Molecules and Cells

Journal Club

How Does Global Warming Sabotage Plant Immunity?

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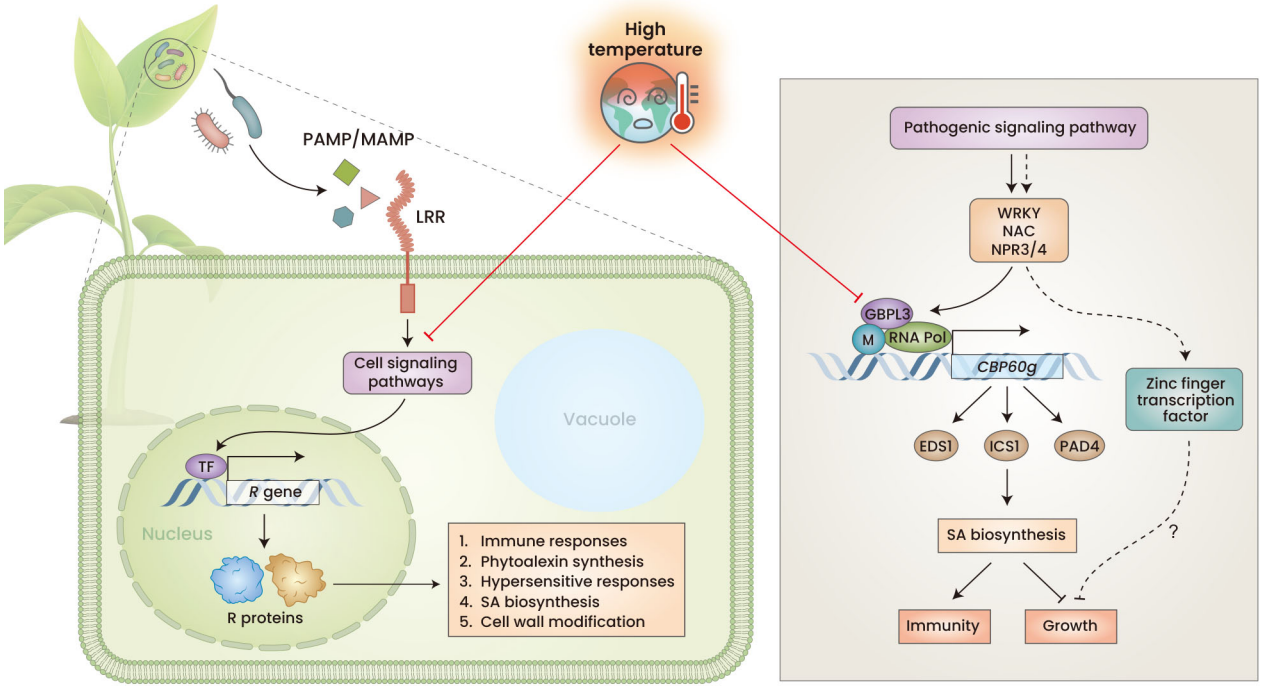
https://doi.org/10.14348/molcells.2022.0150
www.molcells.org



Temperature-induced vulnerability in the plant is regulated through the optimized *CBP60g* expression and salicylic acid (SA) biosynthesis: SA plays a vital role in plant defense. Under high temperatures, plant defense mechanism is compromised. A recent study by Kim et al. (2022) demonstrated that elevated temperature negatively affects GDAC (GBPL3 defense-activated biomolecular condensates) formation and its recruitment to the *CBP60g* promoter. This causes SA biosynthesis suppression. The findings of this work will serve as a benchmark in understanding the molecular mechanism underlying of plant-environment-disease triangle. PAMP, pathogen-associated molecular pattern; MAMP, microbe-associated molecular pattern; LRR, leucine-rich repeat; TF, transcription factor.

Received 26 September, 2022; revised 7 October, 2022; accepted 18 October, 2022; published online 7 December, 2022

eISSN: 0219-1032

PLANT DEFENSE IS COMPROMISED IN HIGH-TEMPERATURE STRESSES

Plants often face a wide range of biotic and extreme environmental stresses as sessile organisms. These stresses cause physiological changes, resulting in plant growth and yield penalty. Since plants lack a circulatory immune system, they solely depend on the cell-autonomous local responses, for example, the transportation of signaling molecules through a membrane-separated vesicular system (Kwon et al., 2020; Won and Kim, 2020). Elevated temperature profoundly impacts plant defense responses and render plants vulnerable to pathogens. Research breakthroughs elucidating the underlying molecular mechanisms of this pathway were made in the last decade. Zhu et al. (2010) reported that a mutation in the resistance (*R*) gene sufficiently changes the temperature sensitivity of the plant immune response and confers resistance at high temperatures. Another study (Cheng et al., 2013) reported that high temperature decreased the secretion of pathogenic effectors but accelerated the proliferation of pathogenic organisms, inhibiting effector-triggered immunity (ETI) while enhancing PAMP-triggered immunity (PTI). Defense-related hormones, such as jasmonic acid, salicylic acid (SA), and ethylene, play an essential role in pathogen-induced hypersensitive responses (Huot et al., 2014). Previous studies have indicated that a higher temperature significantly suppresses pathogen-induced SA production while enhancing other hormone pathways (Gangappa et al., 2017; Huot et al., 2017; Malamy et al., 1992). However, the mechanism underlying the selective inhibition of SA signaling under high ambient temperatures in the presence of pathogens has remained elusive (Gangappa et al., 2017; Huot et al., 2017). Recently, Kim et al. (2022) found that the calmodulin-binding protein 60-like g (CBP60g) transcription factor impacts SA production, basal immunity, and ETI at an elevated temperature. CBP60g proteins are highly conserved in plants. Therefore, they might play an essential role in mediating the triangular interactions among crop plants, the pathogen, and the environment (Kim et al., 2022; Zhu et al., 2010).

