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Light responses in Photoperiodism in Arabidopsis thaliana

ADO1: An Arabidopsis blue light photoreceptor

We have reported the characterization of an Arabidopsis gene encoding the ADAGIO 1 (ADO1) protein (Jarillo et al., 2001a). ADO1 contains a LOV domain, similar to WHITE COLLAR 1 (WC1), a photoreceptor for entrainment of Neurospora circadian rhythms (Froehlich et al., 2002), as well as PHOT1 and PHOT2, the blue light photoreceptors for phototropism (Briggs et al., 2001; Christie et al., 1998; Jarillo et al., 2001b; Kinoshita et al., 2001). Loss of function *ado1* mutants show an unusually long periodicity for their free running circadian rhythm (Jarillo et al., 2001a). This observation holds for plants grown under white light as well as blue light and surprisingly, plants grown under red light also show altered circadian properties.

The similarity of the LOV domain of ADO1 to those of PHOT1, PHOT2 and WC1 (known flavoprotein photoreceptors) as well as the genetic and molecular properties of ADO1, indicate that ADO1 is likely a new class of blue light photoreceptor. Indeed, the LOV domain of the related FKF1/ADO3 has been shown to bind FMN, and exhibit the *in vitro* photochemistry characteristic of PHOT1 (Imaizumi et al., 2003). Furthermore, ZTL/ADO1 has been shown to participate in the circadian and proteasome mediated degradation of the Arabidopsis clock protein, TOC1 (Mas et al., 2003).

We also showed that the *ado1* mutation selectively confers hypersensitivity to red light — when grown under red light (but not blue light) the *ado1* mutant possesses an unusually short hypocotyl. This red light hypersensivity is even more severe in a triple *ado1 ado2 ado3* mutant — *ADO2* and *ADO3* being the two other members of this ADAGIO gene family. This finding of a mutant phenotype under red light is somewhat unexpected for a protein thought to function as a photoreceptor for blue light.

We have pursued our studies of ADO1 by preparing a mutant gene for which we have altered the codon for the cysteine residue conserved in all LOV domains. It is this cysteine residue that forms a covalent adduct with the bound flavin in the photocycle of PHOT1 and FKF1. In the mutant ADO1 this cysteine is replaced by

an alanine. We argue that if ADO1 functions as a photoreceptor in a similar fashion to PHOT1, then this mutant ADO1 should not be able to rescue the altered circadian phenotype of *ado1* mutant plants. We find under white light, that indeed this is the case. Experiments underway are aimed at determining if the altered circadian phenotype under blue and red light are similarly unable to be rescued by the mutant gene, and we are performing similar experiments under red light with respect to the defect in hypocotyl elongation. The results from these experiments will likely support the hypothesis that ADO1 functions as a blue light photoreceptor, and they will address the question concerning whether or not the altered properties of the *ado1* mutant under red light are also a reflection of this photoreceptor function.

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