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Mini Review

Molecular mechanisms of the plant heat stress response

Ai-Li Qu, Yan-Fei Ding, Qiong Jiang, Cheng Zhu *

China Jiliang University, Xueyuan Road 258, Hangzhou 310018, China

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ABSTRACT

High temperature has become a global concern, which seriously affects the growth and production of plants, particularly crops. Thus, the molecular mechanism of the heat stress response and breeding of heat-tolerant plants is necessary to protect food production and ensure crop safety. This review elaborates on the response networks of heat stress in plants, including the Hsf and Hsp response pathways, the response of ROS and the network of the hormones. In addition, the production of heat stress response elements during particular physiological periods of the plant is described. We also discuss the existing problems and future prospects concerning the molecular mechanisms of the heat stress response in plants.

1. Introduction

Future episodes of high temperature are expected to occur more frequently as a consequence of the greenhouse effect. Increasing temperatures have become an important constraint impacting grain yields in crops. By the end of this century, the vulnerability of crop plants will increase with increasing high temperature variability [1]. Therefore, the heat stress response mechanism in plants, particularly in crops, and the cultivation of the heat-tolerant breeds has recently received much attention.

The percentage of seed germination and photosynthetic efficiency declines when plants encounter heat stress. During the reproductive growth period, the functions of the tapetal cells are lost [2], and the anther is dysplastic under heat stress. Increased temperatures inhibit the swelling of pollen grains during flowering and results in anther indehiscence and the poor release of pollen grains [3–5]. In addition, high temperatures impact the number of pollen grain germinating on the stigma, thereby reducing spikelet fertility and grain yields [6].

Plants have evolved various physiological and molecular mechanisms to resist heat stress. Based on the expression data from different plant species under different tissue types, developmental stages and growth conditions, high temperatures affect approximately 2% of the plant genome [7–9]. These genes primarily comprise the response network of heat stress.

2. Proteins involved in heat stress response

Currently, transcriptomics and proteomics have been used to identify heat stress-responsive genes and proteins in plants [6,10,11], which can be divided into two groups. The first group includes signaling components, such as protein kinases and transcription factors. The other group includes functional genes, such as heat shock proteins (Hsps) and catalase (CAT) [12].

2.1. Hsf-hsp-hsbp1

Heat stress factor (Hsf) and Hsp play a central role in the heat stress and acquired thermotolerance in plants (Fig. 1). Hsf serves as the terminal component of signal transduction and mediates the expression of Hsp. However, attempts to increase thermotolerance through the overexpression of single Hsf or Hsp genes has limited effects [13], suggesting that Hsf and Hsp confer heat stress resistance only under synergistic conditions.

Hsf is the central control protein during the heat stress response [13–15]. A total of 19 [14] and 21 [16] Hsf members have been cloned in rice (*Oryza sativa* L.) and Arabidopsis (*Arabidopsis thaliana*), respectively. However, the functions of these proteins are poorly understood [17]. According to the structural features of their oligomeric domains, plant Hsf proteins comprise three conserved evolutionary classes: A, B and C [14,16,18]. HsfA has been relatively well studied [19]. HsfA1a is a master regulator [18,20] and HsfA2 is a major heat stress factor [13,14] in plant heat stress responses. In addition, in Arabidopsis, HsfA4a and HsfA8 act as sensors of the reactive oxygen species (ROS) produced as a secondary stress during the heat stress response [17,21]. Hsp families, including Hsp100, Hsp90, Hsp70, Hsp60 and small Hsps (sHsps) [13], are essential for normal growth and development in plants [22]. N22 is a heat-tolerant genotype in rice. Proteomic analyses have revealed that Hsps are significantly up-regulated in N22 plants after 6 h at 38 °C [6], showing that Hsps play an important role in the resistance of heat stress in plants. Moreover, Hsp70 participates in the feedback control of heat stress [19]. Heat shock factor binding

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* Corresponding author. Fax: +86 0571 86914510.
E-mail address: pzhch@cjlu.edu.cn (C. Zhu).

