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REVIEW PAPER

Role of chromatin in water stress responses in plants

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Abstract

As sessile organisms, plants are exposed to environmental stresses throughout their life. They have developed survival strategies such as developmental and morphological adaptations, as well as physiological responses, to protect themselves from adverse environments. In addition, stress sensing triggers large-scale transcriptional reprogramming directed at minimizing the deleterious effect of water stress on plant cells. Here, we review recent findings that reveal a role of chromatin in water stress responses. In addition, we discuss data in support of the idea that chromatin remodelling and modifying enzymes may be direct targets of stress signalling pathways. Modulation of chromatin regulator activity by these signaling pathways may be critical in minimizing potential trade-offs between growth and stress responses. Alterations in the chromatin organization and/or in the activity of chromatin remodelling and modifying enzymes may furthermore contribute to stress memory. Mechanistic insight into these phenomena derived from studies in model plant systems should allow future engineering of broadly drought-tolerant crop plants that do not incur unnecessary losses in yield or growth.

Key words: ABA, chromatin, drought, epigenetics, genome accessibility, water stress.

Introduction

Plant stress can be defined 'Any unfavourable condition or substance that affects or blocks a plant's metabolism, growth or development' (reviewed by Lichtenthaler, 1998). Plants are exposed to a plethora of environmental stresses throughout their life. Drought attributable to climate change already causes water shortages in large parts of the world (Vorosmarty *et al.*, 2010). Therefore, enhanced response to water deficit is an important trait for both crops and wild plant populations. Water is essential for plant metabolism, transport systems, and for generating the turgor pressure that allows an upright growth habit in herbaceous plants (reviewed by Des Marais and Juenger, 2010). It also adversely affects other aspects of plant growth, for example water stress reduces the rate of nitrogen fixation by legumes and their symbionts (Gil-Quintana *et al.*, 2013). Due to their sessile nature, plants cannot escape from a water-deficient habitat. They instead need to adopt special strategies to cope with water limitation and to avoid substantial impacts on fitness, growth, and development (reviewed by Cramer *et al.*, 2011; Less *et al.*, 2011). The ability of the plant to display tolerance to water stress depends on transcriptional reprogramming (reviewed by Shinozaki and Yamaguchi-Shinozaki, 2007; Ahuja *et al.*, 2010). For instance, factors involved in regulation of stress signal transduction as well as osmolytes and proteins that protect the cell from damage during water stress are induced in response to water deficit (reviewed by Shinozaki and Yamaguchi-Shinozaki, 2007).

In plants, water stress triggers the biosynthesis of the phytohormone abscisic acid (ABA) (reviewed by Xiong and Zhu, 2003). ABA binds to ABA receptors (PYR/PYL/RCAR) in a ternary complex with the clade A protein phosphatase 2C (PP2C) phosphatases, and this triggers a signal transduction cascade that leads to stomatal closure and transcriptional reprogramming (Ma *et al.*, 2009; Park *et al.*, 2009). Briefly, ABA sensing frees SnRK2-type kinases from inhibition by the PP2C phosphatases. SnRK2 autoactivates and subsequently phosphorylates downstream factors that promote

Abbreviations: ABA, abscisic acid; PRMT, protein arginine methyltransferase.

drought tolerance; these include components of ion channels important in the stomatal response and transcription factors that induce stress response gene expression (Fujii *et al.*, 2009; Lee *et al.*, 2009; Umezawa *et al.*, 2009). The first components of this signalling and response pathway were identified in genetic screens for ABA-insensitive mutants more than a quarter of a century ago (Koornneef *et al.*, 1984; Finkelstein, 1994; Finkelstein and Lynch, 2000; Lopez-Molina and Chua, 2000) and include dominant (constitutively active) clade A PP2C phosphatase mutants (*abi1*, *abi2*) and recessive loss-of-function mutants of transcriptional activators of the ABA response (*abi3*, *abi4* and *abi5*) (Koornneef *et al.*, 1984; Finkelstein, 1994; Finkelstein and Lynch, 2000; Lopez-Molina and Chua, 2000). Conversely, loss of function of these PP2Cs or gain of function of the transcription factors leads to ABA hypersensitivity (Parcy *et al.*, 1994; Gosti *et al.*, 1999; Soderman *et al.*, 2000; Lopez-Molina *et al.*, 2001; Merlot *et al.*, 2001; Brocard *et al.*, 2002; Kang *et al.*, 2002; Fujita *et al.*, 2005; Rubio *et al.*, 2009).

Increasing evidence shows that transcriptional reprogramming in stress-responsive gene expression, proper resource allocation to growth versus stress responses, acclimation, and long-term stress memory are at least in part attributable to changes in the chromatin organization (reviewed by Chinnusamy and Zhu, 2009; Mirouze and Paszkowski, 2011; Gutzat and Mittelsten Scheid, 2012). This is not surprising given that chromatin has long been viewed as the interface between the environment and the genome (reviewed by Badeaux and Shi, 2013; Johnson and Dent, 2013; Suganuma and Workman, 2013). In the eukaryotic nucleus, the genome is packaged into the fundamental unit of chromatin, the nucleosome, which is comprised of 147 bp of DNA wrapped around a histone octamer (Luger *et al.*, 1997). The histone octamer consists of two copies each of the histones H2A, H2B, H3, and H4. Nucleosomal arrays are further condensed into higher-order chromatin structures that incorporate the linker histone H1 (Luger *et al.*, 1997). The compaction of the genome in the context of chromatin physically restricts the accessibility of the genomic DNA to regulatory proteins such as transcription factors and RNA polymerase II (reviewed by Petesch and Lis, 2012). Genomic DNA accessibility in the context of chromatin can be altered by various mechanisms including incorporation of histone variants, post-translational modifications of the histones or the DNA, and non-covalent alteration of the positioning or occupancy of the nucleosome (reviewed by Bell *et al.*, 2011). Here, we will review the roles of various mechanisms that affect chromatin organization in water stress responses. We explore the link between water stress perception and modulation of chromatin regulator activity and discuss resource allocation to diverse survival programmes by chromatin regulators. Finally, we consider the role of chromatin in transient or long-term stress memory. Another important mechanism for modulation of gene expression in response to environmental stress relies on non-coding RNAs, several classes of which are critical for changes in chromatin compaction. As there are extensive recent reviews on non-coding RNA-directed gene silencing (reviewed by Wang and Chang, 2011; Wierzbicki, 2012; Castel and Martienssen, 2013) and their roles in development (reviewed by Chen, 2012; Zhang and Chen, 2013) and in abiotic stress responses (reviewed by Contreras-Cubas *et al.*, 2012; Khraiwesh *et al.*, 2012; Ding *et al.*, 2013), we will not cover this topic in the current review.

