced activation by light was also achieved

by developmental factors, but developmental and environmental signals induce distinct histone acetylation profiles on distal and proximal promoter elements of the C4 phosphoenolpyruvate carboxylase gene (Danker *et al.*, 2008; Offermann *et al.*, 2008). Whether the outcome of high-salinity stress is adaptation or cell death has been linked to the time at which the signals appear and disappear (Ismail *et al.*, 2014). The responses to low temperatures leading to cold acclimation or vernalization are controlled by distinct signaling pathways (Bond *et al.*, 2011), and the molecular mechanisms promoting transition to flowering under elevated ambient temperatures appear to be different from the effects induced by recurring heat stress (Stief *et al.*, 2014). The apparently different roles of H3K4me3 and H3K27me3 at developmentally or stress-regulated genes (Liu *et al.*, 2014a,b) are discussed in some detail below.

#### **TRXG/H3K4ME3 AND PCG/H3K27ME3 AT STRESS-RESPONDING GENES**

The counterbalancing roles of the Trithorax group (TrxG) and Polycomb group (PcG) proteins in propagating the memory of transcriptionally active/inactive states during ontogenesis in both animal and plant systems have been widely documented and reviewed (Saleh *et al.*, 2007; Avramova, 2009; Schuettengruber *et al.*, 2011; Molitor and Shen, 2013; Derkacheva and Hennig, 2014). However, the role of TrxG/PcG in a plant's responses to stress, is just emerging (Kleinmanns and Schubert, 2014).

The TrxG methyltransferases ATX1, SDG8, ASHH2 and ASHR1 are involved in both developmental and biotic/abiotic stress responses (Alvarez-Venegas *et al.*, 2003, 2007; Pien *et al.*, 2008; Ding *et al.*, 2009; Ding *et al.*, 2011a,b; Berr *et al.*, 2010; de la Peña *et al.*, 2012; Wang *et al.*, 2014a), but their role in stress memory responses is less well-known. SDG8 has been implicated in memory responses to repetitive mechanical stress of the touch-inducible *TCH3* gene in Arabidopsis (Cazonelli *et al.*, 2014) and ATX1 has been implicated in the memory responses of dehydration stress-response genes (Ding *et al.*, 2012a). Notably, however, ATX1 is not responsible for the memory *per se*, as memory was not fully erased in the lack-of-function *atx1* background (Ding *et al.*, 2012a).

The PcG methyltransferase CURLY LEAF (CLF), which establishes the H3K27me3 marks at developmental genes (Goodrich *et al.*, 1997; Schubert *et al.*, 2006), also functions in a gene-specific manner in the dehydration stress-responding pathway (Liu *et al.*, 2014a,b). Gene-specific roles for H3K27me3 were also reported at biotic stress-responsive genes in rice (Li *et al.*, 2013). Remarkably, however, neither CLF nor H3K27me3 were involved in the memory responses of dehydration stress-response genes (Liu *et al.*, 2014a,b). At the chromatin level, a common feature for all tested genes was the high initial H3K27me3 level during pre-stress (low-transcription) states, which

did not change upon subsequent induction of transcription (in S1) or even after super-induction in S2. However, despite the presence of pre-existing H3K27me<sub>3</sub>, H3K4me<sub>3</sub> accumulated upon induction of transcription. Therefore, the existence of H3K4me<sub>3</sub> and H3K27me<sub>3</sub> at dehydration stress-response genes is not mutually exclusive, and a high level of H3K27me<sub>3</sub> did not affect high-level transcription from the tested genes. In agreement, high amounts of Ser5P Pol II and Ser2P Pol II accumulated at the 5' ends and 3' ends, respectively. Therefore, neither recruitment nor progression of RNA polymerase II were inhibited by the presence of H3K27me<sub>3</sub> (Liu *et al.*, 2014a,b). These results suggest that either the Histone Methyl Transferase establishing the H3K4me<sub>3</sub> mark is able to work on H3K27me<sub>3</sub>-modified nucleosomes, or that the H3K4me<sub>3</sub> and H3K27me<sub>3</sub> marks are present on different histone tails. In structural studies, Schmitges *et al.* (2011) found that that presence of H3K4me<sub>3</sub> inhibits the H3K27-methylating activity, Polycomb Repressive Complex2 (PRC2), only if the target lysine is on the same tail (*in cis*). However, the reverse correlation has not been elucidated.

Therefore, H3K27me<sub>3</sub> does not function as a memory (epigenetic) mark for a specific subset of dehydration stress-responding genes, as the initial (high) H3K27me<sub>3</sub> levels in pre-stressed low-transcription phases did not change upon induced or super-induced transcription. Slightly decreased H3K27me<sub>3</sub> levels were measured at the cold response gene *COR15A* and at salt stress-responding genes after removal of the stress; however, reduced H3K27me<sub>3</sub> levels did not substantially affect the subsequent gene performance (Kwon *et al.*, 2009; Sani *et al.*, 2013), and consequently do not satisfy the criterion for epigenetic marks.

