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RESEARCH HIGHLIGHT

Sustaining plant immunity in rising temperature

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Upon exposure to high temperature, some plant immune responses are compromised. In a recent *Nature* paper, Kim et al. show that ectopic expression of transcription factors involved in regulating the immune signal salicylic acid synthesis and signaling could sustain immune competency at high temperature, suggesting a possible engineering strategy for fighting global warming in agriculture.

Temperature is a major environmental factor that has a large impact on plants and their interactions with pathogens. The temperature effect on plant immunity is complex, with the most prevalent interaction being the suppression of plant immunity by elevated temperature.[1] This phenomenon was first observed as the complete loss of resistance to tobacco mosaic virus (TMV) when temperature was raised from 22 °C to 30 °C.[2] This resistance was later found to be conferred by the *N* gene, which was cloned using a clever selection scheme developed based on the temperature sensitivity of the resistance.[3] The *N* gene is a founding member of intracellular nucleotide-binding site and leucine-rich repeat domain (NLR) immune receptor genes in plants. Since then, several additional NLRs have been shown to be sensitive to elevated temperature.[4] In addition to inhibiting local resistance to TMV, elevating temperature to 26 °C also blocks the establishment of systemic acquired resistance (SAR) in naïve tissues,[5] a broad-spectrum resistance mechanism which was later found to require the production of the immune signal salicylic acid (SA). Consistent with these earlier observations, expression of genes involved in SA biosynthesis, such as *CBP60g* and *ICS1*, and SA signaling, such as *PAD4* and *EDS1*, was found to be significantly inhibited at 30 °C.[6]

With global climate change, susceptibility of the plant immune system to heat poses a great threat to global food supply and the ecosystem. How to sustain plant immunity in a warmer climate is an urgent challenge that needs to be overcome. In a recent report in *Nature*,[7] Kim and colleagues in Sheng Yang He's group have developed a solution which involves constitutive expression of the *CBP60g* gene encoding a master transcription factor of a large number of plant immunity genes to restore SA production and disease resistance at elevated temperature. Normally, expression of *CBP60g* is inhibited by increased temperature due to the reduced number of GBPL defense-activated condensates (GDAC) formed by GUANYLATE BINDING PROTEIN-LIKE 3 (GBPL3) to recruit transcriptional coactivators of the Mediator complex (MED) and the RNA polymerase II (POL II) machinery to the *CBP60g* promoter. This study reveals GBPL3 as a new temperature sensor in plant immunity based on its physical property, i.e., condensate formation, connecting temperature to defense gene transcription (Fig. 1).

How much GBPL3 explains the temperature sensitivity of the plant immunity needs to be assessed in the future. Earlier genetic screens for temperature-resilient resistance have identified NLR proteins as one of the primary components for temperature sensitivity in plant immunity.[8] Different forms of the NLR protein, SNC1, confer disease resistance with different temperature sensitivities, and missense mutations in the NLR proteins SNC1 and N could restore specific defense responses or overall disease resistance at elevated temperature. The temperature sensing mechanism of NLR remains unclear, although reduced nuclear localization is an early consequence of increased temperature on a few NLR proteins (Fig. 1).

The Kim et al. study also demonstrates the feasibility of using fundamental knowledge to combat global climate change. Genetic engineering can be applied to manipulate master regulators of plant immunity for temperature-resilient resistance. While NLRs are potential engineering targets, the modification needs to be individualized for each NLR protein. GBPL3 will be a possible alternative target once its temperature-sensitive condensate formation is better understood. Ectopic expression of *CBP60g* performed by Kim et al. circumvents the above challenges in protein designs, allowing the rescue of its target genes whose expression is repressed under elevated temperature.

We may wonder why nature has not made plant immunity more resilient at high temperature through evolution of those target genes. It is possible that the reduction in NLR- and SA-mediated resistance at elevated temperature is compensated by an increase in pattern-triggered immunity as proposed by Cheng et al.[9] They reasoned that this is correlated with their observation that the secretion of bacterial effectors favors low temperature, whereas bacterial multiplication increases with elevating temperature accompanied by production of more microbe-associated molecular patterns. Another possible explanation is that the cost of maintaining these immune responses at elevated temperature might be too high to favor evolution selection. It is worth noting that SA is a molecule involved in plant thermogenesis through which plant temperature is raised by alternative oxidation to volatilize chemicals to attract insect pollinators in plants like voodoo lilies.[10] It is not surprising that plants would downregulate this heat-generating molecule ("calorigen") under elevated temperature. Moreover, reduction in SA levels has been shown to improve pollen development under mild heat conditions through an increase in the plant hormone jasmonic acid.[11] These potential pleiotropic effects, especially those on fitness, in engineering plant immune responses can be minimized if the responses are only activated transiently upon pathogen challenge or restricted to specific plant tissues. Indeed, when Kim et al. put *CBP60g* downstream of the 5′ leader