Chromatin changes induced by water stress

In this section of the review, we will briefly discuss each of the different mechanisms that increase or decrease the accessibility of the genomic DNA in the context of chromatin as well as the available evidence that links each mechanism to water stress responses.

Histone modifications

Certain amino acids of histones, for example in their N-terminal tails, are frequently post-translationally modified via acetylation, methylation, phosphorylation, ubiquitination, sumoylation, or ADP-ribosylation (reviewed by Bannister and Kouzarides, 2011; Zentner and Henikoff, 2013). These modifications are dynamically established or erased by specialized enzymes called 'writers' or 'erasers', respectively (reviewed by Bannister and Kouzarides, 2011). The functional outcome of these changes in histone modifications is either alteration of the strength of the DNA histone interaction or recruitment of non-histone proteins, the so-called 'readers', to the chromatin (reviewed by Bannister and Kouzarides, 2011; Patel and Wang, 2013; Zentner and Henikoff, 2013).

Typically, histone acetylation is correlated with more open chromatin and hence more active transcription, whereas the converse is true for histone deacetylation (reviewed by Zentner and Henikoff, 2013). By contrast, histone methylation can affect different transcriptional outcomes, depending on the amino acid modified and the degree of modification (mono-, di-, or trimethylation) (reviewed by Li *et al.*, 2007). For example, H3K4 and H3K36 trimethylation are found at actively transcribed genes, whereas methylation of H3K27 and H3K9 are well-known marks for repressed loci and heterochromatin, respectively (reviewed by Zentner and Henikoff, 2013). Histone arginine residues can be methylated by protein arginine methyltransferases (PRMTs). Different PRMT family members can catalyse mono-methylarginine, asymmetric di-methylarginine, and symmetric di-methylarginine, which direct either gene activation or repression (reviewed by Ahmad and Cao, 2012).

Several reports in plants have shown that drought sensing or treatment with the stress hormone ABA induces changes in histone modifications (reviewed by Kim *et al.*, 2010a; Yuan *et al.*, 2013). In one study, a short pulse of ABA or salt stress was sufficient to induce global H3S10 phosphorylation and H4K14 acetylation in cultured *Arabidopsis* and tobacco cells (Sokol *et al.*, 2007). In 15-d-old *Arabidopsis* seedlings, H3K9, H3K23, and H3K27 acetylation was enriched at

coding regions of drought stress-responsive genes after short drought treatment, which was correlated with gene activation. H3K4me3 enrichment with gene activation was similar to H3K9 acetylation (Kim *et al.*, 2008). Genome-wide analysis in 4-week-old rosette *Arabidopsis* leaves under dehydration stress revealed a modest change in H3K4me2 and H3K4me1 levels at a subset of known stress response genes, but the H3K4me3 abundance over gene bodies changed more dramatically at genes whose transcript levels increased or decreased during dehydration (van Dijk *et al.*, 2010). Recent genome-wide analysis in 25-d-old rice seedling also uncovered a positive correlation between H3K4me3 accumulation and the expression levels of some drought-responsive genes during dehydration. This correlation could be extended to genes involved in stress-related metabolite and hormone-signalling pathways (Zong *et al.*, 2013). As changes in transcription direct changes in histone modifications (reviewed by Zentner and Henikoff, 2013), further studies are needed to elucidate whether the observed alterations in post-translational histone modifications are a cause or a consequence of the transcriptional changes triggered by water stress.

Histone (de)acetylases

More direct evidence for a role of histone modifications in water stress responses comes from studies of mutants lacking histone-modifying enzymes. Several studies from rice and *Arabidopsis* have shown that the expression of histone deacetylases is regulated by drought and/or ABA (Sridha and Wu, 2006; Luo *et al.*, 2012). In *Arabidopsis*, expression of the plant-specific HD2 histone deacetylases is repressed by ABA and NaCl (Sridha and Wu, 2006; Luo *et al.*, 2012). Plants overexpressing AtHD2C exhibited ABA hyposensitivity (Sridha and Wu, 2006), whilst *hdc2* mutants displayed ABA hypersensitivity during germination (Luo *et al.*, 2012). The gene expression changes reported for these mutants are inconsistent with the phenotypes of mutants lacking components of ABA signalling pathway (Gosti *et al.*, 1999; Merlot *et al.*, 2001), and may therefore be an indirect consequence thereof. Mutations in either one of the genes coding two RPD3-type histone deacetylases, HDA6 and HDA19, in *Arabidopsis* also cause ABA hypersensitivity (Chen *et al.*, 2010; Chen and Wu, 2010; Zhou *et al.*, 2013). Several embryonic genes including *7S1*, *LEC2*, *2S2*, CRA1, *FUS3*, and *LEC1* were de-repressed in *hda19* seedlings (Zhou *et al.*, 2013) in agreement with a role of histone acetylation in activation of these genes (Ng *et al.*, 2006). Similar phenomena were observed in *HDA6* RNA interference lines (Tanaka *et al.*, 2008) and in wild-type plants treated with a histone deacetylase inhibitor (Tanaka *et al.*, 2008). HDA19 associates with the regulatory regions of the above-mentioned embryonic genes (Zhou *et al.*, 2013). It remains to be seen whether failure to directly repress embryonic genes is also observed during germination and whether depression of such genes causes the germination defects and ABA hypersensitivity of germinating *hda19* mutants. Histone acetyltransferase complex components have also been linked to altered water stress responses. A loss-of-function mutant of ADA2b, a component of the GCN5-containing histone acetyltransferase complex, leads to increased drought tolerance (Vlachonasios *et al.*, 2003, 2011). It is not yet known which gene expression changes are directly triggered by this complex and cause the observed phenotype.

