

OA3.6 - Chloroplast-mitochondria cross-talk in diatoms: A proteomics study of the effects of Cu limitation on global metabolic pathways in an oceanic diatom

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Diatoms are one of the most successful phytoplankton groups in our oceans. They are responsible for over 20% of the Earth's photosynthetic productivity, thus impacting global fisheries, biogeochemical cycles and climate. Their phylogenetic history led to a chimaeric genome with genes derived from the green lineage, the red lineage, bacteria and heterotrophs. These sequential endosymbiosis events led to an increased set of isoenzymes that are targeted to either the same or different cellular compartments and can be regulated in very distinct ways. The novel interactions between metabolic pathways are thought to be one of the reasons for the diatom's global success. In a recent study, we presented how acclimation to Cu limitation affected the photosynthetic electron transport chain of the oceanic diatom *Thalassiosira oceanica* (CCMP 1003), leading to the down-regulation of PSII, *cytb₆f* and plastocyanin and to a potential overreduction of the photosynthetic electron transport chain (Hippmann et al. 2017). In order to understand the overall metabolic changes that help alleviate these stresses, stable isotope dimethyl labelling proteomics was used to compare relative expression of proteins extracted from *T. oceanica* cultures acclimated to limiting or replete Cu. We identified differentially expressed proteins involved in various carbon, nitrogen and stress-related metabolic pathways, and in the case of multiple isozymes, found differences depending on the predicted cellular location. Ferredoxin (Fdx) was highly up-regulated, as were plastidial Fdx-dependent isoenzymes involved in nitrogen metabolism. Furthermore, chloroplast-targeted glutamate and cysteine synthase were up-regulated, supporting glutathione production to counteract reactive oxygen species.

Differential regulation of various glycolytic isoenzymes in the chloroplast and mitochondria support increased channeling of excess electrons from the plastid to the mitochondria, and increased ATP levels in the chloroplast. We propose this to be an effective first line of defence to counteract even slight changes in redox and energy imbalances between chloroplasts and mitochondria. Additional evidence for chloroplast-mitochondrial cross-talk is shown by up-regulation of chloroplast pyruvate kinase and malate dehydrogenase combined with mitochondrial malate dehydrogenase and aspartate aminotransferase, supporting a role for the often proposed malate shunt. The interconnectedness of glycolysis, malate shunt, citrate cycle, and the diatom-specific use of the urea cycle are enhanced under low Cu, whereby carbon skeletons and reducing equivalents are channelled in a directed manner to meet the cellular needs.

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