Characterization of Diatom Chloroplast Response Regulator 1 (CRR1)

Laura N. Hiotaky and Sujith Puthiyaveetil

Department of Biochemistry and Center for Plant Biology, Purdue University, West Lafayette IN 47907, lhiotaky@purdue.edu

Phaeodactylum tricornutum is a unicellular marine alga belonging to a family of photosynthetic eukaryotes called diatoms. Diatoms inhabit marine environments and are subjected to many environmental fluctuations, including the quantity of light. Nevertheless, diatoms display high photosynthetic productivity. Thus, it is crucial to understand the biochemical mechanisms of this photosynthetic acclimation, as diatoms are a major target for biofuel production and agricultural engineering. Diatoms trace their evolutionary origin to red algae through a secondary endosymbiotic event. Diatoms thus carry protist, red algal, and prokaryotic genetic material. One such conserved prokaryotic genetic material is the two-component system (TCS). The TCS consists of a sensor histidine kinase and a response regulator. The sensor histidine kinase senses an environmental stimulus and autophosphorylates on a conserved histidine residue. The phosphoryl group is then transferred from the kinase to a response regulator, eliciting a gene regulatory response. In diatoms, one of the sensor histidine kinases takes the form of chloroplast sensor kinase (CSK). There are two cognate response regulators of CSK in the form of chloroplast response regulator 1 (CRR1) and chloroplast response regulator 2 (CRR2) that are believed to have arisen from a gene duplication event. We hypothesize that CRR1 and CRR2 regulate plastid genes for heat shock proteins, and that CRR1 and CRR2 are activated during abrupt high temperature and light fluctuations. Here we show correlation between the expression of CRR1 and the expression of the heat shock plastid gene *dnaK* in *Phaeodactylum*. Also, we show preliminary data in the optimization of an anti-CRR1 antibody, which will be used in identification of the environmental conditions that activate CRR1 and gene targets of CRR1.