Histone lysine methyltransferases

Loss of function of *Arabidopsis* trithorax-like factor ATX1, which trimethylates histone H3 at lysine 4 (H3K4me3), results in decreased dehydration tolerance compared with wild-type seedlings. ATX1 directly regulates transcription of *NCED3*, which encodes a key ABA biosynthesis enzyme. Activation of *NCED3* transcription upon dehydration or ABA treatment is greatly reduced in *atx1* mutants, suggesting that ATX1-mediated H3K4 methylation is required for *NCED3* induction and possibly ABA accumulation caused by water stress (Ding *et al.*, 2011).

Trithorax group proteins act in opposition to polycomb group proteins (reviewed by Simon and Kingston, 2013). H3K27me3 marks established by the polycomb repressive complex 2 (PRC2) induce a persistent silent state of the transcription of the target locus (reviewed by Simon and Kingston, 2013). In *Drosophila*, PRC1 recognizes H3K27me3 and plays a role in the stable maintenance of gene repression (reviewed by Simon and Kingston, 2013). Whilst PRC2 complex components are conserved in plants and metazoans, this is not true for PRC1 complex components (reviewed by Zheng and Chen, 2011; Holec and Berger, 2012). In barley, exogenous ABA application induced expression of components of the PRC2 complex such as HvE(Z) and HvFIE in seedlings (Kapazoglou *et al.*, 2010). In *Arabidopsis*, mutations in the two *EMBRYONIC FLOWER* (*EMF*) genes display strikingly similar developmental defects (Aubert *et al.*, 2001; Yoshida *et al.*, 2001). EMF2 is a homologue of the Su(z)12 component of the metazoan PRC2 complex. It is currently unclear whether EMF1 is associated with PRC1 or PRC2 function (Beh *et al.*, 2012; Kim SY *et al.*, 2012). The recent identification of EMF1 as a structural homologue of the *Drosophila* PRC1 complex component PSC, its ability to inhibit remodelling activity of SWI/SNF ATPases (Beh *et al.*, 2012), and its ability to act as a potent repressor of transcription (Calonje *et al.*, 2008) provide support for the idea that EMF1 may be associated with PRC1. Genome-wide expression analysis of the *emf* mutants revealed that EMFs regulate plant hormone and stress signalling-related genes (Kim *et al.*, 2010*b*). Both EMF1 and EMF2 bind directly to the promoter of *ABI3*, and expression of *ABI3* and its targets are de-repressed in 7- and 14-d-old *emf* mutant seedlings (Kim *et al.*, 2010*b*). More recently, genome-wide binding studies revealed that genes occupied by EMF1 and marked by H3K27me are significantly enriched for Gene Ontology terms such as 'ABA response' and 'abiotic stress response' (Kim SY *et al.*, 2012). A bypass of the embryo lethality of the single unique PRC2 complex component, FIE, allowed assay of the gene expression defects and post-embryonic phenotypes caused by absence of PRC2 function (Bouyer *et al.*, 2011). This revealed germination defects as well as de-repression of embryonic genes and of positive regulators of ABA responses (Bouyer *et al.*, 2011). Further evidence for a PRC-dependent role in water stress-related responses comes from

conditional knockdown of EMF1, which led to increased salt tolerance, whilst removal of a factor with opposing (trithorax group-related) activity had the opposite phenotype (Carles and Fletcher, 2009; Pu *et al.*, 2013). It remains to be determined in the latter two studies which of the observed changes in gene expression are direct. Moreover, no evidence is available as yet that the observed changes in gene expression contribute to the altered water stress responses.

Histone arginine methyltransferases

Mutants lacking the *Arabidopsis* arginine methyltransferase *PRMT5*/*SKB1* (henceforth referred to as PRTM5 for simplicity), which catalyses symmetric arginine dimethylation, display salt and ABA hypersensitivity (Wang *et al.*, 2007; Schmitz *et al.*, 2008; Zhang *et al.*, 2011). Low doses of exogenous ABA result in the growth arrest of germinated *prmt5* but not wild-type embryos (Zhang *et al.*, 2011). Some of the reported gene expression changes in *prmt5* mutants relative to the wild type (Zhang *et al.*, 2011) are inconsistent with the observed hypersensitive phenotype (Merlot *et al.*, 2001; Rubio *et al.*, 2009; Yoshida *et al.*, 2010). Hence, these changes in gene expression may be an indirect consequence of the mutant phenotype. As PRTM5 activity also regulates mRNA splicing (Deng *et al.*, 2010; Zhang *et al.*, 2011) and circadian gene expression (Hong *et al.*, 2010; Sanchez *et al.*, 2010), it will not be trivial to identify the genes, whose misexpression underlies the ABA hypersensitivity of *prmt5*. Indeed, a genetic screen for Ca^{2+} underaccumulation (*cau*) mutants identified an allele of *prmt5* that displays increased drought tolerance and stomatal closure (Fu *et al.*, 2013). The drought tolerance is at least in part due to de-repression of the direct PRMT5/H4Rsme2 target and calcium accumulation sensor *CAS* (Fu *et al.*, 2013).

In summary, mounting evidence supports the idea that post-translational modifications of histones are critical for correct water stress responses in plants. One of the biggest remaining challenges is to elucidate the causal defects that underpin the observed water stress-related phenotypes of mutants lacking histone-modifying enzymes. After identification of genes whose expression is altered in a given mutant in a manner consistent with the observed phenotypes, direct association of the histone-modifying enzyme in question with loci of interest should be tested. Coupled with expected changes in the histone modifications at these loci in stress and non-stress conditions in the mutant and wild-type background, this will allow identification of candidate direct targets of the histone-modifying enzyme. Subsequent genetic tests will enable elucidation of the role (if any) of the identified candidate direct targets in the water stress phenotypes observed in mutants lacking activity of a given histone-modifying enzyme. As loss of function of histone-modifying enzymes and other mutants that affect the chromatin organization are pleiotropic, it cannot be ruled out that the altered stress phenotype of constitutive mutants is due to secondary effects of the altered plant morphology (leaf size, stature). Phenotypic and molecular investigations of chromatin regulators should therefore rely as much as possible on inducible loss-of-function mutants. Tissue-specific knockdown of chromatin regulators can minimize pleiotropic defects. Temporally inducible knockdown of a histone-modifying enzyme enables analysis of altered water stress responses shortly after knockdown in wild-type-looking plants, significantly reducing the secondary effects typical of constitutive mutants.

