INTRODUCTION

Plants must adapt to environmental changes in order to ensure their survival. Plants store information (memory) over a certain period of time from previously experienced stimuli and develop future responsiveness to these stimuli (Lämke and Bäurle, 2017; Friedrich et al., 2019). Plant memory is completely different from human and animal memory, since plants do not store memory in a brain structure. However, our understanding of heat acclimation is very limited. In the model plant Arabidopsis thaliana, changes in the expression patterns of heat memory genes play a central role in regulating plant survival and adaptation to recurring heat stress. Heat stress-related transcription factors and histone-modifying enzymes function in the sensitized expression of heat memory genes via the deposition and removal of histone modifications. Chromatin-remodeling complexes and miRNA accumulation also trigger the sustained expression of heat memory genes. In this review, I describe studies of heat acclimation that have provided important insights into the molecular mechanisms that lead to flexible and reversible gene expression upon heat stress in plants.

Key words: heat acclimation, heat memory, histone modification, nucleosome positioning, miRNA
tion and nucleosome positioning. These modifications can be implemented in response to stress.

Small RNAs such as microRNAs (miRNAs) also function in gene-silencing pathways to control protein-coding transcript activity and hence plant stress memory (Voinnet, 2009; Axtell, 2013). Dicer cleaves small hairpin precursors (pre-miRNAs) during miRNA biogenesis and produces 21–23-nt-long miRNAs. The resulting miRNAs are loaded onto RNA-induced silencing complexes (RISCs) and cleave messenger RNAs (mRNAs) and/or inhibit their translation via imperfect base pairing with cognate mRNAs. The accumulation of miRNA is also greatly affected by stress. Thus, histone modification-, nucleosome positioning- and miRNA-based changes lead to differences in gene expression and ultimately functional protein accumulation, hence leading to phenotypic changes. Therefore, elucidating how gene expression is affected by stimuli is crucial to understanding how plants employ stress memory for better survival and adaptation.

Temperature stress is one of the most detrimental stresses threatening plant survival. Temperature stress in plants is categorized into three types: high-temperature, chilling or freezing stress. Plants memorize a temperature experience for a certain period of time and establish ways to better respond to subsequent stresses. Temperature memory is heritable not only after cell divisions but also over multiple generations via epigenetic mechanisms. Plants exposed to moderate temperature stress acquire thermotolerance that helps them withstand subsequent exposure to normally lethal temperatures. In this review, I focus on plant responses and adaptation to high temperature (heat). In general, the plant heat response is controlled by the transcriptional regulation of heat shock genes. Here, I describe recent advances in our understanding of how the expression of heat shock genes is regulated in a temporal manner in Arabidopsis plants. Multiple regulators contribute to this regulation. Low-temperature adaptation is covered in detail in other review articles (Hoermiller et al., 2017; Friedrich et al., 2019).

**HEAT MEMORY AND ITS REGULATORS IN PLANTS**

Heat acclimation and heat memory genes Exposure to moderate temperatures allows plants to acquire thermotolerance to help them withstand subsequent exposure to normally lethal high temperatures. This process is referred to as heat acclimation (or acquired thermotolerance), and was first described 60 years ago (Alexandrov, 1964; Vierling, 1991). Non-primed plants cannot survive exposure to lethal high temperatures (Fig.

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**Fig. 1.** Heat memory temperature conditions and memory gene expression. (A) Schematic representation of different heat conditions. Left, basal thermotolerance conditions. Plants that encounter a severe heat stress (HS) fail to survive or adapt to the environment. Right, heat memory conditions. Acclimation to mild stress (ACC) can act as a priming cue. Even after exposure to a severe heat stress (HS) during the memory phase, plants are able to survive. (B) Schematic representation of heat memory gene expression. Left, gene expression under basal thermotolerance conditions. Without ACC, heat memory genes are not sensitized. Right, gene expression under heat memory conditions. After ACC, the expression of heat memory genes continues. Furthermore, induction in response to HS becomes stronger or faster.
Heat memory in Arabidopsis

1A: left). Once plants have been exposed to a mild stress, they are acclimated and maintain their primed state for several days (Fig. 1A: right). During this memory phase, plants are able to survive exposure to even lethal high temperatures.