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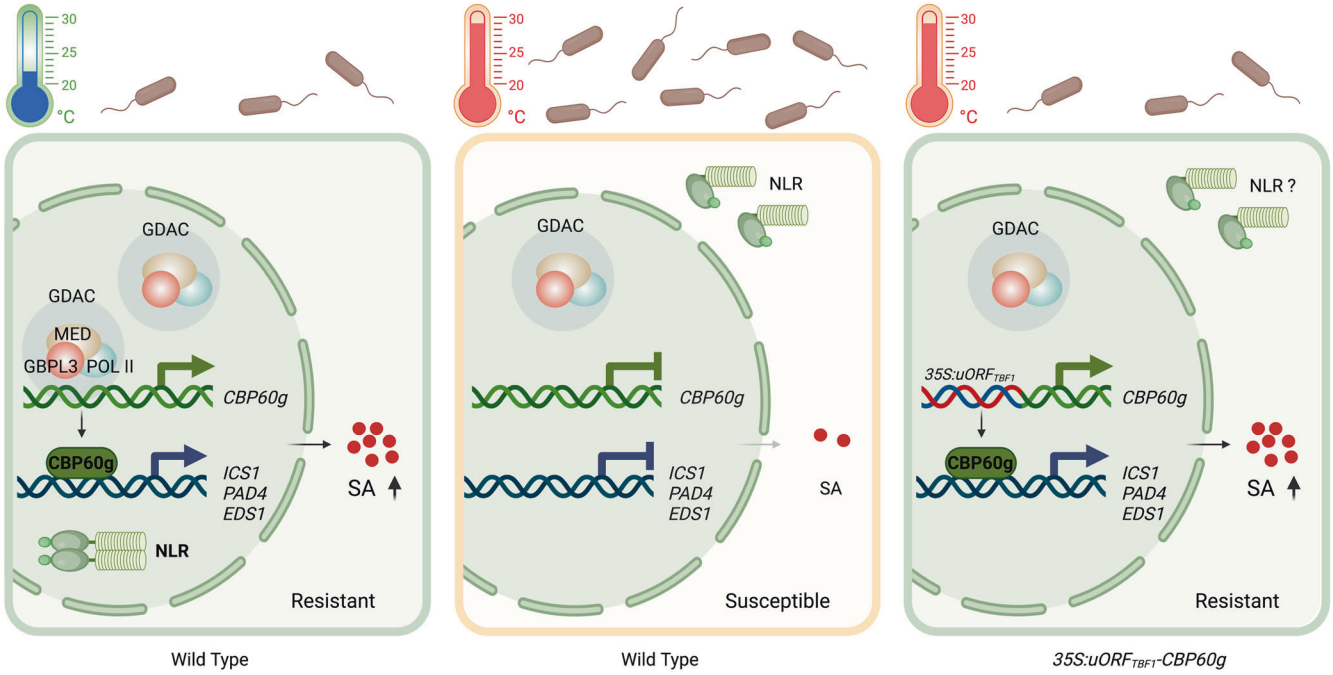

Fig. 1 A model of molecular basis of temperature sensitivity in plant immunity. Dampening of plant immunity by elevated temperature is associated with reduced expression and/or activity of intracellular immune receptors (NLRs) or the immune signal SA. Some NLRs are intrinsically temperature sensitive, and their nuclear accumulation and/or activities are reduced at elevated temperature. The reduced expression of the SA pathway genes, such as *ICS1*, *EDS1* and *PAD4*, is caused by a decrease in their transcription factor CBP60g, whose transcription requires GDAC mediated by the temperature-sensitive GBPL3 to recruit MED and POL II. Heat-resilient resistance can be restored through constitutive expression of *CBP60g* whose translation is made pathogen-inducible by the inclusion of the $uORF_{TBF1}$ 5′ leader sequence in the transcript to minimize fitness cost. Illustration credited to Dr. Xing Zhang of Duke University.

sequence of *TBF1* ($uORF_{TBF1}$), a heat shock factor gene whose translation is activated upon pathogen challenge,[12] they are able to restore normal growth in the *35 S:uORF $_{TBF1}$-CBP60g* transgenic line while maintaining its enhanced resistance at elevated temperature. The conservation of *CBP60g* suggests that this engineering strategy may be applicable in agriculture to sustain plant immunity against global warming.

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ADDITIONAL INFORMATION

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