

CSK as a regulator of chloroplast gene expression under changing light intensities

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Two-component signaling systems involving a sensor histidine kinase and its cognate response regulator are wide spread in most prokaryotes, including cyanobacteria. In this regulatory system, an environmental stimulus provokes a trans-autophosphorylation event in which one sensor kinase monomer phosphorylates a conserved histidine residue in the second sensor kinase monomer. This phosphorylated sensor kinase then transfers the phosphoryl group to its cognate response regulator, altering the conformation of its effector domain, thus provoking a physiological response. A single modified version of this two-component system has been reported in the chloroplasts of *Arabidopsis thaliana*. In this system, Chloroplast Sensor Kinase (CSK) no longer contains the conserved histidine residue and does not appear to have a true cognate response regulator partner. CSK has been shown to regulate chloroplast gene expression by reporting on the redox state of the plastoquinone pool to chloroplast gene expression machinery, utilizing an iron-sulfur cluster within its sensor domain. Preliminary fluorescence data taken with a MultispeQ v1 show CSK mutants have altered photosynthetic efficiency compared to wildtype. Additionally, a CSK knockout mutant appears to have increased transcript abundance of certain chloroplast genes whose transcription is initiated by sigma factor (SIG5) specifically under high light. This effect appears to be most notable later in the day, suggesting that CSK is necessary for proper down regulation of transcription to maintain balance of cellular resources in preparation for darkness. Establishing a role for CSK in stress response and energy homeostasis represents a significant advancement in understanding the function of this elusive sensor kinase.

Figure.1. Hypothesis: When the plastoquinone pool (PQ) is reduced by high-light or other abiotic stresses, CSK is activated. Phosphorylation of chloroplast sigma factor(s) by activated CSK decreases initiation of transcription at specific gene operons to regulate the use of cellular energy and resources.

