Temperature Compensation of the Circadian Clock: A Role for the Morning Loop

Circadian clocks have a remarkable capacity for temperature compensation, which renders clock output largely insensitive to environmental temperature fluctuations (i.e., providing for constant periodicity in the face of wide swings of temperature that most organisms experience daily and seasonally).

The circadian clock in Arabidopsis is thought to be controlled by multiple interlocking feedback loops. The central loop contains the morning-expressed MYB genes CCA1 and LHY, which negatively regulate (and are in turn positively regulated by) the evening-expressed TOC1. This is influenced by a morning loop, including the PSUEDORESPONSE REGULATORS PRR5, PRR7, and PRR9, which repress the expression of CCA1 and LHY, and an evening loop, containing GIANTEA (GI) and ZEITLUPE (ZTL), which feed back to regulate TOC1 (see recent reviews in Harmer, 2009; Pruneda-Paz and Kay, 2010). It has previously been proposed that feedback effects of GI and other evening loop genes, such as ZTL, can largely account for temperature compensation in Arabidopsis (Gould et al., 2006). Now, Salomé et al. (pages 3650–3661) show that the morning loop components PRR7 and PRR9 also play a key role in temperature compensation.

The authors examined prr7 prr9 double mutants at a wide range of temperatures and found a complex clock-defective phenotype. In wild-type plants, period length shortens slightly as plants are exposed to increasing temperatures. By contrast, the period of the prr7 prr9 double mutant lengthened with increasing temperatures and became very long (>35 h) at 30°C (see figure). This temperature overcompensation at high temperatures was not seen in a variety of other clock mutants tested, including the prr5 prr7 double mutant and variety of single mutants of GI, ZTL, LHY, and TOC1.

Because the morning loop genes PRR7 and PRR9 exert a negative effect on the central loop genes CCA1 and LHY, the authors used artificial microRNAs to knock down CCA1 and LHY expression in the prr7 prr9 mutant background. Inactivation of CCA1 and LHY fully suppressed the overcompensation defects in these mutants, and the circadian period remained constant between 12 and 30°C. This suggests that the morning loop is part of the temperature compensation mechanism regulating the activity of central loop components CCA1 and LHY.

In a further set of experiments, the authors knocked down PRR7 and PRR9 with tandem artificial microRNAs in a number of Arabidopsis accessions. Interestingly, the overcompensation phenotype was observed in all accessions tested, including those with weak alleles of FLOWERING LOCUS C (FLC), which was previously shown to be required for temperature compensation at high temperatures (Edwards et al., 2006). This suggests that the overcompensation phenotype of prr7 prr9 mutants is not due to altered FLC activity, and the role of the morning loop in temperature compensation is largely independent of FLC.

This work enhances our understanding of the circadian clock by establishing a role for PRR7 and PRR9 in temperature compensation, specifically in regulating the central clock components CCA1 and LHY in response to changing ambient temperature.

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*Plant Cell* 2010;22;3506; originally published online November 23, 2010;
DOI 10.1105/tpc.110.221111

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