

FKF1: A central signaling hub integrating multi cues to flowering in plants

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Floral transition determines plant reproductive success and crop productivity. The blue-light receptor FKF1 serves as a central signaling hub for flowering regulation. Although its function in photoperiodic flowering is well characterized, the molecular mechanisms underlying FKF1-mediated temperature and nutrient responses, as well as crop latitudinal adaptation, remain poorly defined. We summarize recent progress on FKF1-dependent flowering regulatory pathways across plant species. FKF1 integrates multi-environmental signals through liquid-liquid phase separation, conserved FKF1-GI modules and epigenetic modulation, with distinct functional features between monocots and dicots. Natural functional alleles of FKF1 substantially enhance crop environmental adaptability and yield, offering promising genetic resources for crop improvement and sustainable agricultural development.

In the plant life, flowering represents a pivotal developmental transition. The temporal regulation of flowering transition not only directly determines plant survival and reproductive fitness, but also exerts a profound impact on crop yield and quality, which serves as a core physiological mechanism underlying plant adaptation to fluctuating environments. As a key blue-light receptor, FLAVIN-BINDING, KELCH REPEAT, F-BOX 1 (FKF1) is well known for its canonical role in mediating photoperiodic flowering transition,¹⁻⁴ yet the regulatory mechanisms governing FKF1-dependent temperature and nutrient responses, as well as its molecular basis for modulating latitudinal adaptation, flowering transition, and yield in crops, have long remained elusive. Recent global research advances have gradually revealed that FKF1 acts as a core signaling integration hub, precisely controlling plant flowering by coordinating multiple molecular pathways, mediating target protein degradation via liquid-liquid phase separation (LLPS) and epigenetic modifications, indicating that FKF1 is an universal switch for plant responses to multiple environmental signals, and laying valuable genetic resources for crop genetic improvement and sustainable agriculture.⁵⁻¹⁰

Nitrogen is a vital macronutrient for plant growth, and moderate nitrogen application boosts crop productivity, but excessive fertilization triggers severe environmental pollution, delays flowering, and disrupts crop rotation systems, with the molecular basis of crop adaptation to high-nitrogen environments largely unknown. Our recent functional characterization of FKF1 has uncovered a novel molecular pathway that addresses this long-standing agricultural challenge.⁵ In monocot crops like rice and wheat, FKF1 forms nuclear condensates via LLPS driven by its N-terminal intrinsically disordered region (IDR), facilitating degradation of the anti-florigen protein FT4 to control flowering in response to nitrogen cues. Under moderate nitrogen, FKF1 condensates efficiently ubiquitinate and degrade FT4 for punctual flowering; under high nitrogen, impaired FKF1 LLPS blocks condensate formation, causing FT4 buildup and late flowering transition. The scaffold protein GI acts as a bridge to recruit FT4 into FKF1 condensates, and weakened FKF1-GI binding under high nitrogen worsens flowering delay (Figure 1A). Notably, the LLPS capacity of FKF1 is highly conserved among monocots, whereas dicot FKF1 homologs lack the N-terminal IDR and cannot undergo LLPS, reflecting the species-specific functional characteristics of FKF1. An alanine-array of IDR confers the capacity of BdFKF1 to undergo LLPS. Mutating the alanine acids to hydrophilic glutamate acids at the IDR results in substantially decreased

condensates of FKF1. Moreover, key SNPs in the *OsFKF1* IDR drive natural variation linked to soil nitrogen adaptation, with distinct haplotypes showing differential nitrogen sensitivity, suitable for developing Kompetitive Allele Specific PCR (KASP) markers in crop breeding.

FKF1 plays a core role in temperature-responsive flowering transition, with clear species-specific mechanisms. In *Arabidopsis thaliana*, FKF1 and GI mediate reversible LLPS to promote the 26S proteasome-dependent degradation of the floral repressor SVP, accelerating flowering transition under high temperatures (Figure 1B).⁸ Notably, AtFKF1 lacks the N-terminal IDR and cannot undergo independent LLPS, relying on GI condensate dynamics instead. This mechanism mirrors the nitrogen response pathway in monocots, as GI retains its molecular bridge function, highlighting the evolutionary conservation of the FKF1-GI module in different species. Moreover, FKF1 connects light perception to epigenetic regulation of floral transition in temperate grass model plants. It mediates light-induced degradation of the floral repressor ELF3, which relieves Polycomb Repressive Complex 2-mediated epigenetic repression of the key flowering gene *PPD1*, allowing plants to adjust flowering transition flexibly in response to light changes (Figure 1C).⁷ This pathway reveals a critical link between light signaling and epigenetic modification in flowering control.

FKF1 homologs also serve as key regulators of latitudinal adaptation and yield improvement in major global crops. In maize, a tropical-origin crop widely domesticated for temperate regions, ZmFKF1a regulates photoperiod sensitivity and growth period via a novel blue-light responsive regulatory module independent of the canonical FT pathway, promoting early flowering transition and breaking the bottleneck of tropical germplasm utilization in temperate zones.⁶ Overexpression of *ZmFKF1a* shortens the growth period of photoperiod-sensitive tropical maize while increasing grain yield, making it a valuable target for breeding widely adaptable high-yield varieties (Figure 1d). Another maize homolog, *ZmFKF1b*, has undergone strong artificial selection in modern breeding, correlating with adaptation to different temperature zones.¹⁰ The haplotypes *ZmFKF1b*^{C7} and *ZmFKF1b*^{Z58}, linked to temperature zones, aid in introducing tropical germplasms to temperate regions and developing adaptable high-yield cultivars (Figure 1D). In soybean, the FKF1 homolog *Tof8* has been identified as a key flowering regulator, with superior alleles enhancing high-latitude adaptability and promoting early flowering transition.⁹ The *GmFKF1b*^{H3} allele accelerates flowering and enhances latitudinal adaptability, offering candidate genes for flowering time regulation (Figure 1D). These findings offer new gene targets for optimizing soybean flowering, maturity and yield across diverse latitudes.