TEMPERATURE-SENSITIVE PHASE TRANSITION OF GBPL3 COORDINATES CBP60g MEDIATED DEFENSE SIGNALING

Calcium signaling is essential in plant defense responses associated with PTI and ETI (Wang et al., 2009). Within the cell, calcium signals are transduced by binding calcium ions to calmodulins (CaMs), which subsequently bind to CaM binding proteins (Bouché et al., 2005). In *Arabidopsis thaliana*, *Arabidopsis* hereafter, the CBP60 family comprised seven members (CBP60a-CBP60g). A previous report (Wang et al., 2009) suggested that *CBP60g* is induced by infection with *Pseudomonas* and plays a critical role in disease resistance through the activation of SA signaling. In a recently published study, Kim et al. (2022) discovered that the temperature-induced inhibition of SA biosynthesis in response to pathogens is because of the transcriptional suppression of *CBP60g*. Through bulk RNA-sequencing analysis between Col-0 seedlings, which were challenged with *Pseudomonas syringae* pv. *tomato* (*Pst*) DC3000 at ambient (23°C) and high temperatures (28°C), the authors discovered that the transcription of *CBP60g* and *SARD1*, a closely related gene of *CBP60g* (Wang et al., 2011), were down-regulated at the high temperature. Kim et al. (2022) further examined the upstream regulators that transcriptionally repressed *CBP60g* and *SARD1* expression.

GUANYLATE BINDING PROTEIN-LIKE 3 (GBPL3) forms GBPL defense-activated biomolecular condensates called GDACs, which bind to the promoters of *CBP60g* and other defense-related genes by recruiting mediator complex and RNA polymerase II (Huang et al., 2021). Kim et al. (2022) discovered the disassembly of the GDACs at a higher growth temperature (28°C) and concomitant inhibition in the expression of *CBP60g* and *SARD1*. This observation unveils a previously unknown pathway behind the inhibition of SA production at elevated temperatures when plants are challenged with a pathogenic elicitor.

SECURING PLANT DEFENSE UNDER HIGH TEMPERATURES WITHOUT GROWTH PENALTY

To survive in an unfavorable environment, plants must manage limited resources to relocate to the designated organ for growth and accelerate their defense mechanisms. During this process, plants often activate defense signaling at the expense of growth. However, recent studies (Figueroa-Macías et al., 2021; Neuser et al., 2019) suggested that there could be an alternative instead of this 'trade-off' signaling between growth and defense regulatory mechanism that reprogram developmental pathways based on the hostile environment. When Kim et al. (2022) over-expressed *CBP60g* under the *35S* promoter using the uORFs$_{TBF1}$ strategy, which contains the upstream open reading frame (uORF) region of the *TBF1* gene, they found that transgenic *Arabidopsis* plants could maintain a defense system and SA production even at high temperatures. Notably, the uORFs$_{TBF1}$ region allows controlled protein translation in response to pathogenic infection (Xu et al., 2017). This observation, with the other conclusion led by Kim et al. (2022), strongly proposed that *CBP60g* is the missing link that inversely regulates plant vulnerability toward the pathogen under high temperatures.

Recent studies (Jing et al., 2019; Okada et al., 2021; Poncini et al., 2017) reported that root growth was impaired by perceiving biotic or abiotic stress signals. Consistent with this finding, we also identified that plant elicitor peptide 1 (PEP1), a general sensor of biotic and abiotic stresses, affects the reprograming of *Arabidopsis* root apical meristem and vascular development (Dhar et al., 2021). In addition, PEP1 strongly impacts the cell-to-cell symplastic connection, which is responsible for transporting developmental signals over a long distance. A molecular link between PEP1 perception and reprograming of the developmental signal is yet to be uncovered, which could also be employed in engineering plants with intact danger sensing without root growth penalty.

In summary, the study led by Kim et al. (2022) provided comprehensive evidence of how environmental factors control the SA-induced defense signaling pathway in connection to plant immunity. The author's results will serve as a bench-

mark for understanding the concept of the plant-environment-disease triangle and prompt future researchers to identify the mechanistic approach toward the underlying defense response pathways.

ACKNOWLEDGMENTS

We thank all members of the Lee lab for their discussions and comments at various stages. This work was supported by the grants NRF-2018R1A5A1023599 and NRF-2021R1A2C3006061 to J.-Y.L. from the National Research Foundation of Korea. S.D. was supported by the Brain Korea 21 Four Program.

AUTHOR CONTRIBUTIONS

S.D. and J.-Y.L. wrote the paper.

CONFLICT OF INTEREST

The authors have no potential conflicts of interest to disclose.

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