A molecular switch for regulation of photosynthetic light use efficiency in mosses and green algae, named LHCSR

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Light is the energy source for photosynthetic organisms and yet it causes photoinhibition when in excess because unquenched chlorophyll (Chl) excited states yield reactive oxygen species when reacting with O2 (Barber and Andersson, 1992; Miller et al., 2008; Takahashi and Badger, 2011). A major photoprotective mechanism is Non-Photochemical Quenching (NPQ) rapidly dissipating Chl excited states as heat, which prevents photodamage. In vascular plants the fast NPQ component is qE (Energy quenching). It requires PSBS protein (Li et al., 2000) and the two xanthophylls lutein (Lut) (Pogson et al., 1998) and zeaxanthin (Zea) (Niyogi et al., 1998). In the green algae as C. reinhardtii and in lower plants, qE activation relies on the LHCSR proteins (Peers et al., 2009; Alboresi et al., 2010). LHCSR is a chlorophyll (Chl) α-xanthophyll-binding protein binding 8 Chls and 4 xanthophylls per polypeptide. Two Xanthophyll binding sites have strong selectivity for, respectively, Lut (site L1) and violaxanthin (Viola), site L2. Two additional sites, N1 and V1 bind Viola (Pinnola et al., 2017; Bonente et al., 2011). Moss LHCSR is constitutively expressed in P. patens and its activity is controlled by the xanthophyll cycle. The form binding Viola has low activity while is switched to an highly active form upon binding Zea in high light (Pinnola et al., 2013). LHCSR is expressed in low amount and difficult to purify. We obtained sufficient amounts of this pigment-protein complex by overexpressing and purifying a “tagged” version of P. patens LHCSR (PpLHCSR) from transgenic tobacco plants (Pinnola et al., 2015). The availability of the protein allowed for the first time the systematic and comparative study of the inactive and active form by spectroscopic methods and hypothetical models of activation and catalysis of quenching reactions could be verified. In its lowly active form LHCSR has a fluorescence lifetime similar any other LHC protein thus acting as a light harvesting antenna and transferring energy to Reaction Centers (RC) to fuel charge separation. Under excess light conditions thylakoid lumen becomes acidic, leading to two effects on LHCSR: first, Zea is produced from pre-existing Viola and binds to sites L2 and V1; second, the protein is protonated on three acidic residues exposed to the lumen. Together, these events cause LHCSR to switch from light harvesting (3.7 ns) into its quenched (80 ps) conformations, thus effectively competing for excitons with PSII RC and dissipating energy into heat. This model was supported by data from Fluorescence Lifetime analysis in detergent solution, by transient absorption spectroscopy and Single Molecule Spectroscopy (Pinnola et al., 2016, 2017; Kondo et al., 2017).

Full understanding of this molecular switch requires high resolution structural data for the two conformations. At present we are pursuing crystallization for X-ray diffraction analysis and isolation of supramolecular complexes including LHCSR for cryo-electron microscopy. Because of its unique property of including both the protein domains responsible for pH detection and those catalyzing quenching reactions, LHCSR appears to be the best model for elucidation of one of the most enigmatic and elusive aspects of photosynthesis despite the process has been studied for some 50 years already.

References


