A-17

THE EFFECT OF H_2O_2 ACCUMULATION ON DEGRADATION OF PHOTOSYSTEM II AND I BY MV TOXICITY

Jin-Hong Kim and Choon-Hwan Lee

Dept. of Molecular Biology, Pusan National University, Pusan 609-735, Korea

The basic mechanism of paraquat (MV) toxicity was investigated in rice (*Oryza sativa* L.) leaf segments by monitoring the changes in Fv/Fm (photochemical efficiency) and the extent of degradation of photosystems. Paraquat treatment induced a significant decrease of D1 protein, a core protein of PSII, with an increasing concentration (2.5uM to 100uM), but didn't show notable differences in the amount of PsaA/B, core proteins of PSI. To identify reactive oxygen species responsible for the degradation of D1 protein, we used various compositions of paraquat with two inhibitors, diethyldithiocarbamic acid (DDC) and hydroxyurea (HU) as a superoxide dismutase inhibitor and an ascorbate peroxidase inhibitor, respectively. Unexpectedly, no decrease in D1 protein was observed when 5mM DDC was co-treated with 5uM MV. On the contrary, in the case of 5uM HU co-treatment, fragmentation of D1 protein was observed, indicating the loss of PSII activity. These results were also closely correlated with the changes of Fv/Fm, suggesting that DDC could protect PSII by increasing superoxide and decreasing H2O2. Also, rapid accumulation of zeaxanthin after paraquat and inhibitor treatment was commonly observed except in the case of HU, irrespective of its protective role. It means that the decline of reduced ascorbate, an antioxidant and a substrate for de-epoxidation, could be caused by the rapid accumulation of zeaxanthin, helping to accumulate H2O2. Therefore, we suggest that the main target for the paraquat toxicity should be PSII, expecially D1 protein rather than PSI, and the main reason for the toxicity be H2O2 accumulation coupled with the increased superoxide.