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Impairment of Auxin Transport by Protein Kinase Inhibitors

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Treatment of *Pisum sativum* tissue with the protein kinase inhibitor staurosphorin resulted in impairment of ^{3}H-indoleacetic acid transport in etiolated stem segments. The transport inhibition was accompanied by the increase in net uptake of labeled auxin in tissue. The magnitude of auxin accumulation in tissue treated with the phytotropin 1-N-naphthylphthalamic acid (NPA) which specifically blocks the efflux of auxin in the plasma membrane was reduced by the protein kinase inhibitor, suggesting that inhibition of protein phosphorylation could lead to hinderance to the auxin exporting function of NPA receptors. The flavonoid genestein which is also known to inhibit protein kinase likewise reduced NPA-induced auxin accumulation. However, the flavonoid did not bring about auxin accumulation. In view of the finding that the flavonoid also competes with NPA for a commom binding site, a mechanism for the flavonoid effect on the NPA action will be proposed.

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Defined Structural Changes of the Photosystem II Reaction Center Inactivated by Heat Treatment

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Structural changes in the electron acceptor side of the PSII reaction center by heat treatment (45 °C for 5 min) have been monitored. In heat-treated spinach chloroplast thylakoids, the inhibitory effect of DCMU on the electron transport activity of the PSII reaction center from diphenyl carbazide to dichlorophenolindophenol, the artificial electron donor and acceptor, respectively, became reduced approximately 3.8 times and [^{14}C]-labeled DCMU binding on the D1 polypeptide, one of the major component of the PSII reaction center, decreased to 25-30 % of intact thylakoid membranes, implying that the conformational changes of the DCMU binding pocket, resided on the D1 polypeptide, occur by heat treatment. The accessibility of trypsin to the NH_2-terminus of the cytochrome b-559 α-subunit, assayed with Western blotting technique using the antibody generated against the COOH-terminal domain (Arg-68 to Arg-80) of α-cytocrome b-559, was also increased, indicating that heat-treatment caused changes of the structural environments near the stromal side of the cytochrome b-559 α-subunit, allowing trypsin more easily to cleave the NH_2-terminal domain. Therefore, the structural changes in the electron acceptor side of the PSII reaction center complexes could be one of the reasons why the oxygen evolving activity of heat-treated thylakoid membranes decreased.