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PUBLIC LECTURE

Oxidative damage to protein synthesis during photoinhibition of photosystem II

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Slatyer Seminar Room, R.N. Robertson Building (Bldg. 46), Linnaeus Way, ANU

Photosystem II (PSII) is sensitive to strong light, and the light-induced inactivation of PSII is referred to as photoinhibition. The action of reactive oxygen species (ROS) in photoinhibition has been a long-lasting question. We have found that the repair of PSII from photodamage is particularly susceptible to inactivation by ROS [see reviews 1,2]. The ROS-induced inhibition of repair in the cyanobacterium *Synechocystis* sp. PCC 6803 was attributed to the suppression of synthesis *de novo* of proteins, at the level of translational elongation, that are required for the repair of PSII, such as the D1 protein [1,2]. Molecular analysis revealed that the ROS-induced suppression of protein synthesis was associated with the specific inactivation of elongation factor G (EF-G), a key protein in translational elongation, due to the formation of an intramolecular disulfide bond between two specific cysteine residues [3,4]. The inactivation of EF-G was reversed via the reduction of the target disulfide bond by thioredoxin, suggesting that protein synthesis might be reactivated by the reducing power that is generated from the photosynthetic transport of electrons and mediated by thioredoxin and EF-G [4]. Thus, protein synthesis might be regulated by photosynthesis in a redox-dependent manner. Expression of mutated EF-G with a target cysteine residue replaced by serine in *Synechocystis* resulted in the protection of PSII from photoinhibition [5]. This protection was attributable to the enhanced repair of PSII via acceleration of the synthesis of the D1 protein, which might have been due to reduced sensitivity of protein synthesis to oxidative stress. The particular sensitivity of EF-G to ROS was also observed in *Escherichia coli*, suggesting that the redox regulation of translation might also be conserved in non-photosynthetic organisms [6]. The physiological role of the redox regulation of protein synthesis will be discussed.

References

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