Histone variants

In most organisms including *Arabidopsis*, there are multiple genes that code for the highly conserved canonical histones (H3, H4, H2A, and H2B), which are mostly expressed during the S phase of the cell cycle (reviewed by Talbert and Henikoff, 2010; Burgess and Zhang, 2013; Skene and Henikoff, 2013). Other less conserved subtypes of histones called histone variants are expressed throughout the cell cycle (reviewed by Talbert and Henikoff, 2010; Skene and Henikoff, 2013). The canonical histones are replaced with histone variants independent of DNA replication. Although they generally do not differ much in sequence from the canonical histones, histone variants can impart distinct characteristics to the nucleosomes, such as stronger or weaker association with the genomic DNA and incompatibility with certain post-translational modifications (reviewed by Talbert and Henikoff, 2010; Skene and Henikoff, 2013). Recent genome-wide studies have revealed the genomic distribution of a subset of the plant histone variants (reviewed by Zilberman *et al.*, 2008; Talbert and Henikoff, 2010; Costas *et al.*, 2011; Wollmann *et al.*, 2012; Skene and Henikoff, 2013).

In plants, linker histone (H1) variants have been linked to the water stress response. The linker histone variant *HIS1-3* gene in *Arabidopsis* is specifically induced by salt, drought, and ABA (Ascenzi and Gantt, 1997; Zhu *et al.*, 2012). Similarly, the tomato linker histone variant *H1-S* gene is also induced by and accumulates in the chromatin in response to water deficit (Scippa *et al.*, 2000). H1-S also accumulates in a drought-tolerant genotype of tomato (Trivedi *et al.*, 2012). Indeed, knockdown of H1-S levels by antisense RNA in transgenic tomato triggered an altered physiological response to water loss such as altered stomatal conductance, transpiration, and net photosynthetic rate (Scippa *et al.*, 2004). Transgenic plants showed an increased association of the heterochromatin with the nuclear membrane under water stress conditions (Scippa *et al.*, 2004); this may trigger increased silencing of these regions (Hubner *et al.*, 2013). Although upregulation of expression of variants of the linker histone H1 in response to drought is a conserved response in higher plants, detailed mechanistic insight into how this histone variant affects chromatin structure or gene expression during water stress is as yet not available. The H2A variant H2A.Z is largely conserved through evolution (reviewed by Talbert and Henikoff, 2010). Genome-wide studies have revealed that the localization of H2A.Z correlates inversely with DNA methylation in both heterochromatin and in gene bodies of active genes (reviewed by Zilberman *et al.*, 2008). It has been proposed that the anti-correlation between H2A.Z and DNA methylation is primarily due to the exclusion of H2A.Z from methylated DNA (Coleman-Derr and Zilberman, 2012). Moreover, Gene Ontology terms enriched among genes upregulated in *h2a.z* triple mutants include 'Response to water deprivation'

and 'Response to ABA' (Coleman-Derr and Zilberman, 2012). The authors propose that H2A.Z deposition in gene bodies confers higher variability in the expression of inducible genes including those that respond to water stress. By contrast, gene-body DNA methylation may stabilize constitutive expression of housekeeping genes by antagonizing H2A.Z deposition (Coleman-Derr and Zilberman, 2012). It will be of interest to determine the effect of reduced availability or incorporation of these and additional histone variants on water stress responses in plants. Given their widespread roles in chromatin stability, conditional disruption of histone variant availability or incorporation may allow more precise investigation of such phenotypes.

DNA methylation

Methylation on the fifth carbon of cytosine bases is an important epigenetic mark that influences chromatin structure and gene expression (reviewed by Jones, 2012). In plants, cytosine methylation is found in the context of CG, CHG, and CHH (H=A, C, or T). Symmetric CG maintenance methylation is catalysed by DNA methyltransferase I (MET1), a homologue of the mammalian methyltransferase DNMT1 (reviewed by Chan *et al.*, 2005; Goll and Bestor, 2005; Law and Jacobsen, 2010). Symmetric CHG maintenance methylation is catalysed by Chromomethyltransferase 3 (CMT3), a plant-specific methyltransferase. Asymmetric CHH methylation is maintained through *de novo* methylation by Domains Rearranged Methyltransferase 2 (DRM2), a homologue of the mammalian DNMT3A/b and the RNA-directed DNA methylation pathway (reviewed by Chan *et al.*, 2005; Goll and Bestor, 2005; Law and Jacobsen, 2010). DDM1 is a SWI/SNF superfamily chromatin remodeller required for all DNA methylation (CG, CHG, and CHH) over long transposable elements and in heterochromatin (Vongs *et al.*, 1993). DDM1 was recently shown to cooperate with the CMT2 methyltransferase to mediate CHH DNA methylation in parallel with the RNA-directed DNA methylation pathway (Zemach *et al.*, 2013).

In *Arabidopsis*, centromeric and pericentromeric regions, repetitive DNA sequences, and transposons are heavily methylated. Many genic regions are also highly methylated, and this is correlated with high gene expression, whereas promoters are mostly depleted of DNA methylation (reviewed by Zhang *et al.*, 2006; Saze *et al.*, 2012). In plants, DNA methylation is associated with diverse biological processes including development and environmental responses (reviewed by Law and Jacobsen, 2010; Saze *et al.*, 2012; Sahu *et al.*, 2013).