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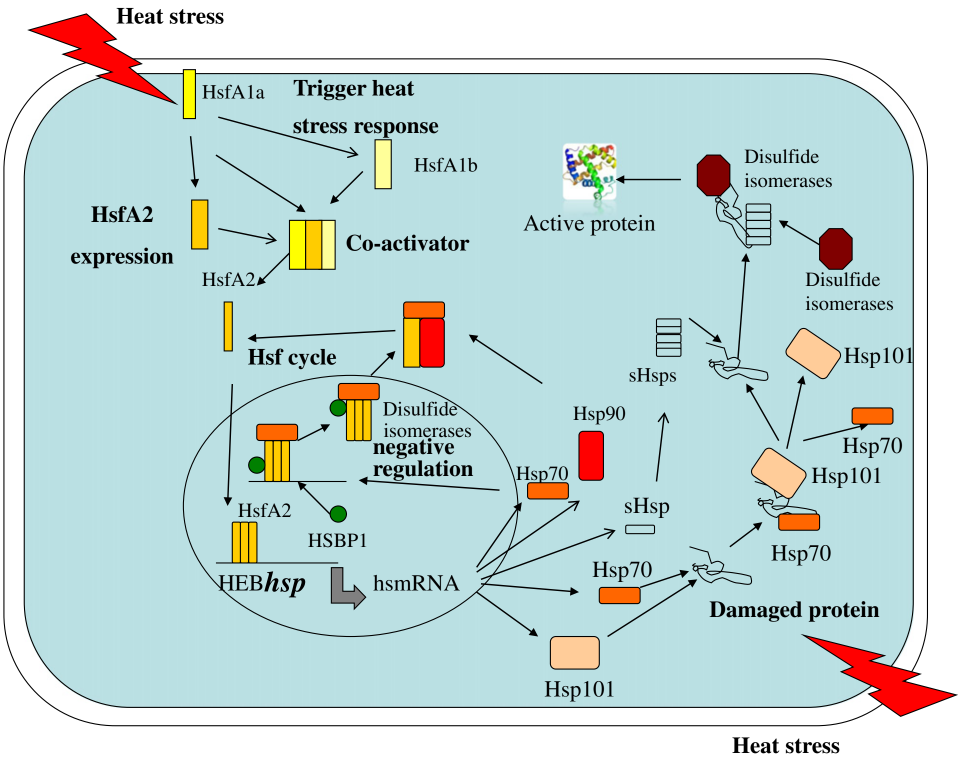

Fig. 1. HSF-HSP-HSBP1 pathway. HsfA1a triggers the heat stress response through the induction of HsfA1b and HsfA2 expression, which also forms co-activators with both proteins. HsfA2 induces the expression of various Hsps. Hsf70, Hsf101 and sHsp participate in the repair of damaged proteins. HSBP1 and Hsp70 participate in negative regulation when the heat stress response diminishes. During this process, active HsfA2 homotrimers are converted to inactive monomers that participate in the recycling of Hsfs.

protein1 (HSBP1) is a negative regulon that interacts with the hydrophobic heptads of HsfA1a (Hsf1).

When the content of the inactive protein exceeds the threshold value during heat stress, the inert monomers form active homotrimers [19]. The expression of heat shock genes is activated through the binding of Hsf to the heat shock promoter element (HSE). Hsps primarily assist in the folding and intracellular distribution, assembly and degradation of proteins through the stabilization of partially unfolded proteins. However, these proteins do not contain specific information for correct folding, but rather prevent unproductive interactions [23]. Peptidyl-proly l isomerase or protein disulfide isomerase act as direct folding catalysts [24,25].

During the attenuation of the heat shock response, HSBP1, as the negative regulon, binds to the negative domain of HsfA1a. The transcriptional activity of HsfA1a is repressed through the direct binding of Hsp70 and the resulting change in the conformation. HSBP1 interacts with Hsp70 to form the disintegration complex, and subsequently the active HsfA1a homotrimers are converted into inactive monomers, thereby inhibiting the master regulative function of HsfA1a [19] and interrupting the heat stress response signal. The complex of Hsf homotrimers and Hsp70 has been detected, confirming the negative function of the Hsp70 in the heat stress response [26]. When heat stress re-occurs, the entire cycle is reinitiated, including the change between the active and inactive Hsfs, and the utilization of the monomers [14].

2.2. The response of reactive oxygen species

Oxidative stress is produced as a secondary stress during the heat stress response, which results in the abundant production of reactive oxygen species (ROS). In plants, the accumulation of H_2O_2 is a rapid process [17,27]. This signal is affected through histidine kinases and Hsfs (Fig. 2). HsfA4a acts as a sensor of the H_2O_2 signal [28]. However, HsfA5 is a negative regulon of this pathway [29]. Upon attenuation of the ROS signal, HsfA5 forms a heterooligomer with HsfA4 and interferes with the DNA-binding capacity of active HsfA4a homotrimers. Hsfs transfer the ROS signal to downstream transcription factors through the MAPK signal pathway. These transcription factors primarily include the Zat family, WRKY transcription factor gene family, multiprotein bridging factor 1c (MBF1c) and NADPH oxidases (Rboh).

