Photoinactivation and Photoprotection of Photosystem II in Plants

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A unifying mechanism for photoinhibition of photosystem II (PSII) will be presented. This mechanism is based on current observations from *in vivo* studies rather than from *in vitro* studies with isolated thylakoids or PSII membranes. *In vitro* studies have limited relevance for *in vivo* photoinhibition because very high light is used with photon exposures rarely encountered in nature, and most of the multiple, interacting, protective strategies of PSII regulation in living cells are not functional. It is now established that the photoinactivation of PSII in vivo is a probability and light-dosage event which depends on the photons absorbed and not the irradiance *per se*. As the reciprocity law is obeyed and target theory analysis strongly suggests that only one photon is required, we propose that a single dominant molecular mechanisms occurs in vivo with one photon inactivating PSII under limiting, saturating or sustained high light. Two mechanisms have been proposed for photoinhibition under high light, acceptor-side and donor-side photoinhibition [Aro et al, (1994) Biochim Biophys Acta 1143: 13-134], and another mechanism for very low light, the low-light syndrome [Keren et al. (1995) J Biol Chem 270: 214-223]. Based on exciton-radical pair equilibrium model of exciton dynamics, we propose a unifying mechanism for the photoinactivation of PSII *in vivo* under steady-state photosynthesis that depends on the generation and maintenance of increased concentrations of the primary radical pair, $P680^{+}Pheo^{-}$, and the different ways of charge recombination is regulated under varying environmental conditions. We suggest that the primary cause of damage is $P680^{+}$, rather than singlet O_2, formed from triplet P680, or other reactive oxygen species.