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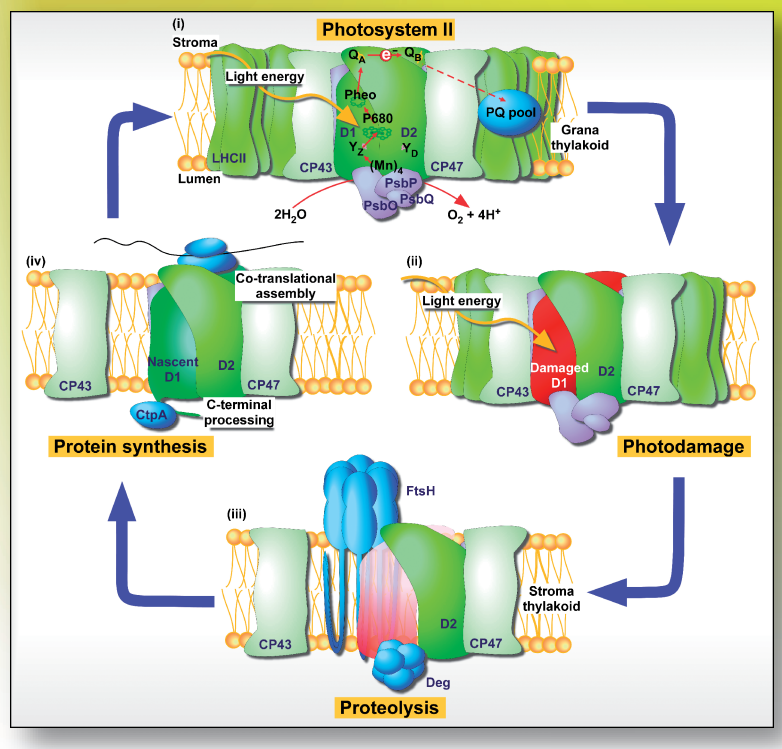

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	Immunochemistry
	Physiological Chemistry
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	Protein Synthesis
	DNA–Protein Interaction
	RNA Processing
	Genetic Engineering
	Genetic Diseases
	Molecular Genetics
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Fields:	Topics
Cell:	Cell General
	Biomembranes, Organelles, and Protein Sorting
	Muscles
	Cytoskeleton, Cell Motility, and Cell Shape
	Extracellular Matrices and Cell Adhesion Molecules
	Cell Cycle
	Receptors and Signal Transduction
	Stress Proteins and Molecular Chaperones
	Cell Death
	Differentiation, Development, and Aging
	Neurobiology
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Biotechnology:	Biotechnology General
	Biomimetic Chemistry
	Biomaterials
	Bioactive Substances
	Synthetic Peptides and Oligonucleotides
	Gene and Protein Engineering
	RNA Technology
	Glycotechnology
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COVER: Schematic view of the repair cycle in Photosystem II (PSII). Light energy constantly damages photosynthetic proteins. In particular, photooxidative damage at PSII, a large pigment-protein complex consisting of more than 20 subunits and cofactors in the thylakoid membrane, is detrimental to plant survival. Photosynthetic organisms have evolved an efficient PSII repair cycle, where photodamage is targeted to reaction center protein D1 and the photodamaged D1 was efficiently degraded. As indicated in this scheme, an efficient PSII repair requires partial PSII disassembly, specific recognition and degradation of photodamaged D1, insertion of newly-synthesized D1, and PSII reassembly. Kato and Sakamoto (pp. 463–469) describe a proposed mechanism of D1 degradation by prokaryotic proteases in the PSII repair. Accumulating evidence implicates that ATP-dependent membrane metalloprotease FtsH plays a central role in D1 degradation, aided by Deg proteases peripherally attached to thylakoid membranes. [See Kato and Sakamoto, p. 463].