Of particular note is that, despite strongly induced transcription and a high level of H3K27me<sub>3</sub> at two memory genes, *LTP3* and *LTP4*, their transcription dramatically increased in *clf* mutant plants (Liu *et al.*, 2014b). Remarkably, *LTP3* and *LTP4* transcription was also strongly induced in the *msi* background (Alexandre *et al.*, 2009), supporting involvement of the PRC2 complex in the transcriptional responses of a specific subset of dehydration stress memory genes.

Collectively, the results reveal a novel aspect of CLF/H3K27me<sub>3</sub> (PRC2) as a mechanism that limits, rather than prevents, transcription from stress-responding genes. This is a major difference from the repressive 'off' role of PcG at the developmentally regulated gene *AGAMOUS* (*AG*) (Goodrich *et al.*, 1997; Liu *et al.*, 2014b). Therefore, as a silencing mechanism, CLF/H3K27me<sub>3</sub> (PRC2) play different roles at developmental genes and at genes whose expression is altered rapidly in response to environmental conditions: restricting the cellular specificity and suppressing ectopic expression of developmental genes (Goodrich *et al.*, 1997; Bratzel *et al.*, 2010; Farrona *et al.*, 2011) but

defining the range of dynamic expression, without preventing transcription, from specific dehydration stress-responsive genes (Liu *et al.*, 2014a,b). Given that H3K4me<sub>3</sub>-based inhibition of plant PRC2 activity is co-determined by its Su(z)12 subunit (Schmitges *et al.*, 2011), it is important to establish whether/how the roles of CLF/H3K27me<sub>3</sub> at developmental and at stress-response genes are linked to the nature of the subunits of the specific PRC2 complexes (Chanvivattana *et al.*, 2004; Schubert *et al.*, 2006).

#### CHROMATIN AND HISTONE MARKS DURING THE INITIATION AND ELONGATION PHASES OF TRANSCRIPTION

Emerging evidence suggests that chromatin patterns, particularly during responses to stress, are more complex than the simple concept of 'activating/silencing' functions usually associated with gene expression. Although sometimes equated with transcription, gene expression represents distinct processes including transcription, mRNA maturation, export from the nucleus, and mRNA stability. Furthermore, the transcription process consists of discrete phases, each one of which may be specifically influenced by chromatin structure. The mRNA transcript levels, routinely measured as an indicator of transcription, do not reveal the dynamic of the process or which transcription phases have been affected (Ding *et al.*, 2012a,b; Sidaway-Lee *et al.*, 2014). Thus, the question of how chromatin/histone marks mechanistically achieve their effects remains largely unanswered. This question is directly linked to the problem of causality. It is therefore important to establish whether/which modifications affect deposition of the basal transcriptional machinery, and thus contribute to induction of transcription, or whether they facilitate or hinder progression of the polymerase along the template, and thus are a consequence of initiated transcriptional states.

Recent studies have provided insights into the roles of H3K4me<sub>3</sub>, H3K36me<sub>3</sub>, histone H3 acetylation (H3Kac) and ubiquitination of H2B (H2Bub) in the transcriptional process. Which transcription phases are affected by the silencing modifications H3K27me<sub>3</sub>, H3K9me<sub>3</sub>/me<sub>2</sub> and methylated cytosines is less clear.

The increased presence of H3K4me<sub>3</sub> and H3K36me<sub>3</sub> at transcriptionally active genes has defined them as 'activating' marks (Alvarez-Venegas and Avramova, 2005; Zhao *et al.*, 2005; Xu *et al.*, 2008; van Dijk *et al.*, 2010). Despite the almost universal distribution of the H3K4me<sub>3</sub> mark at the 5' ends of transcribed eukaryotic genes, this modification may play different roles in mammalian genes than in yeast, *Drosophila* or plant genes (Fromm and Avramova, 2014). Thus, while H3K4me<sub>3</sub> marks are deemed necessary for recruitment of the pre-initiation complex and polymerase II at the promoters of mammalian genes (Vermeulen *et al.*, 2007), H3K4me<sub>3</sub> was not required for formation of the pre-initiation complex or promoter accessibility in

Set1-dependent yeast genes (Ng *et al.*, 2003) and ATX1-dependent plant genes (Ding *et al.*, 2011b, 2012b). Moreover, the integrity of ATX1/AtCOMPASS (complex associated with Set1), but not its enzyme activity, was essential for assembly of the pre-initiation complex and polymerase II recruitment during the initiation phase of transcription. Accumulation of H3K4me3 downstream of the transcription start site is critical for transition to the elongation phase (Ding *et al.*, 2012b). Deficiencies in H3K4me3 levels at yeast genes and the *Drosophila* hsp70 locus due to dSet1/COMPASS depletion have been also linked to impaired transcriptional elongation (Ng *et al.*, 2003; Ardehali *et al.*, 2011). How histone marks restricted to promoter-proximal nucleosomes activate the process downstream, and how the chromatin environment ensures the optimal release of polymerase II into productive elongation are major open questions (Kwak and Lis, 2013).

