Thylakoid protein phosphorylation optimises electron transfer under fluctuating light

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In higher plant chloroplasts photosystem II (PSII) and its light harvesting antenna (LHCII) are reversibly phosphorylated by STN7 and STN8 kinases dependent pathways according to the quantity of light. Low light (LL) favors LHCII protein phosphorylation by the STN7 kinase whereas at high light (HL) the LHCII proteins are dephosphorylated. PSII core proteins, on the contrary, are maximally phosphorylated at HL by the STN8 kinase. The aim of the study was to elucidate the role of PSII-LHCII phosphorylation in regulation of the redox state of the photosynthetic electron transfer chain (ETC), possible interactions with non-photochemical quenching (NPQ) and in the repair cycle of PSII. As tools of investigation we used mutant *Arabidopsis thaliana* plants, *stn7* and *stn8*, impaired in short-term acclimation mechanisms. The plants were grown under different white light intensities and under fluctuating light.

STN8 kinase and accordingly the phosphorylation of the PSII core proteins were shown to be essential for dynamic movement of damaged PSII centers from grana to stroma thylakoids for repair.

The *stn7* mutant showed a distinct phenotype of severely retarded growth compared to WT only under fluctuating light. Chlorophyll *a* fluorescence measurements revealed that plants lacking the STN7 kinase have a much higher reduction level of ETC upon low light periods of illumination under fluctuating light compared to wild-type. Similar measurements under fluctuating light were carried out on leaves from plants grown under continuous moderate light: the stn7 mutants are capable of developing a long-term compensation for the missing LHCII protein phosphorylation mechanism, involving a change in photosystem stoichiometry.

We conclude that LHCII phosphorylation, catalyzed by the STN7 kinase, is required to equally distribute the excitation energy between PSII and PSI under fluctuating light conditions and consequently to maintain the redox balance of ETC. Furthermore, it also became clear that the induction of NPQ diminishes the importance of the STN7 kinase.

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