Mechanism for winter cold-mediated epigenetic silencing of a potent floral repressor in plants

Subject Code: C02

With the support by the Chinese Academy of Sciences (CAS), the research team of Plant Environmental Epigenetics led by Prof. He Yuehui (何跃辉) at the Shanghai Center for Plant Stress Biology, CAS and CAS Center for Excellence of Molecular Plant Sciences, discovered a molecular epigenetic mechanism underlying how winter cold enables plants to flower in spring, which was published in *Nature Genetics* (2016, 48: 1527—1534).

Plants are sessile and must adapt to local environments to complete their life cycles. In response to environmental stimuli or changes, plants exhibit developmental plasticity and adjust their growth and development accordingly. One classic example is vernalization: plants acquire competence to flower in spring after exposing to winter cold, a seasonal temperature drop in subtropical and temperate regions. Plants typically experience winter at an early developmental stage. When temperature rises in spring, the "memory of winter" lasts and is transmitted throughout cell divisions in an *epi*-genetic manner. This enables plants to flower and set seeds.

Winter cold or prolonged cold exposure overcomes a blocker to flowering. In the model flowering plant *Arabidopsis thaliana*, a MADS-box transcription factor FLOWERING LOCUS C (FLC) is a central repressor of flowering, and winter cold silences *FLC* expression to enable flowering. This silencing is carried out by a group of chromatin modifiers, Polycomb group proteins (PcG), assembled into protein complexes to introduce repressive chromatin modifications at target loci, leading to transcriptional repression. Cold triggers the enrichment of a PcG complex known as PRC2 in a small region (nucleation region) at the *FLC* locus, to catalyze the repressive histone 3 lysine-27 trimethylation (H3K27me3). After plants return to warm temperatures, the H3K27me3 mark is stably maintained and further spread to cover the entire *FLC* locus. During subsequent growth and development at warm temperatures, the PcG-mediated silenced state of *FLC* is transmitted throughout cell divisions, and thus enables plants to flower at a late stage when the timing is right. Hitherto, how H3K27me3 and Polycomb silencing are established in the nucleation region at *FLC* during vernalization is unknown.

He's Group first identified a *cis*-regulatory DNA element in the nucleation region of PcG silencing at *FLC*, termed as *Cold-Memory Element* (*CME*). CME contains two identical motifs that are specifically recognized and bound by a homodimer of two homologous transcription factors known as VAL1 and VAL2. In addition to recognizing CME, VAL proteins also read the H3K27me3 mark; moreover, they recruit another H3K27me3 reader LIKE HETERCHROMATIN PROTEIN 1 (LHP1) that directly interacts with an H3K27 methyltransferase complex (PRC2). CME-VAL-LHP1-PRC2 establishes the H3K27me3 peak in the nucleation region during vernalization through positive feedbacks, and functions to maintain H3K27me3 in mitotic cell divisions after return to warm temperatures. Uncovering of this novel molecular epigenetic mechanism for flowering control by winter cold would enable genetic manipulations of flowering times in crops to broaden their seasonal adaptability.

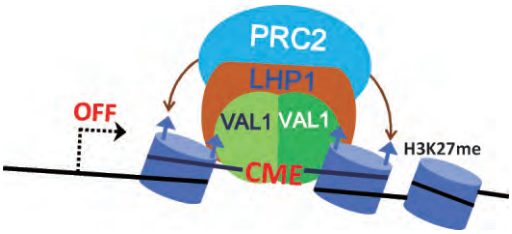

Figure The *cis*-acting cold memory element (*CME*) and a *trans*-acting reader VAL1 function together with Polycomb partners LHP1 and PRC2 to establish H3K27me3 and Polycomb silencing at *FLC* in response to winter cold.

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