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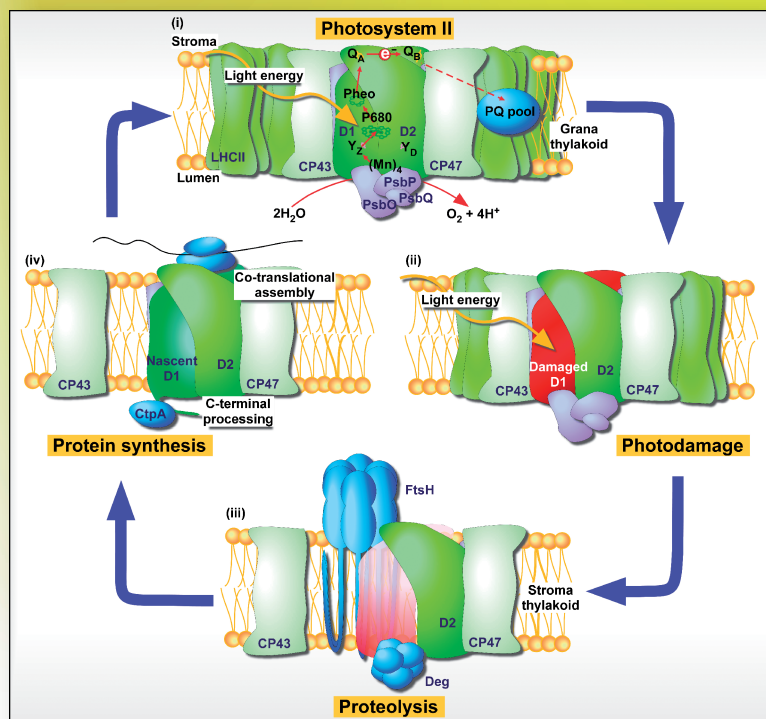
# JB

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**BIOCHEMISTRY  
MOLECULAR BIOLOGY  
CELL  
BIOTECHNOLOGY**

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**Quality Control of the Cellular Protein Systems**



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- Biochemistry in Cell Membranes
- Biochemistry in Diseases and Aging
- Neurochemistry
- Immunochemistry
- Physiological Chemistry
- Biochemical Pharmacology
- Analytical Biochemistry

##### **Molecular Biology:** Molecular Biology General

- Genes and Other Genetic Materials
- Replication and Recombination
- Gene Expression
- Protein Synthesis
- DNA-Protein Interaction
- RNA Processing
- Genetic Engineering
- Genetic Diseases
- Molecular Genetics
- Molecular Evolution
- Bioinformatics

**Fields:** Topics

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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  
Tumor and Immunology

**Biotechnology:** Biotechnology General

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Biomaterials  
Bioactive Substances  
Synthetic Peptides and Oligonucleotides  
Gene and Protein Engineering  
RNA Technology  
Glycotechnology  
Immunological Engineering  
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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].