

The heat is on: hSFA1 drives regeneration after wounding

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Plants can repair damaged tissues and regenerate new organs after injury by reprogramming the developmental fate of somatic cells. In both natural and in vitro contexts, this process depends on extensive cellular reprogramming driven by the integration of wound signals, hormone pathways, and chromatin-based regulation (Ikeuchi et al. 2019). Classical root meristem regulators also play central roles in regeneration, highlighting how plants redeploy developmental programs after damage (Sugimoto et al. 2010; Kareem et al. 2015). A hallmark of this response is callus formation, which generates a mass of cells containing pluripotent cells that can later acquire new identities. Under tissue culture conditions, exogenous auxin and cytokinin can guide these pluripotent cells toward root or shoot formation. Still, how plants translate wound perception into the early transcriptional events that establish regenerative competence remains incompletely understood.

In recent work, Duncan Coleman and colleagues (Coleman et al. 2026) identify the Arabidopsis HSFA1 family of heat shock transcription factors (Ohama et al. 2016) as an early wound-responsive transcriptional hub that promotes regenerative competence. Through RNA-seq analysis of wounded tissues, they uncovered a transcriptional program enriched for heat stress response genes, including HSFA1 targets. The authors further show that wounding rapidly promotes nuclear accumulation of HSFA1 proteins, identifying this as an early event in the regenerative response. Genetic analyses of loss-of-function mutants (*hsfa1 abde*) and overexpression lines demonstrated that callus formation after wounding depends strongly on HSFA1 activity: mutants formed less callus, whereas plants with elevated HSFA1d activity showed enhanced regeneration (Fig. 1a). Taken together, these findings place HSFA1 upstream of the transcriptional program required for callus formation and shoot regeneration.

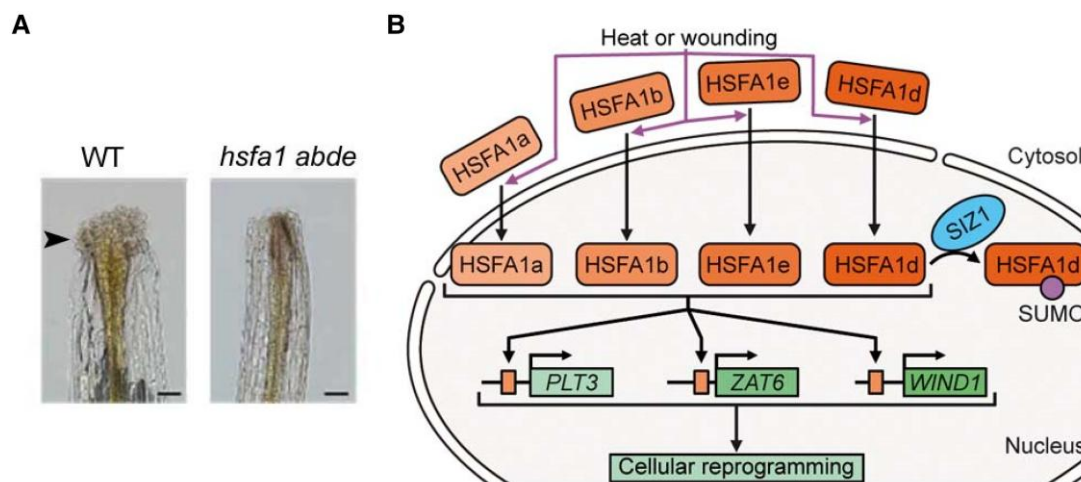


Figure 1 HSFA1 functions in the regenerative response after mechanical damage: a) callus formation after wounding in hypocotyls from wild-type (WT) plants or the *hsfa1* quadruple mutant. WT plants show greater callus formation. Black arrowhead indicate callus formation in WT. b) Schematic model of the signaling pathway activated after wounding, involving HSFA1 and the downstream regulation of PLT3, ZAT6, and WIND1. Adapted from Coleman et al. (2026).

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By combining time-series transcriptomics with chromatin-binding analyses, the authors place HSFA1 directly upstream of key reprogramming regulators, including WIND1, PLT3, and ZAT6 (Fig. 1b). The resulting regulatory network connects wound-induced stress signaling to the gene expression changes that reset cellular identity. The authors also identify SIZ1-mediated SUMOylation as a mechanism that attenuates HSFA1d activity, adding an important post-translational layer of control to the model (Fig. 1b). Together, these findings establish HSFA1 as a central integrator of wound signaling and cellular reprogramming and provide a mechanistic framework for understanding how stress-responsive transcription factors promote regeneration in plants.

Recent related articles in *The Plant Cell*

- Xu et al. (2024) demonstrated that WRKY23 and bHLH041 regulate callus pluripotency during in vitro regeneration in Arabidopsis.
- Wittmer et al. (2025) proposed a strategy for induced regeneration through somatic embryogenesis without exogenous phytohormones and directly addressed regenerative recalcitrance in Arabidopsis and pepper.

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Conflicts of interest

None declared.

Data availability

No new data were generated or analysed in support of this research.

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