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Building Heat-Resilient Reproduction: The *XBAT31–HSFB2a/B2b* Module in Reproductive Thermotolerance

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Abstract

Reproductive development is particularly vulnerable to heat stress (HS), yet the ubiquitin-mediated regulatory mechanisms that protect reproductive tissues during thermal stress remain poorly understood. Recent work identifies the Arabidopsis E3 ligase *XBAT31* as a positive regulator of reproductive thermotolerance by promoting ubiquitin-mediated degradation of the repressors *HSFB2a* and *HSFB2b*. Loss of *XBAT31* reduces *HSF/HSP* expression and fertility under HS, highlighting the *XBAT31–HSFB2a/HSFB2b* module as a promising target for heat-resilient crop engineering.

Keywords: Heat stress; Crop; Reproduction; Fertility; Mechanism

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Main text

In recent years, rising global temperatures made HS a pervasive threat that affects nearly all aspects of life. Plant reproductive development is a tightly coordinated process highly vulnerable to temperature disturbances during this developmental period. Although vegetative tissues in crops such as rice, wheat, maize, and tomato often demonstrate transcriptional or physiological plasticity under HS, reproductive development remains notably sensitive. Reproductive vulnerability to HS, manifesting as reproductive developmental abnormalities, leads to disrupted tapetum development, impaired pollen wall formation, reduced pollen viability, inhibited anther dehiscence, embryo abortion, sterile siliques, defective fertilization, and impaired seed development [1, 2]. Considerable advances have been made in elucidating thermotolerance mechanisms in vegetative tissues, particularly involving *HEAT SHOCK TRANSCRIPTION FACTORS (HSFs), HEAT SHOCK PROTEINS (HSPs),* phytochrome signaling, and ubiquitin-mediated protein degradation pathways. Nevertheless, our understanding of the mechanisms that shape specific adaptations to safeguard reproductive tissues during thermal extremes associated with HS remains fragmented and insufficient.

Plant thermomorphogenesis is tightly regulated by modulating the abundance of key proteins, such as the *Arabidopsis B-Box* (*BBX*) protein family, *HSPs, HSFs, PHYTOCHROME INTERACTING FACTORs (PIFs), EARLY FLOWERING 3 (ELF3),* and *CONSTITUTIVELY PHOTOMORPHOGENIC 1 (COP1),* which coordinate transcriptional and post-translational responses to temperature cues, offering optimal plant growth and development under elevated ambient temperatures [3]. For instance, Zhang et al., [4] identified a monomeric RING-type E3 ubiquitin ligase, *XB3 ORTHOLOG 1 IN ARABIDOPSIS THALIANA (XBAT31)*, as a positive regulator of thermomorphogenesis that promotes *ELF3* degradation via ubiquitination at elevated temperatures in *Arabidopsis*. This process requires *BBX18*, highlighting the post-translational regulatory mechanism of *ELF3* in response to heat. HSFs are critical regulators of plant thermotolerance. In eukaryotes, HS activates the heat shock response, which involves the expression of various HSPs regulated by HSFs. In plants, HSFs primarily act as transcriptional activators (class A HSFs) and repressors (class B HSFs) of the heat shock response [5]. A recent study by Zhang et al., [6] shed new light on how *XBAT31*, through its *E3* ligase activity, functions as a key modulator of reproductive thermotolerance in *Arabidopsis by* ubiquitination and degradation *of class B HSFs, HSFB2a* and *HSFB2b*. This regulatory axis acts as a molecular safeguard, ensuring the appropriate activation of thermotolerance pathways specifically in reproductive tissues under HS (Figure 1).

