Integration of flowering time signals in *Arabidopsis thaliana*

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The induction of flowering is a central event in the life cycle of plants. When timed correctly, it helps ensure reproductive success, and therefore has adaptive value. Because of its importance, flowering is under the control of a complex genetic circuitry that integrates diverse endogenous signals (hormones, carbohydrates) and environmental signals such as temperature and light, in particular day length (photoperiod). Under inductive photoperiod, *FLOWERING LOCUS T* (*FT*), a key regulator of flowering time in *Arabidopsis thaliana*, is induced in the leaf vasculature and acts as a long distance signal (florigen) that transmits the information to initiate flowering from leaves to the shoot meristem.

The disaccharide trehalose-6-phosphate (T6P) has been shown to act as signaling molecule in coordinating carbohydrate status with developmental processes. *Arabidopsis thaliana* plants deficient in the T6P-synthesizing enzyme TREHALOSE-6-PHOSPHATE SYSNTHASE 1 (TPS1) flower extremely late. We show that TPS1/T6P signaling regulates the expression of several flowering-time genes throughout the plant. In the leaf vasculature T6P is absolutely required for the induction of *FT* [*1*]. Our results demonstrate that environmental (photoperiod) and physiologic (TPS1/T6P) signals converge on *FT*. Together these two inputs ensure that flowering commences only when the conditions are optimal, that is, day length exceeds a certain minimum and the carbohydrate state supports the energy-demanding transition to flowering and seed production.

Apart from photoperiod and TPS1/T6P, temperature also has a marked effect on *FT* expression. However, relatively little is known about how flowering is controlled by ambient temperature. Genetic analyses have identified a number of genes involved in temperature-dependent regulation of flowering. In particular the MADS-box transcription factors *SHORT VEGETATIVE PHASE* (*SVP*) and *FLOWERING LOCUS M* (*FLM*) have been shown to play key roles in this process. We could recently show that two splice variants of *FLM* regulate flowering in opposition [*2, 3*]. Genetic, molecular and splice variant-specific ChIP-seq analyses revealed that the proteins encoded by the two splice variants compete for interaction with the floral repressor SVP. The SVP/FLM-β complex is predominately formed at low temperatures, when expression of FLM-β is strongest, and actively prevents precocious flowering. In contrast, the competing SVP/FLM-δ complex is impaired in DNA binding and acts as a dominant negative activator of flowering at higher temperatures. Our findings reveal a novel mechanism that modulates the timing of the floral transition in response to changes in ambient temperature.

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