Heat shock proteins (HSPs) are molecular chaperones that play critical roles in heat acclimation, not only in plants but also in animals. The major function of HSPs is to control the refolding of misfolded proteins, thereby helping maintain cellular homeostasis. HSPs are divided into five groups based on their approximate molecular weights: small HSPs (sHSPs), HSP60, HSP70, HSP90 and HSP100 (Al-Whaibi, 2011). Recent studies have revealed that sHSPs and related non-canonical HSPs, such as HEAT-STRESS-ASSOCIATED32 (HSA32), are particularly important for heat acclimation. In general, HSP expression is strongly induced by heat only while the heat treatment is applied. By contrast, nearly all sHSP genes exhibit sustained expression after the cessation of heat stress, which represents the memory phase (Swindell et al., 2007; Stief et al., 2014; Lämke et al., 2016). Also, sHSP genes (such as HSP21, HSP22 and HSP17.6C) show stronger or more rapid induction in primed plants than in non-primed plants after exposure to lethal high temperatures (Yamaguchi et al., 2021) (Fig. 1B). Hence, many sHSP genes can be considered heat memory genes.

Heat memory regulators Since plants are exposed to diurnal and seasonal temperature fluctuations (Nagano et al., 2012, 2019), heat acclimation should reflect not only HSP regulation, but also general mechanisms that govern cellular and/or metabolic homeostasis on a daily basis. Mainly due to technical limitations, three major approaches were initially taken to screen heat acclimation-defective mutants: forward-genetics-, transcriptome- and gene annotation-based approaches. Initially, defective in long term acquired thermotolerance mutants were identified (Wu et al., 2013). More recently, a forward-genetics approach based on the visualization of heat memory genes was established. This approach enables heat memory regulators to be identified in a less biased manner (Brzezinka et al., 2016; Urrea Castellanos et al., 2020). Transcript profiles of Arabidopsis under heat acclimation conditions have also been utilized to narrow down the responsible genes. The mutant phenotypes were examined based on their modes of regulation (Sedaghatmehr et al., 2016). For the candidate gene approach, mutants defective in stress responses and/or epigenetic regulation were grown under heat acclimation conditions (Stief et al., 2014; Yamaguchi et al., 2021).

Consistent with the sensitized expression patterns of sHSPs, mutations in genes such as HSP21, HSP22, HSP17.6C and HSA32 lead to defects in heat acclimation. Conversely, overexpression of these genes led to enhanced growth compared to wild-type plants during heat acclimation (Lämke et al., 2016; Sedaghatmehr et al., 2016; Yamaguchi et al., 2021). Thus, not only is the expression of these genes sensitized by acclimation, but the resulting proteins also confer thermotolerance. The expression of some of these genes is directly regulated by the heat stress transcription factor HEAT SHOCK FACTOR A2 (HSFA2). Histone 3 lysine 27 trimethylation (H3K27me3) demethylases, SWI2/SNF2 chromatin-remodeling complexes and the miRNA156–SQUAMOSA PROMOTER BINDING PROTEIN (SPL) module also contribute to the activation of sHSPs. All heat memory regulators identified to date except for the FORGETTER2 (FGT2)–PHOSPHOLIPASE D a2 (PLDA2) pathway are involved in the transcriptional regulation of heat memory genes (Table 1). The identification of the protein phosphatase FGT2 pointed to the importance of phosphatidic acid-dependent signaling or membrane composition dynamics during heat acclimation. The regulation of HSP genes by epigenetic regulators is discussed in the following section.

MOLECULAR MECHANISMS OF HEAT MEMORY GENE REGULATION

Heat memory and histone modification By combining microarray analysis with reverse genetics using T-DNA insertion lines, HSFA2 was identified as a key regulator of heat acclimation (Charng et al., 2007). Although HSFs are conserved proteins in both plants and animals, the number of HSF genes differs among organisms (Wu, 1995; Nover et al., 2001). Within the Arabidopsis family of 21 heat stress transcription factors, HSFA2 is the most highly expressed member in response to heat. hsfa2 mutants show strong defects in heat acclimation (Charng et al., 2007). HSFA2 directly binds to downstream targets such as HSP21, HSP22, HSP18.2 and ASCORBATE PEROXIDASE 2 via heat stress elements and induces their expression in response to heat stress (Nishizawa et al., 2006; Schramm et al., 2006; Nishizawa-Yokoi et al., 2009). HSFA2 expression and HSFA2 binding at target loci peak soon after mild acclimation stress and decline relatively quickly (Nishizawa et al., 2006; Charng et al., 2007; Lämke et al., 2016). However, HSFA2-regulated sHSP targets show sustained expression for a few days after the cessation of acclimation stress (Charng et al., 2007). Furthermore, even after the complete decline of sHSP gene expression, some of these heat memory genes are sensitized to subsequent heat shock in an HSFA2-dependent manner (Yamaguchi et al., 2021). The role of HSFA2 in extending the duration of activation of heat memory genes is exerted by the sustained accumulation of permissive histone modifications. Dimethylation or trimethylation of histone H3 lysine 4 (H3K4me2 or H3K4me3) has been correlated with competent/active
genes. Hyper H3K4me2 and H3K4me3 persist even after HSFA2 binding at the HSP22 and HSP18.2 loci has declined (Lämke et al., 2016). This elevated H3K4me2 and H3K4me3 enables the sensitized response to subsequent heat stock. However, how HSFA2 affects the deposition of these two permissive marks at the molecular level remains an open question. To further understand this mechanism, identification of the interacting partners of HSFA2 is required. Such partners may include histone methylases of H3K4me2 and H3K4me3.