XBAT represents a small thermoresponsive subfamily of E3 RING-type ligases with five members (*XBAT31–XBAT35*) in *Arabidopsis*, which are structurally and functionally related to the rice *XB3* family [4]. Recently, Zhang et al., [6] found that the loss-of-function mutants *xbat31-1* and *xbat31-2* exhibited a marked increase in sterile siliques under HS (subjected to 38 °C), indicating compromised fertility. Furthermore, the authors provide compelling genetic evidence for the essential role of *XBAT31* in reproductive heat resilience by the transcriptional activation, and the loss-of-function mutant *xbat31-1*. Transcriptome profiling revealed the transcriptional suppression of several HSF (*HSFA2, HSFB2a,* and *HSFB2b*) and HSP (*HSP15.7, HSP17.6 A,* and *HSP17.8*) genes in *xbat31-1* under HS.

Moderate HS induces acquired thermotolerance. It is sustained through transcriptional memory by HS memory-related genes, such as *HSFA2* and *HSFA3*, via heteromeric complexes with additional *HSFB2a* and *HSFB2b* [7, 8]. For instance, the expression of *HSFA2* and *HSFB2b* and *HSP17.6A, HSP17.8, and HSP23.6* is induced by elevated temperatures in *the hsfb2b* mutant [9]. However, the expression of *HSFB2a* and *HSFB2b* was upregulated by HS in flower buds in wild-type *Arabidopsis*, indicating their role at the reproductive stage. Phenotypic analysis of *HSFB2a* and *HSFB2b* in relation to thermotolerance in reproductive tissues showed that *hsfb2a–hsfb2b* double mutants retained greater silique fertility. Furthermore, the expression of *HSP15.7, HSP16.7A HSP17.8, HSFA2, HSFB2a,* and *HSFB2b* was upregulated in *hsfb2a hsfb2b* double mutants compared to that in the *xbat31-1* mutant, that showed antagonistic expression patterns. This view reinforces the idea that *HSFB2a/HSFB2b* act as negative regulators of reproductive thermotolerance and suppress the expression of HS-responsive genes [6].

Mechanistically, Zhanget al., [6] demonstrated that *XBAT31* physically interacts with *HSFB2a* or *HSFB2b in vivo* and *in vitro* (yeast two-hybrid and pull-down assays). In addition, *in vivo* analysis confirmed that *XBAT31* interacts with *HSFB2a* and *HSFB2b* in the nucleus, consistent with the subcellular localization of these proteins, as previously demonstrated by Zhang, Shao, Ding, Wang, Davis and Liu [4]. *In vitro* ubiquitination assays further revealed that *XBAT31* polyubiquitinates *HSFB2a* and *HSFB2b* in a RING domain-dependent manner, leading to their proteasomal degradation. Overexpression of *HSFB2a-myc/HSFB2b-myc* resulted in a heat-sensitive

phenotype at the reproductive stage and a higher percentage of sterile siliques. Notably, the protein levels of *HSFB2a/HSFB2b* remained elevated in *xbat31-1* mutant backgrounds even under prolonged heat exposure, whereas transcript levels remained unchanged, indicating post-translational regulation [6].