Studies from various plant species have shown that abiotic stress may trigger hyper- or hypomethylation in different genomic contexts: hypomethylation of promoters, hyper- or hypomethylation at coding regions, and hypomethylation of transposons (Sahu *et al.*, 2013). For example, genome-wide analysis identified differentially methylated DNA regions in *Arabidopsis* seedlings treated with simulated drought (treatment with polyethylene glycol). The methylome was widely affected by changes in the water potential, with the most dramatic DNA hypermethylation observed near the transcription start site (±500 bp) of protein-coding genes related to stress responses (Colaneri and Jones, 2013). Moreover, it has been proposed that DNA methylation may contribute to stress adaptation. Mangrove trees grown near a salt march had smaller statures than riverside-grown trees and their genomes were globally hypomethylated (Lira-Medeiros *et al.*, 2010). Likewise, in rice, changes in DNA methylation in response to drought were more pronounced in drought-tolerant genotypes (Wang *et al.*, 2011). The altered DNA methylation may contribute to increased differential gene expression upon drought sensing. A subset of the DNA methylation changes induced by drought remained after removal of the stress (Wang *et al.*, 2011). In *Arabidopsis*, low relative humidity was linked to *de novo* DNA methylation and stable repression of genes involved in stomata development, resulting in lower stomata frequency (Tricker *et al.*, 2012, 2013). A T-DNA insertion distal to the *AtHKT1* gene, which encodes a sodium transporter, has been identified as a suppressor of *sos3* (*salt overly sensitive 3*). The insertion prevents a distal enhancer element and RNA-directed DNA methylation from controlling expression of *AtHKT1*, which plays an important role in salt tolerance (Baek *et al.*, 2011). *met1-3* mutants and *met1-3*-derived epigenetic recombinant inbred lines show normal germination in non-stress conditions; by contrast, they fail to germinate in the presence of 150 mM NaCl, a concentration that does not impact germination in the wild type (Reinders *et al.*, 2009). Defects in DNA methylation may thus affect phenotypic plasticity (a topic that has received attention from an evolutionary perspective) in response to adverse environmental conditions (Lira-Medeiros *et al.*, 2010; Wang *et al.*, 2011; Draghi and Whitlock, 2012). It will be critical to identify which of the observed DNA methylation changes contribute to altered water stress response or plasticity.

Non-covalent changes in chromatin state

ATP-dependent chromatin remodelling ATPases alter histone–DNA interactions non-covalently by utilizing the energy derived from ATP hydrolysis to promote changes in nucleosome occupancy, nucleosome positioning, or nucleosome composition (reviewed by Clapier and Cairns, 2009; Hargreaves and Crabtree, 2011; Narlikar *et al.*, 2013). Chromatin remodelling can either increase or decrease the accessibility of a given piece of genomic DNA to *trans*-acting factors and hence facilitate or obstruct transcription, respectively (reviewed by Clapier and Cairns, 2009; Hargreaves and Crabtree, 2011; Narlikar *et al.*, 2013). Four well-studied subfamilies of ATP-dependent chromatin remodellers are the SWI/SNF, ISWI, CHD, and INO80/SWR1 families. Each subfamily has unique domains, which endow it with specialized functions for particular nuclear processes (reviewed by Clapier and Cairns, 2009; Hargreaves and Crabtree, 2011; Narlikar *et al.*, 2013). Among these ATP-dependent chromatin remodellers, only the SWI/SNF and CHD subgroups have been implicated in water stress responses in plants.

SWI/SNF ATPases are conserved from yeasts to humans and plants (reviewed by Flaus *et al.*, 2006; Kwon and Wagner, 2007; Hu *et al.*, 2013; Narlikar *et al.*, 2013). Plant genomes contain three types of SWI/SNF subfamily chromatin remodelling ATPases called BRAHMA (BRM), SPLAYED (SYD), and MINUSCULE (MINU) (reviewed by Jerzmanowski, 2007; Kwon and Wagner, 2007; Sang *et al.*, 2012). The catalytic ATPase subunit forms a core complex together with SWIRM- and SANT- domain proteins (SWI3) and SNF5-domain proteins. Additional accessary proteins, which are frequently tissue- and developmental-stage specific, control targeting and activity of the complex (reviewed by Clapier and Cairns, 2009; Hargreaves and Crabtree, 2011; Kwon and Wagner, 2007). *In vitro* remodelling activity has not yet been demonstrated for members of this subfamily in plants. In *Arabidopsis*, the BRM complex containing SWI3C and SNF5 (BSH) has been linked to ABA and drought response (Han *et al.*, 2012). Germinating *brm* mutants display ABA hypersensitivity and enhanced growth arrest relative to the wild type. Consistent with the mutant phenotype, de-repression of the positive ABA response regulator *ABI5* (Lopez-Molina *et al.*, 2001) was observed (Han *et al.*, 2012). *ABI5* is a direct BRM target and, based on genetic epistasis tests, the *brm* mutant growth arrest is due to the *ABI5* de-repression (Han *et al.*, 2012). BRM repressed *ABI5* expression in the absence of stress by promoting high occupancy of the +1 nucleosome close to the *ABI5* transcription start site (Han *et al.*, 2012). In addition, *brm* mutants displayed increased drought tolerance at multiple stages of development. The molecular underpinnings of this response remain to be elucidated (Han *et al.*, 2012). The MINU1/AtCHR12 ATPase (henceforth referred to as MINU1 for simplicity) has been implicated as a negative regulator of a temporary growth arrest caused by drought and heat stress in adult *Arabidopsis* plants (Mlynarova *et al.*, 2007). Overexpression of MINU1 induces temporary growth arrest under drought as well as salt and heat stress (Mlynarova *et al.*, 2007). Intriguingly, the expression of several stress-inducible dormancy-related genes was reduced in the inflorescence and 4-week-old rosette leaves of MINU1-knockout and increased in MINU1-overexpressing plant. Whilst it is not yet known whether these genes are directly regulated by MINU1 or responsible for the observed phenotypic defects, MINU1 may play a role in the induction of stress response genes upon perception of the stimulus.

