The Mechanism of Nonphotochemical Quenching: The End of the Ongoing Debate

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Dear Editor,

Oxygenic photosynthes is created the Biosphere as we know it. It allowed heterotrophic life, including ourselves, to flourish and supported it for millions of years. However, the delicate oxygenic photosynthetic machinery is susceptible to damage due to occasional, periodic, or constant exposure to intense light. Excess light causes absorption of too many photons by the light-harvesting antenna and overexcitation of its pigments that can damage the photosynthetic membrane, particularly the components of the oxygen-evolving PSII. This inhibits plant development and productivity. A physiological mechanism of photoprotection called nonphotochemical quenching (NPQ) is the major and fastest response carried out in the thylakoid membranes to harmlessly dissipate the excess light energy (Demmig-Adams et al., 2014). Two opposing views existed on the proteins, pigments, and dynamic processes involved in NPQ (Ruban et al., 2012). The recent work on LHCII minor antenna mutants revealed the truth.

One view considers the existence of specific photoprotective pigment-protein complexes that are crucial in extinguishing excess photons in the light-harvesting antenna. These minor antenna complexes, composed of CP24, CP26, and CP29, are proposed to carry and activate the quenching pigment zeaxanthin that could remove excess excitation energy from the bulk antenna (LHCII) chlorophylls (Holt et al., 2005; Ahn et al., 2008). The other view is that the LHCII antenna itself possesses an inherent ability to protect itself against overexcitation by changing its conformation from a light-harvesting to a photoprotective state (Ruban et al., 2012). In the case of higher plants, it was proposed that the major trimeric LHCII complex of the PSII antenna undergoes aggregation, which is triggered by the proton gradient generated across the photosynthetic membrane in excess light (Ruban, 2018). The aggregated LHCII loses its ability to efficiently deliver absorbed energy to the PSII reaction center because it promptly leaks this energy in the form of heat via established quenching interactions between the bound chlorophyll and carotenoid (specifically lutein) pigment cofactors (Ruban et al., 2012).

The easiest way to verify the first view would be to knock out the expression of the proteins of the proposed photoprotective light-harvesting complexes, CP24, CP26, and CP29 (Niyogi, 1999). Such work has recently been accomplished by obtaining No Minor monomeric LHCII antenna complexes (termed NoM) Arabidopsis (*Arabidopsis thaliana*) plants (Dall'Osto et al., 2017). This and the report that followed (Townsend et al., 2018), however, have clearly demonstrated that the NoM mutant possesses normal levels of quenching, indicating that quenching does not originate from the minor antenna proteins but rather entirely resides in the major trimeric LHCII complexes.

Another protein, PsbS, has been found to be crucial for the major and quickly reversible component of NPQ, qE (Li et al., 2000). Surprisingly, it was discovered not to bind pigments, hence it could not be directly involved in the quenching of excitation energy (Dominici et al., 2002). However, the view that regards the entire LHCII antenna as a site of NPQ/qE has no trouble reconciling these two seemingly contradictory observations and also clearly explains experiments on xanthophyll biosynthesis mutants with altered levels of quenching (Ruban et al., 2012; Ruban, 2018). This view proposes the allosteric modulatory role of PsbS and carotenoid zeaxanthin in NPQ and states that all that is needed for quenching is LHCII trimers and the proton gradient. PsbS and zeaxanthin are suggested to finely tune the sensitivity of LHCII to protons, hence allowing for fundamental physiological control over light harvesting in nature (Ruban, 2018).

The facts remain clear: there is plenty of NPQ without the minor antenna in the NoM mutant, with its prompt component qE being even more pronounced and faster recovering than that of the wild type (Townsend et al., 2018). Moreover, quenching can be generated in the absence of PsbS by an enhanced proton gradient (Ruban et al., 2012). But how could the gradient trigger NPQ in LHCII? Perhaps this is the time to refocus the research on the regulation of light energy partitioning

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in the photosynthetic membrane by adopting a more holistic view of the interplay between the proton gradient, membrane environment, and protein dynamics. For example, several reports showed significant thinning of the photosynthetic membrane (up to 25% reduction in thickness) induced by the proton gradient (Murakami and Packer, 1970) and correlation of this change with NPQ (Johnson et al., 2011). The membrane thinning was discovered to trigger the hydrophobic mismatch between membrane proteins and the lipid bilayer (Killian, 1998). This mismatch caused thermodynamically driven protein conformational changes and protein aggregation (Killian, 1998). If hydrophobic mismatch takes place in the thylakoid membrane, it could possibly alter the pK for protonation of LHCII amino acids by immersing them in a more hydrophobic environment (Ruban et al., 2012) and therefore stabilizing the protective conformation. This seems to be a feasible scenario for the self-regulation of the photosynthetic light harvesting that relies not upon a single protein complex or pigment but rather upon the integrative properties of the photosynthetic membrane. The key feature here is the thermodynamic relationship between different forces in the membrane environment that alter the state of LHCII complexes for the regulated physiological response to the light stress (Ruban, 2018).

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