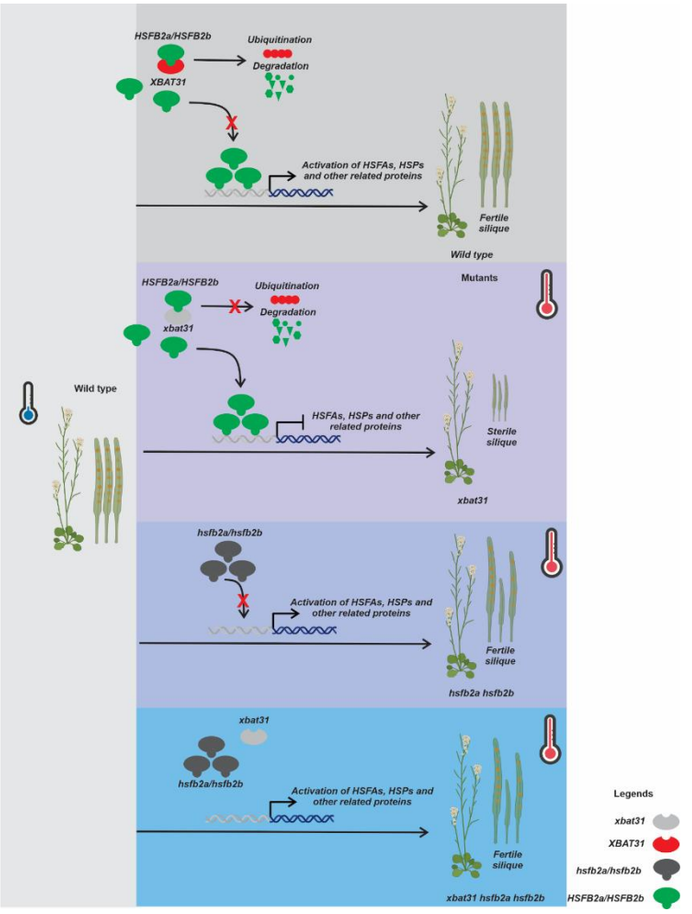

Figure 1. The *XBAT31 HSFB2a/HSFB2b* module is integral to sustaining fertility under HS. *XBAT31* functions as a pivotal regulator of thermotolerance during the reproductive phase by inhibiting *HSFB2a/HSFB2b* through their ubiquitination and subsequent degradation. This inhibition alleviates downstream suppressive effects on HSFA, HSP, and other HS-related genes that are critical for thermotolerance during the productive stage. Conversely, *xbat31* mutant plants demonstrate an increased accumulation of *HSFB2a/HSFB2b*, leading to the suppression of downstream HS-responsive genes and a subsequent reduction in fertility under HS.

To investigate the epistatic relationship between *XBAT31* and *HSFB2a/HSFB2b*, Zhang et al., [6] generated an *xbat31 hsfb2a hsfb2b* triple mutant by crossing, single (*xbat31*) and double (*hsfb2a hsfb2b*) mutants. Phenotypic observation at the reproductive stage revealed enhanced HS sensitivity in *xbat31* with more sterile siliques than the thermotolerant double mutant and triple mutant, which had more fertile siliques. Notably, double and triple mutants exhibited similar HS sensitivity, indicating that *xbat31* is hypostatic to double mutants in regulating reproductive thermotolerance in *Arabidopsis*.

These findings delineate *XBAT31–HSFB2a/HSFB2b* as a tractable target for the engineering of thermotolerant crops. This may allow the reconfiguration of their interaction interface or enhancement of *XBAT31* activity specifically at the reproductive stages, enabling spatiotemporally restricted HSP induction under HS. A key step is to transform this module by CRISPR/Cas-mediated editing or transgenic overexpression in major agronomic crops, such as rice, wheat, and maize, where reproductive HS is a major driver of yield loss. Given the broad functional conservation of HSFs and E3 ubiquitin ligases, this pathway may represent a transferable engineering node.

In summary, Zhang et al., [6] identified a novel thermotolerant regulatory module in *Arabidopsis* that integrates HS perception with protein turnover to protect reproductive development. By linking E3 ligase-mediated degradation of HSF repressors to the activation of stress-inducible genes, this study highlights how plants maintain fertility under challenging environmental conditions. The elucidation of the *XBAT31–HSFB2a/HSFB2b* pathway not only deepens our mechanistic grasp of thermotolerance but also opens translational opportunities for safeguarding crop productivity in an era of climate volatility.

Data availability

Not applicable

Acknowledgments

Not applicable

Conflict of interest

The authors declare that they have no conflict of interest.

Author Contribution

The authors confirm contribution to the paper as follows: study conception and design: SB; data collection: SB, AHW; figure preparation: SB; draft manuscript preparation: SB, AHW; manuscript revision: SB, AHW. All authors discussed the content, reviewed the results and approved the final version of the manuscript.

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