Elevated Temperature Disrupts NLR-Mediated Calcium Influx and Compromises Effector-Triggered Immunity

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ABSTRACT (<300 words)

Climate change threatens crop health by weakening immune responses to pathogens. Effector-triggered immunity (ETI), driven by NLR receptors, is particularly vulnerable to elevated temperatures. While some NLRs remain functional under heat stress, others lose activity, but the basis for this variation remains unclear.

We used whole-plant imaging of Arabidopsis expressing genetically encoded calcium biosensors to monitor cytosolic Ca^{2+} dynamics during ETI under permissive (22 °C) and elevated (28 °C) conditions. Elevated temperature consistently suppressed Ca^{2+} influx for NLRs that exhibited heat-sensitive ETI.

Both TIR- and CC-type NLRs showed examples of temperature resilience and sensitivity, indicating that subclass alone does not dictate thermal vulnerability. These findings suggest a key event upstream of Ca^{2+} influx is a temperature-sensitive checkpoint in ETI signaling.

This work advances our understanding of how rising temperatures impact plant immunity and identifies calcium signaling as a critical target for preserving disease resistance in crops under climate stress.

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