Molecular Basis of the ABA Dependent Modulation of CONSTANS Activity in Drought Escape Response

Alice Robustelli Test, Matteo Riboni, Sara Castelletti, Massimo Gabbiati, Elirini Kaiserli, Chiara Tonelli and Lucio Conti

Institute of Molecular Cell & Systems Biology, University of Glasgow (UK)

The drought escape (DE) response allows some plants to adaptively shorten their life cycle to make seeds before severe stress leads to death. In Arabidopsis, DE occurs under long day conditions (L.Ds), when photoperiod-stimulated GIGANTEA (GI) promotes the transcriptional activation of the florigen gene FLOWERING LOCUS T (FT) and TWIN SISTER OF FT (TSF). The phytohormone ABA participates in this process in an unknown manner, upstream of the florigen genes. A key question is how does ABA activate the florigen genes? Here we use ABA signalling and photoperiod perception mutants to demonstrate that ABA transduces water status information upon the florigen gene FT by stimulating GI signalling and CONSTANS (CO) activity.

We have previously shown that Arabidopsis wild-type plants low watering (LW) conditions promote flowering compared to normal watering (NW) (a) as a result of a drought-dependent upregulation of the florigen genes FT and TSF, specifically under LD conditions. No FT and TSF upregulation occurs in gi mutants at any time points, whereas in aba1 mutants transcript accumulation of the florigen genes is reduced, especially under LW (b).

Unlike FT, TSF is upregulated in mutants of co under drought conditions (a,b). Mutants of cfd, characterised by increased levels of CO, produce a DE response comparable to wild type under LDs (c). Pointing to a specific role of GI in activating DE is the observation that cfd mutants are DE-insensitive under LDs. The pattern of FT upregulation reflects the DE response in these genotypes (d). We conclude that transduction of ABA signals onto FT promoter requires both photoperiod-stimulated GI and CO.

We sought to elucidate the role of ABA upstream of FT, whether the canonical ABA signalling cascade could be involved and whether ABA had any effect on GI/CO functions. As expected, in the Ler wild-type background FT levels (but surprisingly not TSF) were increased under LW (a), Mutants impaired in ABA signalling (aba1-1 or with reduced ABA accumulation (aba1-1) displayed a general reduction in FT TSF accumulation under LW. The pattern of CO transcript accumulation was unaffected in both ABA-defective backgrounds. Increased FT/TSF levels were apparent in 35S:GI plants as a result of CO upregulation. Such phenotype was strongly suppressed in 35S:CO abi1-1 double mutants without a comparable decrease in CO accumulation. ABI1promoter::GUS fusions revealed that ABI1 expression overlaps with FT and the site of ABA biosynthesis in the phloem (b).

We conclude that ABA may stimulate a specific aspect of GI function responsible for FT/TSF genes transcriptional activation. ABA in conjunction with photoperiod-stimulated GI positively affects CO activity rather than CO transcription accumulation to promote FT transcriptional activation. We thus tested the hypothesis that ABA accumulation and signalling could be necessary for CO function.

Overexpression of CO under the 35S promoter cannot fully rescue the aba1-6 flowering phenotype compared to the wild type (Col-0) in independent transgenic plants (a,b). In a complementary approach, when overexpressed in plants with enhanced ABA signalling (abi1 abi2 hab1/1), CO causes an extreme early flowering phenotype in half of the T1 plants (c,d). These genetic data support the notion that ABA promotes CO activity and/or stabilization. CO and GI co-localize in the nucleus in association with the blue light receptor FK1 (e). Since FK1 promotes CO accumulation, ABA might affect the formation of the Co-GI-FKF1 complex and thus CO accumulation. In summary, our data suggests a role for ABA in altering photoperiodic perception and signaling through modulation of CO function in a specific temporal window where GI and blue light photoreceptors (e.g. FK1) are active (f).