Upstream regulatory mechanisms of a cold-induced epigenetic switch in plants

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Abstract

Proper timing of the flowering transition in plants is a key developmental process necessary for adaptation to various ecological niches. Many plant species require exposure to the prolonged cold of winter to flower, a temperature-sensing process referred to as vernalization. In *Arabidopsis thaliana*, prolonged exposure to cold epigenetically represses the expression of a floral repressor, *FLOWERING LOCUS C* (*FLC*). This cold-mediated repression occurs through an evolutionarily conserved Polycomb repressive complex 2 (PRC2) associated with the vernalization-unique protein VERNALIZATION INSENSITIVE 3 (VIN3) and long noncoding RNAs (lncRNAs) complementary to *FLC*. The vernalization-specific PRC2 complex results in increased deposition of an epigenetic silencing mark H3K27me3 at FLC chromatin. However, the system by which cold perception leads to the induction of vernalization-specific factors involved in *FLC* targeting is largely unknown. In this study, we describe the first example of a vernalization-hypersensitive mutant in which the period of cold exposure required to saturate the vernalization response is shortened. This is also the first example of a potential upstream regulator of VIN3 and lncRNAs complementary to *FLC*. The causative locus encodes a methyltransferase, SET DOMAIN GROUP7 (SDG7), that is required for proper timing of expression of vernalization specific factors in cold-induced polycomb silencing (1). In summary, our work is an important contribution to the understanding of how environmental signals result in epigenetic gene silencing.