Of note, the activating functions of SDG8, H2Bub and H3Kac have been linked to transcriptional elongation as well (Chen *et al.*, 2006; Nelissen *et al.*, 2010; Creppe and Buschbeck, 2011; To and Kim, 2014; Wang *et al.*, 2014a). Aspects of chromatin structure linked to transcriptional elongation have been reviewed by Van Lijsebettens and Grasser (2014).

#### NUCLEOSOMAL OCCUPANCY AND THE H2A.Z VARIANT IN MEMORY RESPONSES

Nucleosomes pose a physical barrier for progression of RNA polymerase II. Clearly, chromatin remodeling factors that reduce histone–DNA contacts or evict the nucleosomes during the passage of polymerase II and restore the structure afterwards are essential for transcription. Active/inactive transcriptional states induced by both developmental and stress-generated signals have been associated with altered nucleosome occupancies and H2A.Z patterns (Saleh *et al.*, 2008; March-Díaz and Reyes, 2009; Berr *et al.*, 2010; Han *et al.*, 2012). H2A.Z has been defined as the temperature-sensing mechanism in plants (Kumar and Wigge, 2010), and the distribution of H2A.Z along gene sequences is critical for differential expression in response to temperature changes (Sidaway-Lee *et al.*, 2014). Activation of response genes and repetitive sequences upon heat stress has been linked to both H2A.Z and a transient loss of DNA-bound nucleosomes (Kumar and Wigge, 2010; Lang-Mladek *et al.*, 2010; Pecinka *et al.*, 2010; Han and Wagner, 2014). However, the induced and super-induced transcription of memory genes was not associated with nucleosome depletion (Ding *et al.*, 2012a), and priming of *WRKY6*, *WRKY29* and *WRKY53* did not involve nucleosome removal either (Jaskiewicz *et al.*, 2011). Loss of nucleosomes in correlation with transcription was reported at the non-memory response gene *RD29A* (Kim *et al.*, 2012a,b).

Whether H2A.Z is involved in transcriptional memory/priming of plant genes has not been reported. The role of

H2A.Z and chromatin in the stress memory behavior of yeast are discussed.

#### MEMORY OF A STRESS IN YEAST

Initially, H2A.Z was considered a key factor in yeast transcriptional memory (Brickner *et al.*, 2007). However, subsequent studies found that, although both H2A.Z and acetylation of H2A.Z were important for strong and rapid induction of the memory gene *GAL1*, neither H2A.Z nor H2A.Zac were important for transcriptional memory (Halley *et al.*, 2010). Most importantly, the transcriptional memory of *GAL* genes did not appear to have a chromatin basis or to involve the inheritance of chromatin states; instead, a catabolic enzyme was found to control transcriptional memory in yeast (Zacharioudakis *et al.*, 2007), and cytoplasmic inheritance of the signaling factor Gal1 was required (Kundu and Peterson, 2010). Likewise, the histone deacetylases SIR2 and Rpd3, and the histone variant H2A.Z were not required for the memory expression of H<sub>2</sub>O<sub>2</sub> tolerance genes after pre-treatment with mild stressors (Berry *et al.*, 2011). Instead, the cytosolic catalase Ctt1p was identified as the factor maintaining the memory of acquired H<sub>2</sub>O<sub>2</sub> tolerance (Guan *et al.*, 2012).

Collectively, the studies in yeast argue against a mechanism involving self-propagating chromatin marks and support a model whereby transcriptional memory is based on cytoplasmic factors rather than having a chromatin basis. Interestingly, short-term epigenetic memory of the *HO* gene depends on chromatin-related co-factors, as the increased ‘firing’ frequency of the *HO* promoter results from enhanced activator binding due to slow turnover of the histone acetylation marks after a previous ‘on’ cycle (Zhang *et al.*, 2013).