The CHD subgroup chromatin remodeller PKL has also been implicated in the ABA response. CHD chromatin remodellers have two tandem chromodomains known to bind methylated lysines, and these domains were shown recently to couple ATP hydrolysis to remodelling (Hauk *et al.*, 2010). Like SWI/SNF ATPases, CHD remodellers can both promote and repress transcription. The vertebrate Mi2-NuRD complex contains histone deacetylase and methyl CpG-binding domain (MBD) proteins in addition to a CHD domain chromatin remodeller (reviewed by Clapier and Cairns, 2009). PICKLE (PKL) is the best-characterized CHD remodeller in *Arabidopsis* and most closely resembles CHD3. Recently, *in vitro* chromatin remodelling activity was demonstrated for PKL (Ho *et al.*, 2013). PKL is required for repression of embryonic genes during seedling development and promotes the developmental transition to vegetative growth (Henderson *et al.*, 2004). *pkl* mutants display exaggerated ABA responses during germination, and fail to germinate in conditions where the wild type germinates properly (Perruc *et al.*, 2007). The ABA-dependent growth arrest of geminating *pkl* plants is mediated mainly by failure to developmentally repress genes strongly expressed during embryogenesis, including *ABI3* and *ABI5*. Increased expression of *ABI3* and *ABI5* in *pkl* mutants relative to the wild type in the presence of ABA treatment is correlated with a reduced level of two repressive histone modifications, H3K9me2 and H3K27me2, at the promoters of these genes (Perruc *et al.*, 2007). Epistasis tests revealed almost *abi5*-like germination and growth responses in *pkl abi5* double mutants, suggesting that the majority of the phenotypic defects can be explained by failure to repress *ABI5*. It is not known whether *ABI5* is directly regulated by PKL. Elucidation of the direct PKL targets is critical, as there is currently evidence for PKL acting both as a trithorax group protein (to counteract polycomb repression) and as a promoter of polycomb repression (Aichinger *et al.*, 2009; Zhang *et al.*, 2012; Jing *et al.*, 2013).

Trade-offs between growth and water stress responses?

Although the underlying mechanisms are largely unknown, growth arrest in adverse environments is thought to be advantageous for plant survival (Lopez-Molina *et al.*, 2001; Achard *et al.*, 2006; Skirycz and Inze, 2010). One hypothesis is that limited resources available to monocarpic (annual) plants in particular can be allocated either to stress response or to continued growth (Bennett *et al.*, 2012). In support of this idea, ABA and drought stress not only induce expression of stress response genes but also repress expression of genes linked to growth and metabolism (reviewed by Shinozaki and Yamaguchi-Shinozaki, 2007; Chaves *et al.*, 2009; Sreenivasulu *et al.*, 2012). In addition, when major drought-responsive transcription factors are overexpressed, transgenic plants display growth retardation in non-drought conditions (reviewed by Shinozaki and Yamaguchi-Shinozaki, 2007). In conditions when the stress does not threaten survival, growth inhibition may lead to an unnecessary reduction in plant growth and hence productivity and yield (Tardieu, 2003; Bennett *et al.*, 2012).

Consistent with the hypothesized trade-off between growth and drought response, several chromatin regulators have been implicated in stress-mediated temporal growth arrest at different stages of plant development. A highly dehydration-sensitive developmental phase in the life of a plant is immediately after germination (Lopez-Molina *et al.*, 2001). Several chromatin regulators act at this stage to trigger water stress-dependent growth arrest, which resembles the growth arrest during late-embryogenesis in seed development. In several cases, the hyperactive stress response is due to a delay or failure to repress the embryonic developmental programme (which is geared towards desiccation tolerance and growth arrest) upon germination.

One example of this type of regulator is PKL. The hypersensitive germination response to ABA of *pkl* mutants is due to failure to developmentally repress *ABI3* and *ABI5* accumulation and is restored by removing *ABI5* function (Perruc *et al.*, 2007). Other embryonic genes such as *LEC1*, *LEC2*, and *FUS3* are constitutively de-repressed and cause formation of embryonic structures on adult *pkl* mutant plants (Dean Rider *et al.*, 2003; Henderson *et al.*, 2004; Aichinger *et al.*, 2009). Likewise, a delay in the developmental repression of the embryonic programme is observed under conditions of reduced histone deacetylase activity (Tanaka *et al.*, 2008). Double mutants between *pkl* and histone deacetylase *hda6* enhanced persistence of embryonic traits and embryonic gene expression (Tanaka *et al.*, 2008). Polycomb group protein and RETINOBLASTOMA-RELATED protein (RBR) are also required for persistent silencing of late embryonic genes including *ABI3* by increasing their histone H3K27 trimethylation (Bouyer *et al.*, 2011; Gutzat *et al.*, 2012; Kim SY *et al.*, 2012; Yang *et al.*, 2013). Although the role of RBR in the abiotic stress response has not been investigated, seedlings with reduced RBR function arrest their growth after germination in non-stress conditions; this is accompanied by de-repression of embryonic genes linked to ABA responses including *ABI3* and *ABI5* (Gutzat *et al.*, 2012).

The SWI/SNF ATPase BRM, by contrast, displayed normal developmental downregulation of embryonic genes (*ABI3*, *ABI5*) at the onset of autotrophic growth and was instead required for repressing expression of positive regulators of water stress responses in the absence of the stimulus (Han *et al.*, 2012). Moreover, the overall reduced vegetative growth of *brm* mutants under non-stress conditions is partly restored by removing *ABI5* function or by disturbing ABA signalling pathway. However, a role for BRM in repression of the embryonic programme cannot be entirely ruled out. Several embryonic genes were expressed in mutants lacking BRM and its close homologue SPLAYED based on transcriptome studies (Bezhani *et al.*, 2007). Of note, the expression of key embryogenesis regulators such as *ABI3*, *LEC1*, and *LEC2* was either not changed or only marginally upregulated (*FUS3*) in adult *brm* hypomorph mutants (Tang *et al.*, 2008). The SWI/SNF ATPase MINU1 is thought to be required for induction of stress-inducible genes that mediate growth arrest under abiotic stress, although direct targets of MINU1 remain to be identified (Mlynarova *et al.*, 2007).

Taken together, these studies highlight a role for chromatin modifying and remodelling enzymes at the nexus of growth versus stress response pathways, both by modulation of developmental programmes and by enabling proper stimulus-dependent changes in gene expression.

Links between stress signalling pathways and chromatin modifying or remodelling enzymes

As outlined above, many chromatin changes including a change in histone variant incorporation, histone modifications, nucleosome occupancy or positioning, or DNA methylation accompany stress-induced changes in gene expression. A critical question is how chromatin regulator activity is controlled to allow precise stimulus-dependent changes in the accessibility of the genome. One way to achieve this may be a direct communication between components of the stress signal transduction pathway and chromatin modifying or remodelling activities.

