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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

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

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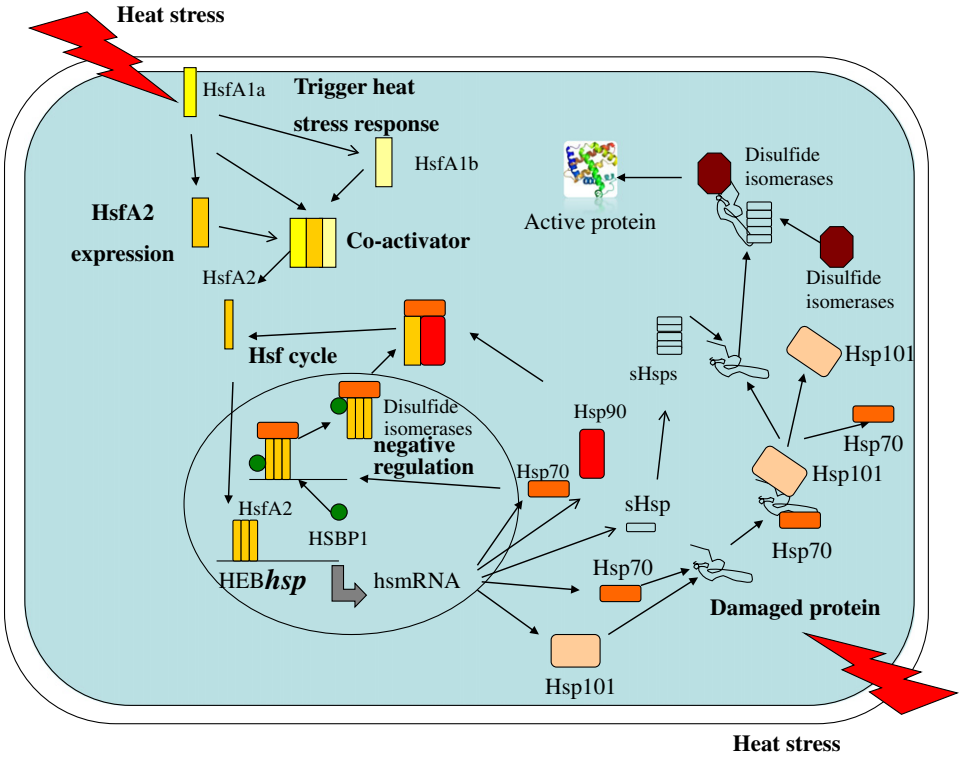

Fig. 1. HSF-HSP-HSBP1 pathway. HsfA1a triggers the heat stress response through the induction of HsfA1b and HsfA2 expression, which also forms co-activators with both proteins. HsfA2 induces the expression of various Hsps. Hsf70, Hsf101 and sHsp participate in the repair of damaged proteins. HSBP1 and 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].