Using a gene annotation-based approach, a specific group of JUMONJI-C DOMAIN CONTAINING PROTEINS (JMJs) was identified as regulators of heat acclimation (Yamaguchi, 2021; Yamaguchi and Ito, 2021a). JMJs have been characterized as lysine demethylases whose functions are highly conserved in the plant and animal kingdoms (Pan et al., 2007; Xiao et al., 2016). Thus far, five Arabidopsis JMJ proteins have been shown to function as demethylases of H3K27me3: JMJ11/EARLY FLOWERING 6 (ELF6), JMJ12/REDATIVE OF EARLY FLOWERING 6 (REF6), JMJ13, JMJ30 and JMJ32 (Lu et al., 2011; Crevillén et al., 2014; Gan et al., 2014; Cui et al., 2016; Yan et al., 2018; Qiu et al., 2019). All these H3K27me3 demethylases except JMJ13 are redundantly required for heat acclimation (Yamaguchi et al., 2021). elf6 ref6 jmj30 jmj32 quadruple mutants display defects in heat acclimation. Acclimation induces sustained H3K27me3 demethylation at the HSP22 and HSP17.6C loci by JMJs, poising the HSP genes for subsequent activation (Yamaguchi et al., 2021) (Fig. 2B). Upon sensing heat after a 3-day interval, JMJ30 directly and rapidly reactivates these HSP genes. Although the jmj30 single mutant does not show any heat memory phenotype, JMJ30 functions redundantly with other JMJs to control this memory. In elf6 ref6 jmj30 jmj32 quadruple mutants, heat memory genes are not sensitized due to higher levels of H3K27me3. Hence, the activation of HSP22 and HSP17.6C upon subsequent heat

| Molecule | Description | Plant phenotype | Molecular phenotype in the mutant/transgenic background | References |
|----------|-------------|----------------|--------------------------------------------------------|------------|
| HSA32    | Non-canonical heat shock protein | hsa32: Heat acclimation defect | Decrease or increase of chaperone function | Chang et al., 2006 |
| HSFA2    | Heat shock transcription factor | hsfa2: Heat acclimation defect | Low H3K4me3 at HSP22 | Lämke et al., 2016 |
| JMJs     | H3K27me3 demethylases | jmqj: Heat acclimation defect | High H3K27me3 at HSP22 / HSP17.6C | Yamaguchi et al., 2021 |
| HSP22    | Small heat shock protein | hsp22: Heat acclimation defect | Decrease or increase of chaperone function | Li et al., 2018 |
| HSP17.6C | Small heat shock protein | hsp17.6c: Heat acclimation defect | | Yamaguchi et al., 2021 |
| FGT1     | PHD finger protein | fgt1: Heat acclimation defect | High nucleosome occupancy at HSA32 | Brzezinka et al., 2016 |
| CHR11    | SWI2/SNF2 chromatin remodelers | chr11 chr17: Heat acclimation defect | High nucleosome occupancy at HSA32 | Brzezinka et al., 2016 |
| CHR17    | SWI2/SNF2 chromatin remodeler | brm: Heat acclimation defect | High nucleosome occupancy at HSA32 | Brzezinka et al., 2016 |
| miR156/SPL microRNA/Transcription factors | 35S:MIR156h: Heat resistance | Decrease or increase of SPL mRNA | Stief et al., 2014 |
| AGO1     | RNA slicer | ago1: Heat acclimation defect | High miR156 | Stief et al., 2014 |
| DCL1     | Dicer | dcl1: Heat acclimation defect | High miR156 | Stief et al., 2014 |
| HSP21    | Small heat shock protein | amir-HSP21: Heat acclimation defect | Decrease or increase of chaperone function | Sedaghatmehr et al., 2016 |
| HSP101   | Heat shock protein | hsp101: Heat acclimation defect | Decrease of chaperone function | Wu et al., 2013 |
| FGT2     | Type 2C protein phosphatase | fgt2: Heat acclimation defect | Changes in lipid homeostasis | Urrea Castellanos et al., 2020 |
| PLDA2    | Phospholipase D a2 | plda2: Heat acclimation defect | Changes in lipid homeostasis | Urrea Castellanos et al., 2020 |
Heat memory in Arabidopsis

Heat memory in Arabidopsis stress is delayed compared to wild type. JMJ-mediated H3K27me3 removal and HSFA2-mediated H3K4me3 deposition occur at the same HSP22 locus (Fig. 2B) (Yamaguchi and Ito, 2021b). However, whether these regulatory mechanisms are connected at the molecular level or if the two pathways act independently remains unknown. The relationship between JMJs and HSFA2 needs to be further examined in the future.

