Phosphorylation and oxidative damage mediates disassembly of Photosystem II in *Arabidopsis thaliana*

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The photosynthetic electron transport chain of oxygenic photoautotrophs is composed of several large hetero-oligomeric pigment protein complexes. Photosystem II (PSII) utilizes radiant light energy to oxidize water into dioxygen and protons, reducing the mobile electron carrier plastoquinone and contributing to the generation of a transmembrane ΔpH gradient. In doing so, the electron transport chain creates usable forms of chemical energy necessary for carbon fixation. Although PSII is necessary for functional electron transport, PSII also forms various reactive oxygen species (ROS) as a byproduct of its reactions. The direct result of these ROS manifests as irreversible protein damage to subunits of PSII, particularly the D1 subunit, leading to a loss of its catalytic activity. To ensure functional electron transport, the damaged PSII supercomplex undergoes a rapid disassembly repair cycle that involves the degradation of the damaged D1 subunit, followed by its *de novo* synthesis and reassembly to form the PSII holocomplex. Disassembly of the PSII supercomplex, specifically removal of the CP43 subunit, ensures access of the D1 subunit by the integral membrane protease FTSH. Although many aspects of the PSII repair cycle are have been investigated, the mechanisms governing disassembly of PSII are not well understood. More specifically, it is unclear if PSII disassembly is controlled through a strictly controlled mechanism. Here, we utilize BN-PAGE to show the disassembly of PSII through removal of CP43 is induced through the addition of exogenous ROS. Additionally, we show through the use of *Arabidopsis* phosphorylation mutants that phosphorylation of PSII is important for the disassembly of large dimeric PSII supercomplexes. Together, these results demonstrate that both controlled and damage-mediated disassembly mediate the PSII repair cycle.

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