The question of whether histones in the context of chromatin can directly receive and deliver signals from cellular signal transduction cascades to facilitate specific cellular responses has recently received much attention (reviewed by Badeaux and Shi, 2013; Johnson and Dent, 2013; Suganuma and Workman, 2013). Another intersection between cellular signal transduction and chromatin is indirectly through post-translational modifications of chromatin modifying or remodelling enzymes (reviewed by Badeaux and Shi, 2013). Studies in mammals revealed that histone and DNA methyltransferases are directly phosphorylated by a downstream component of phosphoinositide signalling, the AKT kinase (Cha *et al.*, 2005; Esteve *et al.*, 2011). Likewise, SWI/SNF chromatin remodellers have been shown to be phosphorylated by p38 (Simone *et al.*, 2004), as well as acetylated (Bourachot *et al.*, 2003) and SUMOylated upon signal perception (Galisson *et al.*, 2011).

Signalling transduction by SnRK2 kinases and PP2C phosphatases plays an important role in coordinating whole-plant water stress responses. Calcium-dependent protein kinases (CDPKs) are also critical for proper water stress response, ABA signalling and reduction of reactive oxygen species accumulation (Asano *et al.*, 2012), whilst the inositol polyphosphate 1-phosphatase FIERY1 acts a negative regulator of ABA and stress signalling (Xiong *et al.*, 2001). Thus far, there is no report that links these signalling components directly to the chromatin. However, links between other signal transducers and chromatin regulators have been identified. The clade A PP2C phosphatase HYPERSENSITIVE TO ABA 1 (HAB1) interacts physically with SWI3B, a core subunit of the putative *Arabidopsis* SWI/SNF complex. HAB1 is recruited to ABA response genes, and this recruitment is eliminated upon ABA treatment (Saez *et al.*, 2008). HAB1 may perhaps directly dephosphorylate SWI/SNF complexes containing SWI3B in an ABA-dependent manner. In agreement with this idea, recent phosphoproteomics analyses performed by the Zhu and Shinozaki laboratories revealed that several chromatin regulators, including the BRM SWI/SNF ATPase, are substrates of SnRK2 type kinases in the ABA response pathway (Umezawa *et al.*, 2013; Wang *et al.*, 2013). Whether the observed phosphorylation/dephosphorylation of SWI3B or BRM by SnRK2 kinases/PP2C phosphatases modulates SWI/SNF complex activity remains unknown. The *Arabidopsis* trithorax-like protein and histone H3 lysine 4 methyltransferase ATX1 (Alvarez-Venegas *et al.*, 2003) is involved in dehydration response in both ABA-dependent and -independent pathways (Ding *et al.*, 2011). Intriguingly, ATX1 also interacts directly with phosphatidylinositol (Ptdlns5P), and this negatively influences the ATX1 activity (Ndamukong *et al.*, 2010). Dehydration stress increases accumulation of phosphatidylinositol, a precursor of secondary

messengers in stress signalling (Ndamukong *et al.*, 2010). An increase in the cellular levels of Ptdlns5P keeps ATX1 in the cytoplasm, thereby diminishing ATX1 binding to target genes linked to proper water stress responses (Ndamukong *et al.*, 2010). The phosphoproteomics studies mentioned above identified additional chromatin regulators as phosphorylated upon dehydration or ABA treatment in a SnRK2 kinase-dependent manner (Umezawa *et al.*, 2013; Wang *et al.*, 2013). Although there was little overlap between the phosphorylated peptides identified in the two studies, chromatin-associated proteins identified include putative components of HDAC complexes (e.g. SIN3-like 2, HD2B), histone acetyltransferase complexes (e.g. SNS1, Eaf7 superfamily), histone methyltransferases (e.g. ATXR2, SDG2), chromatin remodelling ATPases (e.g. CHR2/BRM, CHR5/CHD1) and NUCLEOLIN LIKE 1, a nucleolar protein linked to rRNA gene methylation and expression (Umezawa *et al.*, 2013; Wang *et al.*, 2013). In addition, the *Arabidopsis* histone acetyltransferase GCN5 was shown to specifically interact with PP2C6.6, a clade E PP2C with no visible mutant phenotype. GCN5 is dephosphorylated by PP2C6.6 *in vitro*, and loss of PP2C6.6 activity induces GCN5-mediated histone acetylation (Servet *et al.*, 2008). A possible link to water stress responses is supported by the reported expression of PP2C6.6 in guard cells (Galbiati *et al.*, 2008).

The possibility that chromatin regulator activity is modulated upon stress sensing is intriguing with regard to the question of how these factors can execute specific roles in the organism. It is furthermore of practical significance. As chromatin regulators broadly alter the stress-inducible transcriptome, they may be able to direct tolerance not only to a unique stress but to combinations of stresses that are frequently encountered in the field (Mittler and Blumwald, 2010; Yang *et al.*, 2010). The ability to precisely modulate the activity of chromatin regulators—via targeted post-translational modifications for example—should allow utilization of their broad reprogramming capacity whilst minimizing detrimental effects on growth or yield.

Stress-induced transient or long-term epigenetic memory

In higher plants, stress memory phenomena known as 'priming' or 'acclimation' have been described (reviewed by Bruce *et al.*, 2007; Conrath, 2011). Pre-exposure to mild stimuli can make plants more stress resistant and boost responses to recurring stress exposure. Well-known examples of priming are seed priming to enhance germination efficiency and crop yield, temperature acclimation, and systemic acquired resistance (reviewed by Bruce *et al.*, 2007; Conrath, 2011; Gutzat and Mittelsten Scheid, 2012). One mechanism proposed for long-term 'storage' of the stress memory is a mitotically heritable, or epigenetic, change in the chromatin organization. Another could conceivably rely instead on post-translational modification of chromatin regulators. Epigenetic 'stress memory' could be maintained during subsequent development within the life span of an organism that experienced the priming stress in 'somatic memory' or might perhaps even be transmitted to the progeny across generations in 'transgenerational inheritance', a meiotically heritable change in the chromatin organization.