Heat memory and nucleosome positioning

Other key regulators of heat memory have been identified in a heat memory reporter-based forward-genetics screen. The HSA32 reporter, which recapitulates the endogenous HSA32 expression pattern, was used in this analysis (Charng et al., 2006; Brzezinka et al., 2016; Urrea Castellanos et al., 2020). Two causal genes were identified, one of which functions in heat memory gene expression. FGT1 encodes a protein with an ATP-binding DExD/H-like helicase domain, a helicase C-like domain and a PHD finger. The PHD finger of FGT1 binds to the N-terminal region of H3, which is consistent with a possible function as a co-activator, like its ortholog in Drosophila, Strawberry notch (Majumdar et al., 1997). The fgt1 mutant is specifically impaired in heat acclimation due to defects in the sustained induction of HSA32 after acclimation. FGT1 physically interacts with chromatin remodelers belonging to different groups: the SWI/SNF chromatin remodeler BRAHMA (BRM) and the ISWI chromatin remodelers CHROMATIN-REMODELING PROTEIN 11 (CHR11) and CHR17 (Huanca-Mamani et al., 2005; Bezhani et al., 2007; Li et al., 2012, 2014). Likewise, brm and chr11 chr17 display heat acclimation defects. FGT1 and BRM regulate the expression of shared heat memory genes by controlling proper nucleosome positioning in response to heat acclimation (Fig. 2B). Interestingly, FGT1 and BRM are associated with the expression of heat memory genes in the absence of stress, suggesting that their recruitment itself is not heat-dependent, unlike HSFA2 and JMJs. The molecular mechanism underlying how nucleosome positioning is altered only in response to heat stress is not well understood.

Heat memory and miRNAs

Because miRNAs are known to modulate plant stress responses, the role of ARGONAUTE1 (AGO1), a component of the RISC that binds miRNA during heat acclimation, was first examined using a candidate gene approach (Voinnet, 2009;
Axtell, M. J. (2013). The hypomorphic ago1 mutant shows a reduced capacity for heat acclimation, suggesting that the miRNA pathway is specifically required for heat acclimation. Phenotypic analysis of a dicer-like1 (dcl1) mutant with defects in the biogenesis of miRNAs also supported this hypothesis. Transcriptome analysis further identified differentially expressed pri-miRNAs in acclimated ago1 mutants compared to acclimated wild-type plants. Among these pri-miRNAs, the accumulation of miR156 in response to acclimation is the most robust (Fig. 2C). Several studies have uncovered the important functions of miRNA156 and its targets, SPLs, in regulating the switch from the juvenile to the adult phase in plants (Wang et al., 2009; Wu et al., 2009; Yamaguchi et al., 2009). Consistent with the accumulation of miR156, SPL genes are post-transcriptionally downregulated by miR156 after acclimation (Fig. 2C). The resulting down-regulation of SPLs triggers the sustained induction of heat memory genes (Fig. 2C). How miR156 is upregulated in response to acclimation, and how heat memory genes are downregulated following the reduction in SPL protein levels, are still not understood. miRNA levels could be regulated by stress-inducible transcription factors, such as HSFA2. Although some of the SPLs function as transcriptional repressors (Gou et al., 2011), perhaps SPL proteins directly regulate the expression of heat memory genes.

CONCLUSIONS AND OUTLOOK

Since plants are sessile organisms, their ability to adapt to environmental changes is essential for their survival. In the past 10 years, researchers have identified numerous regulators of plant stress responses, and various interactions between these regulators, using a candidate gene approach, forward/reverse-genetics approaches and omics data. In particular, memory of a previously experienced environment helps to determine the adaptation capacity of a plant to future stress conditions. However, interactions between these regulatory factors have not been characterized. Although environmental responses after exposure to stress are fully time-dependent, technologies to continuously track stress responses over time are not yet well established. The generalization or simplification of environmental responses based on only a small amount of omics data at certain time points may be misleading. The visualization of stress levels coupled with the integration of a great deal of omics data via high-resolution readouts will provide new insights and/or a more precise understanding of the complexity of environmental responses in plants.

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