The Zat family responds to diversified stress, including heat stress. Zat7 [30], Zat10 [28] and Zat12 [31] respond to the heat stress. Zat12 is necessary for the expression of APX, Zat7and WRKY25 [31,32]. WRKY25 responds to heat and oxidative stress [32]. MBF1c, a highly conserved transcriptional coactivator, is up-regulated through heat stress. Mbf1c mutants were unable to accumulate SA and trehalose during heat stress; in contrast, MBF1c-overexpressing plants showed the up-regulation of both proteins [33]. SA and trehalose are important signaling factors in plant defense responses. In addition, Rboh acts as the positive amplifier, enhancing the production of ROS signaling and maintaining this signal in an active state for longer time through the oxidation of NADPH [34] during stress.

The MAPK signal pathway activates downstream redox-sensitive transcription factors. In response to oxidative stress, these transcription factors are coordinated through specific oxidative stress-sensitive *cis*-elements in gene promoters [35], which primarily encode antioxidant enzymes and non-enzymatic antioxidants. Among multitudinous antioxidant enzymes, ascorbate peroxidase (APX) and catalase (CAT) primarily act as the scavengers of ROS under heat stress in plants [36].

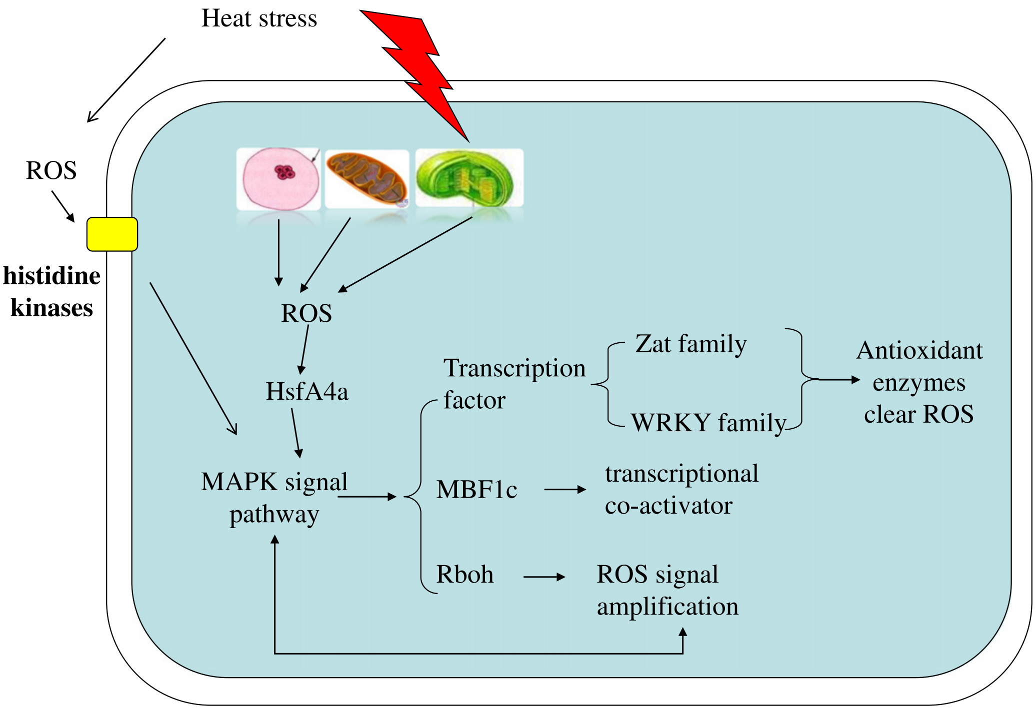

Fig. 2. The response of ROS. During heat stress, endogenous and exogenous ROS accumulates. Endogenous ROS are sensed through histidine kinases, and HsfA4a act as the sensors of exogenous ROS. Subsequently, the MAPK signal pathway is activated and induces the expression of transcription factors, MBF1c and Rboh. The transcription factors, including the Zat and WRKY families, induce the expression of antioxidant enzymes, and MBF1c acts as a transcriptional co-activator. Rboh enhances the production and maintenance of ROS signaling.