Unlike the mitotically heritable response to prolonged cold (reviewed by Song *et al.*, 2012; Zografos and Sung, 2012), the mechanisms underlying long-term somatic stress memory are not well understood. Previous studies have shown that histone tail modifications such as H3 acetylation or H3K4 methylation occur at drought-responsive genes upon drought sensing, and correlate with active transcription of dehydration response genes (Kim *et al.*, 2008). However, drought-induced H3K9Ac marks and RNA polymerase II occupancy rapidly declined upon rehydration (Kim JM *et al.*, 2012). By contrast, H3K4me3 decreased much more gradually during a 5 h rehydration period (Kim JM *et al.*, 2012), suggesting that H3K4me3 could be a mitotically heritable epigenetic mark for water stress memory. In accordance with this study, another group proposed that H3K4me3 and stalled RNA polymerase II (PolII Ser5P) could function in mitotic stress memory (Ding *et al.*, 2011). Recurrent dehydration induces a higher rate of expression of dehydration response genes such as *RD29B* and *RAB18* than primary dehydration. This is accompanied by higher H3K4me3 and Ser5P PolII accumulation at these loci (Ding *et al.*, 2011). During rehydration, the *RD29B* and *RAB18* transcript levels revert to basal expression, but H3K4me3 and Ser5pP PolII association with both loci remain elevated. The observed stress memory endured until 5 d after recovery (Ding *et al.*, 2011). Likewise, H3K4 hypermethylation mediated by the Set1 histone methyltransferase in *Saccharomyces cerevisiae* was proposed to provide molecular memory of recent transcriptional events (Ng *et al.*, 2003). It was suggested that elevated H3K4 trimethylation is important for genes to be rapidly switched on and off by environmental stimuli and that it acts to prevent the associated genes from being silenced (Ng *et al.*, 2003). The combined data suggest the presence of a conserved mechanism for stress memory in metazoans.

One of the main difficulties in monitoring epigenetic profiles for long-term stress memory are confounding epigenetic changes caused by altered plant growth and development in stress-challenged plants. Another challenge is determining the period for which plants can 'remember' the priming event. An enhanced response to the second treatment shortly after the primary treatment could result from 'leftover' proteins and metabolites that were induced by the first stress treatment. Recently, Sani *et al.* (2013) developed an experimental protocol to monitor epigenetic profiles, which aims to avoid these problems. They showed that a mild transient salt treatment of young *Arabidopsis* seedlings establishes long-term somatic memory. This was accompanied by specific changes in the H3K27me3 profile, which remained after 10 d of subsequent growth, and resulted in drought/high-salt tolerance priming in the pre-treated plants without morphological differences between primed and non-primed adult plants (Sani *et al.*, 2013).

Interestingly, H3K4me3 is generated by a methyltransferase that belongs to the trithorax group of proteins

(TrxG), whilst H3K27 is trimethylated by the PRC2 complex of polycomb group proteins (PcG). Recently, several elegant *in vitro* and *in vivo* studies have shown that mitotic epigenetic inheritance of methylation at H3K4 and H3K27, which have been linked to stress memory in plants (above), may be mediated by the continued presence of TrxG and PcG proteins at the replication fork and on mitotic chromatin (Lanzuolo *et al.*, 2011; Follmer *et al.*, 2012; Fonseca *et al.*, 2012; Lengsfeld *et al.*, 2012; Lo *et al.*, 2012; Petruk *et al.*, 2012).

Naturally occurring DNA methylation-based epialleles and epigenetic recombinant inbred lines generated in the laboratory are stably inherited for many generations in plants (reviewed by Mirouze and Paszkowski, 2011; Paszkowski and Grossniklaus, 2011; Becker and Weigel, 2012; Zhang *et al.*, 2013; Roux *et al.*, 2011; Schmitz *et al.*, 2013). Several reports have attempted to demonstrate stress-induced epigenetic states that are inherited by the non-stressed progeny, so-called meiotic or transgenerational epigenetic inheritance (reviewed by Boyko and Kovalchuk, 2011; Hauser *et al.*, 2011; Paszkowski and Grossniklaus, 2011; Gutzat and Mittelsten Scheid, 2012; Grossniklaus *et al.*, 2013). For example, a recent study reported salt stress-induced epigenetic inheritance of DNA methylation, histone modifications, and gene expression (Bilichak *et al.*, 2012). However, clear evidence for stress-induced chromatin modifications that are stably inherited by subsequent generations and contribute to phenotypic plasticity is still lacking in plants (reviewed by Mirouze and Paszkowski, 2011; Pecinka and Mittelsten Scheid, 2012; Grossniklaus *et al.*, 2013). As genetic changes—for example due to transposon activation—are also observed in these lines, careful assessment of the epigenetic nature of the inherited trait is required. Criteria to shore up more unambiguous support for epigenetic transgenerational stress inheritance were suggested recently and include well-controlled stress treatments and phenotypic analyses, a comprehensive or synoptic view of associated chromatin changes, and establishment of causality, as well as heritability for more than two generations (reviewed by Pecinka and Mittelsten Scheid, 2012; Grossniklaus *et al.*, 2013).

Conclusion

At a time when we face the twin challenges of human population growth and loss of arable land due to climate change, it is critical to understand the molecular mechanisms that regulate water stress tolerance and mitotic inheritance of stress responses during priming. Evidence is mounting for a role of DNA methylation, histone modifications, and altered nucleosome occupancy, positioning, or composition in both responses. As stresses in nature do not occur in isolation (reviewed by Mittler and Blumwald, 2010; Yang *et al.*, 2010), it is possible that changes in chromatin organization may endow the plants with the ability to survive combinations of stresses and to remain primed for further stress responses. Challenges for the future are: (1) to elucidate which chromatin alterations may be instructive for altered stress responses, rather than a consequence thereof; (2) to understand which chromatin alterations lead to stress tolerance that is mitotically (or meiotically) heritable; and (3) to devise ways to modulate the activity of 'instructive' chromatin regulators in ways that allow enhanced primary or heritable stress tolerance without causing growth or yield trade-offs.

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