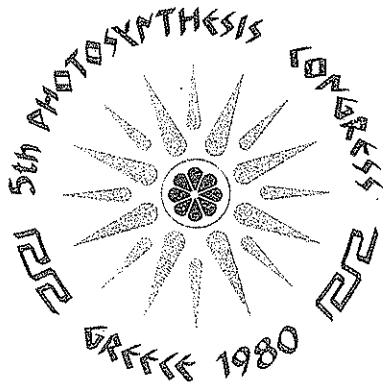


Fifth International Congress on Photosynthesis

September 7 - 13, 1980
Halkidiki, Greece



ABSTRACTS

PHOSPHORYLATION OF POLYPEPTIDES OF THE LIGHT-HARVESTING CHL *a/b* COMPLEX

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Incubation of isolated chloroplast thylakoids with γ - ^{32}P -ATP results in phosphorylation of surface-exposed segments of several membrane proteins. The incorporation of ^{32}P is light-dependent, is blocked by diuron (an inhibitor of electron transport) but is insensitive to uncouplers of photophosphorylation (such as nigericin). Polypeptides of the light-harvesting chlorophyll *a/b* protein complex are the major phosphorylated membrane proteins. Addition of ATP to isolated chloroplast thylakoids at 20°C results in a time-dependent reduction of chlorophyll fluorescence emission; this is blocked by diuron but not by nigericin. ADP could not substitute for ATP. Chlorophyll fluorescence induction transients after incubation of thylakoids with ATP showed a decrease in the variable component. Chlorophyll fluorescence at 77°K of phosphorylated thylakoids showed an increase in long wavelength emission compared with dephosphorylated controls. We conclude that a membrane-bound protein kinase can phosphorylate surface-exposed segments of the light-harvesting pigment-protein complex, altering the properties of its interaction with the two photosystems such that the distribution of absorbed excitation energy increasingly favours photosystem I. The level of phosphorylation of the light-harvesting complex is determined by the relative activities of the protein kinase and a thylakoid-bound phosphatase. Both enzymes require about 5 mM Mg^{2+} for maximum activity but only the kinase is light-dependent. When illuminated thylakoids are placed in darkness the kinase activity declines to the dark level within about 10 min. Reactivation of the kinase may be achieved either by illumination or by dithionite in the dark. In the light dithionite reverses the inhibitory effects of diuron on protein phosphorylation and redistribution of absorbed excitation energy to photosystem I. We conclude that protein kinase activity requires the reduction of a component of the electron transport chain near the electron-accepting side of photosystem II. We suggest that the distribution of excitation energy between the two photosystems is controlled *in vivo* by a feedback mechanism in which an imbalance between the exciton fluxes to the reaction centres of the photosystems is detected by a change in the redox state of an electron carrier and is corrected by the activation or inactivation of the protein kinase that phosphorylates the light-harvesting chlorophyll *a/b* polypeptides. Evidence that the critical electron carrier is plastoquinone will be presented.

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Sponsored by: The Greek Ministry of Culture and Science
The Greek Atomic Energy Commission
The National Tourist Organization of Greece
The International Union for Pure and Applied Biophysics

Acknowledgements:

The generous support of the following organizations is gratefully acknowledged.

Olympic Airways

National Bank of Greece

Hellenic Scientific Representations Ltd.

"Botrys" Société Hellenique de Vins et Spiritueux S. A.

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