#### LENGTH OF STRESS MEMORY

Whether/which changes in chromatin structure that occur during a plant’s history of responses to environmental stresses are inherited through mitotic and meiotic divisions have been critically analyzed in a number of recent comprehensive reviews (Birney, 2011; Hauser *et al.*, 2011; Paszkowski and Grossniklaus, 2011; Schmitz and Ecker, 2012; Gutzat and Mittelsten-Scheid, 2012; Pecinka and Mittelsten-Scheid, 2012; Eichten *et al.*, 2014; Han and Wagner, 2014). Without discussing the issue in more detail here, it is important to emphasize the importance of distinguishing between environmental adaptation (considered stable and heritable) and acclimation (considered plastic and reversible) (see Hauser *et al.*, 2011). Mitotic/meiotic inheritance of stress-acquired traits is linked to short-/long-term memory responses and, consequently, to the plant’s acclimation/adaptation potential. It is also noted that the mechanisms establishing short- or long-term acquisition of stress-induced states may be different, as suggested by the responses to heat stresses and



thermotolerance acquisition in plants (Bokszczanin and Fragkostefanakis, 2013).

Among the stress-triggered chromatin traits, the most intensely studied is the trans-generational propagation of changed DNA methylation patterns, often associated with reactivation of transcriptionally silent loci (Verhoeven and vanGurp, 2012; Boyko *et al.*, 2010; Boyko and Kovalchuk, 2011; Bilichak *et al.*, 2012; Saze *et al.*, 2012; Downen *et al.*, 2012; Migicovsky *et al.*, 2014). As changes in DNA methylation, occurring sporadically or triggered by environmental stresses, may be inherited by successive generations, they are a potential factor in adaptive and evolutionary mechanisms in plants. It is also important that mechanisms for epigenetic reprogramming, involving chromatin remodeling factors and small non-coding RNAs, function during gametogenesis and in early embryo development to counteract and restrict the transmission of acquired chromatin states (Hsieh *et al.*, 2009; Mosher *et al.*, 2009; Slotkin *et al.*, 2009; Lang-Mladek *et al.*, 2010; Iwasaki and Paszkowski, 2014).

Other mechanisms contributing to the loss of epigenetic memory are random DNA damage, followed by replacement of methylated cytosines by unmethylated cytosines during the repair process (Blevins *et al.*, 2014), or spontaneous loss of methylation leading to sporadic emergence of transcriptionally active epi-alleles (Becker *et al.*, 2011; Schmitz *et al.*, 2011; Schmitz and Ecker, 2012). Histone deacetylase6 (HDA6) may function as a memory factor in the perpetuation of CG methylation patterns as heritable epigenetic marks at silenced loci through mitosis and meiosis. Importantly, the identity of a silent locus (established by HDA6) and its silencing (achieved by HDA6-facilitated Methyltransferase1-dependent CG methylation) are two separable processes (Blevins *et al.*, 2014).

Mitotic and meiotic transmission of histone modifications is less well-understood. The maintenance of H3K27me3 during mitoses is facilitated by DNA polymerase  $\alpha$  (Hyun *et al.*, 2013). However, trans-generational inheritance of H3 modifications is less likely, as parental histone H3 is removed from the zygote nucleus, thus limiting the propagation of H3 variants and acquired H3 modifications across generations (Ingouff *et al.*, 2010). Furthermore, the dehydration stress transcriptional memory of *Arabidopsis* memory genes persisted for 5 days in the absence of inducing signals but was lost after 7 days under well-watered conditions. The high levels of Ser5P Pol II and H3K4me3 were also retained for 5 days and decreased to the initial pre-stressed levels after 7 days, consistent with their proposed roles as memory marks for these genes (Ding *et al.*, 2012a). Therefore, the transcriptional memory of dehydration stress-response genes in *Arabidopsis* is a short-term memory that is unlikely to be transmitted to the next generation.

## CONCLUDING REMARKS

Transcription memory behavior indicates that the molecular mechanisms regulating production of different transcript amounts in response to a single stress stimulation versus multiple stress stimulations are different. The ability of chromatin to respond to a stress through both dynamic and stable changes in its structure makes it a potential memory mechanism that may propagate acquired chromatin traits to subsequent generation of cells. However, there is a lack of understanding of how chromatin modifications affect the transcriptional process mechanistically, whether a change in chromatin structure determines a transcriptionally active/inactive state or is a consequence of an established state, and which/how chromatin modifications survive mitosis and/or meiosis. Current models for the memory of acquired stress tolerance and adaptation in yeast that exclude chromatin-based mechanisms suggest that the role of chromatin as a 'memory' factor in plants should be also critically assessed. The length of stress-induced memory is a factor in the adaptive mechanism. As different mechanisms may be involved in short- and long-term memory transmissions, further efforts are required to establish how, mechanistically, environmental factors affect the genome's flexibility, and whether/which acquired chromatin traits are passed on to successive generations as mechanisms for adaptation in a changing environment. Lastly, in addition to the super-induced transcript levels produced from memory genes upon repeated stress, there are at least three more types of transcriptional memory responses (Ding *et al.*, 2013). Only super-induced memory-type transcription is discussed in this review as nothing is currently known about how the other memory responses are achieved. Providing answers to the fascinating questions of how transcriptional memory is achieved opens possibilities for exciting research.

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