2.3. The dehydration-responsive element binding protein (DREB) of the AP2/ERF family

The AP2/ERF family is a large plant-specific transcription factor, including the DREBs, which activate the expression of abiotic stress-responsive genes [12,37]. DREB2 functions during the heat and osmotic stress response [38], and osmotic stress is one of the secondary stresses of heat stress. DREB2A and DREB2B are induced under high temperatures. In Arabidopsis, DREB2A, which has a negative regulatory domain in the central region, plays an important role in heat stress. The deletion of this region transforms DREB2A into a constitutively active form (DREB2A CA). The overexpression of DREB2A CA in transgenic Arabidopsis enhanced tolerance to heat stress [39]. *OsDREB2B* encodes DREB2-type transcription factors to improve tolerance to heat stress in rice. *OsDREB2B* expression is regulated through alternative splicing and generates two types of transcripts, namely functional (full-length protein) and nonfunctional (proteins with premature stop codons) forms. During heat stress, the full-length protein regulates the stress-responsive gene expression, whereas under normal growth conditions, the nonfunctional protein is formed [12], which prevents the careless consumption of plant resources.

2.4. Other regulatory proteins

Proteomics is a useful technology to study differential protein expression [6,40–42]. The protein levels in the leaves and anthers have been studied under heat stress in rice [6,43]. The majority of these proteins were identified as sHsps, regulatory proteins and proteins that function in energy, metabolism and redox homeostasis [43]. Anthesis is the most sensitive stage to temperature in plants [3,6,42,44]. In addition, small cold shock proteins (sCSPs) are significantly up-regulated in the anthers of N22 plants under heat stress [6]. Thus sCSPs might function as RNA chaperones and Rho transcription termination factors in plants. The dirigent-like protein and subtilisin-like serine protease also influence anther dehiscence and increase the degree of dehiscent in N22 plants more than other heat-sensitive genotypes [6,40]. In addition, the Fe requirement for the development proper microspores or pollen germination and Fe-deficiency protein was largely up-regulated through heat stress in heat-sensitive genotypes and hindered pollen viability [6,41]. Similar cases were observed in wheat [45], barley [46] and Arabidopsis [47], indicating that the heat stress response in plants has a certain degree of homology. Moreover, in wheat, some proteins involved in starch synthesis, such as glucose-1-phosphate adenyltransferase, were down-regulated under heat stress [48]. In barley, the expression of *S*-adenosylmethionine synthetase (SAM-S) was increased [46]. The enzymes involved in malate metabolism and the Calvin cycle have been identified in Arabidopsis [47].

3. The regulatory pathway under heat stress in plants

3.1. Hormone pathway

Hormones are produced through environmental signals, such as heat stress, in the cell. Auxin is a phytohormone that orchestrates many physiological mechanisms under heat stress and common condition [49]. In plants, particularly in developing anthers [50], endogenous auxin is reduced in response to heat stress. In 2010, Tadashi [51] reported that endogenous auxin levels were significantly reduced, and the expression of the *YUCCA* auxin biosynthesis genes was repressed through heat stress and male sterility in barley and Arabidopsis during heat stress. However, the application of auxin completely reversed these conditions. These results indicated that heat stress represses auxin signaling in an anther cell-specific manner.

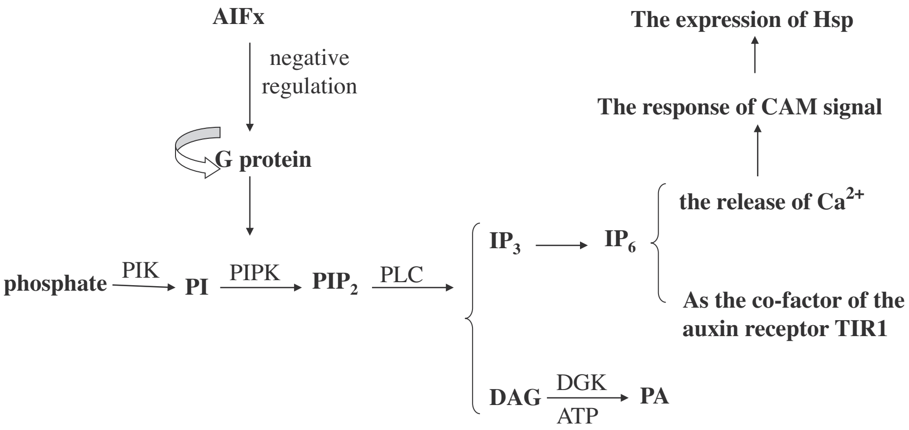

Fig. 3. Phospholipids pathway. Heat stress induces a rapid increase in the levels of PA and PIP_2. The G protein activates or localizes PIPK and is negatively regulated through AlF_x. PIP_2 produces DAG and IP_3. IP_3 is rapidly converted to IP_6, which is responsible for the release of Ca^{2+} and is a co-factor of the auxin receptor TIR1.