Collectively, recent studies demonstrate that FKF1 integrates light, temperature, and nutrient signals to precisely control flowering transition through LLPS, target protein degradation, and epigenetic regulation. These findings not only clarify the central role of FKF1 in plant environmental adaptation but also identify critical genetic targets for breeding widely adaptable, high-yield, and high-quality crops. Further exploration of FKF1 regulatory networks across more species and environmental contexts will unlock its full breeding potential, offering robust support for global food security and sustainable agricultural development.

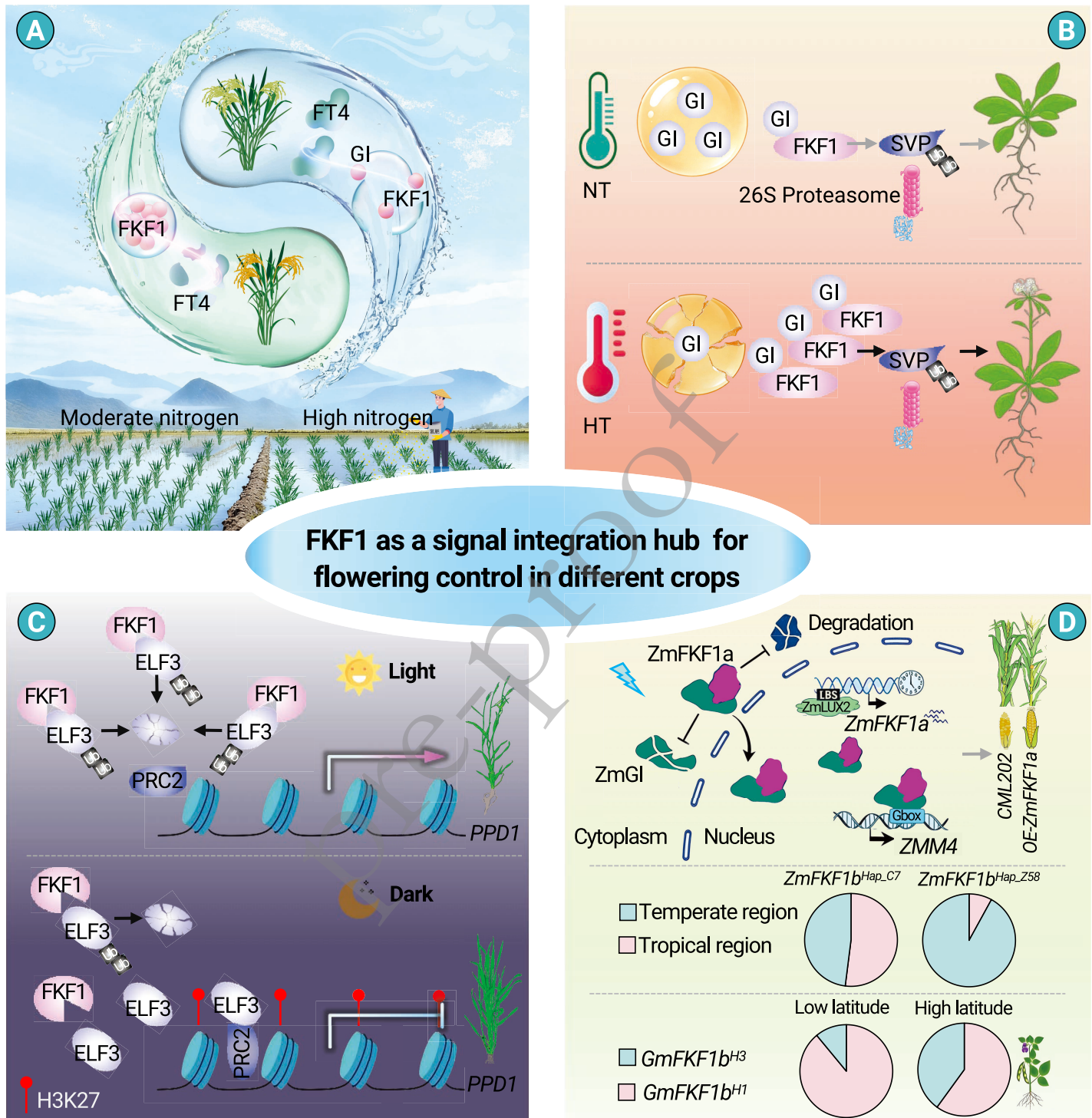


Figure 1. FKF1 integrates multiple signals to regulate flowering transition A Taiji-inspired water droplet diagram shows the dynamic balance between anti-florigen FT4 abundance and FKF1 LLPS activity under distinct nitrogen conditions in rice, driving the “stay-green and delayed maturation” phenotype under HN condition. B Working model of FKF1-GI complex regulating high temperature-responsive flowering transition in *Arabidopsis*. C Proposed working model of FKF1-ELF3-PRC2 module regulating flowering under light/dark conditions in temperate grasses. D (Upper) Working model of ZmFKF1a-ZmGI complex regulating maize early flowering. (Middle) ZmFKF1b underwent artificial selection during maize inbred breeding, causing significant haplotype differentiation. (Bottom) Distinct *GmFKF1b* alleles drive latitudinal adaptation of cultivated soybean.

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AUTHOR CONTRIBUTIONS

Y.L. and P.H. conceived and supervised the research. Y.L. and H.Y. wrote the article. All authors contributed to the manuscript and approved the final version.

DECLARATION OF INTERESTS

The authors declare that they have no conflict of interest.

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