Salicylic acid (SA) functions in plant growth and development and plays a role in the heat stress response. SA prevented oxidative damage through the detoxification of superoxide radicals, altering the capacity of antioxidants and preventing damage to membranes in plants. Therefore, SA can enhance heat resistance, improve fertility and increase yield [52]. Indeed, the application of SA in rice [52] and Arabidopsis [53] increased antioxidant capacity.

Abscisic acid (ABA), a key stress hormone, is responsible for stomatal closure upon osmotic stress. ABA is related to the Rboh regulation. Rboh mediates ABA-induced ROS generation in guard cells. RbohD and RbohF are the major catalytic subunits involved in this process [34,53]. However, two ABA functions have been identified in plants: high levels of ABA promote growth during the vegetative period and inhibit growth during the reproductive period, causing male sterility and a reduction in the setting rate [12,54].

3.2. Phospholipid pathway

There have also been extensive reports of a role for phospholipid-based signaling in response to various abiotic and biotic stresses [55], including heat stress [56] (Fig. 3). The remodeling of membrane phospholipids is characteristic of these responses, and phosphoinositide signaling is the early event following the onset of heat stress. During subsequent incubation at 40 °C, membrane lipid metabolism is not largely affected [57]. While low levels of phosphoinositide signaling induce stress resistance, higher levels could trigger cell damage [57].

Phosphatidyl inositol 4,5-bisphosphosphate (PIP_2) and phosphatidic acid (PA) act as key mediators of signaling pathways, membrane dynamics and cytoskeletal organization [58–60]. Under heat stress, the levels of PIP_2 and PA are increased within 2 min of pre-labeling with 32P-orthophosphate [57].

Under heat stress, the transfer from phosphate to PI is catalyzed through PIK. Through the activation of PLD and PIPK, heat stress induced the rapid increase of PA and PIP_2 levels [57]. The G protein transduces the heat-initiated signal required for PIP_2 and PA accumulation and might be required to activate or localize PIPK during heat stress. A role for G protein-based signaling has been identified as a component of the heat stress response [61], which is negatively regulated through AlF_x [62]. PIP_2 is transformed from PI and is transported to the nuclear envelope and nucleolus. The DAG produced through the PLC-catalyzed hydrolysis of PIP_2 is rapidly converted to PA through the action of diacylglycerol kinase (DGK) at the expense of ATP [57]. IP_3 is the other product of PIP_2, which is rapidly converted to IP_6 [63]. IP_6 is responsible for the release of Ca^{2+} [64], rather than IP_3 [63], and IP_6 is a co-factor of the auxin receptor TIR1 [65]. In Arabidopsis, heat stress does not affect TIR1 [51], which might be important for the heat stress response through the auxin pathway.

4. Conclusions

Recent global warming has resulted in an annual combined loss of approximately 40 megatons or $5 billion in crops [51]. Heat stress impacts plant growth and produces osmotic and oxidative secondary stresses. In addition, the peroxidation of phospholipids and remodeling of membrane phospholipids occurs, and proteins unfold, misfold, or aggregate. To resist heat stress, many enzymes, non-enzymes, antioxidants, and hormones are produced [66], comprising the heat stress response networks.

However, in the near future, the concurrence of many types of stresses becomes more and more common. Therefore, understanding the interactions of the response networks to various stresses and cultivating various stress-resistant plant breeds will be beneficial. Many studies have identified abundant heat-tolerant genes, most of which are quantitative trait loci (QTL). However, these QTLs are largely Hsf and Hsp genes, which do not highly contribute to heat tolerance [67]. A few of these QTLs have been associated with spikelet fertility. Thus, the use of classical and modern breeding protocols, identification of genetic diversity for high temperature tolerance, use of presowing seed treatments and planting materials and development of plants with high temperature tolerance will be important [66].

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