Volume 56(1) 75-92, 2012
Acta Biologica Szegediensis
http://www.sci.u-szeged.hu/ABS

formation of long-range chiral order of chlorophyll and carotenoid molecules, as manifested in Ψ-type CD. The role of LHCII in determining the CD is mainly exerted via organising the PSII supercomplexes into chirally organized macro-assemblies, rather than via increasing the number of interacting chromophores. PSII associated with LHCII appears to possess higher ability to form macro-domains than either the core complexes or LHCIIs on their own. Coexistence of the LHCII and PSII core in the membrane - but without coupling (by LMW proteins) - appears to result in less ordered arrays.

Our data also reveal specific functions of some of the protein and lipid compounds in the light adaptation processes of plants.

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Role of Pituitary Adenylate Cyclase-Activating Polypeptide (PACAP) in the trigeminovascular system

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Abnormal activation of trigeminovascular system (TS) plays important role in the development of migraine, but its precise mechanism is still unknown. Recent clinical and experimental data have suggested that Pituitary Adenylate Cyclase-Activating Polypeptide-38 (PACAP-38) might contribute to the evolution of migraine-attacks. Therefore, we aimed to examine the PACAP-immunoreactivity (IR) in blood plasma of migraineurs, in interictal and ictal periods as well as in blood plasma and nerve tissues in two animal models of activated TS.

Adult Sprague-Dawley rats were got involved in the experiments. Electrical stimulation (ES) of the trigeminal ganglion (TRIG) was applied at 10 Hz, 1 mA for 30 min and in a separate group of rats 10 mg/kg dose of nitroglycerol (NTG) was injected ip. Peripheral blood samples were collected, and three brain regions, involved in migraine (caudal trigeminal nucleus-TNC; TRIG; cervical 3-5 of the spinal cord-SC) were dissected at different time-points after the stimulation of the TS.

In the clinical study 40 control subjects and 60 migraineurs were examined, selected by the criteria of the International Headache Society. Blood samples were collected from 21 migraineurs in the interictal and drug-free ictal periods.

In both case, the blood samples were taken to tubes containing EDTA and protease inhibitor, then the plasma was separated (2000 rpm 10 min 4°C). The plasma and nerve tissue samples were stored at -80°C till the PACAP radioimmunoassay measurements.

In rats the plasma PACAP-IR significantly increased 90 and 180 min after ES compared to the sham-stimulated and intact control groups. ES also evoked a remarkable elevation of PACAP level in the TNC at the 180 min time-point. In the NTG-model plasma PACAP-IR remained unchanged, but significant PACAP-IR increase was observed in the TNC 90 and 180 min following the chemical stimulation. The level of this peptide was not substantially altered in the TRIG and the SC in either model.

Significantly lower PACAP-38-IR were detected in human interictal migraine samples, than in the control group (Student's *t*-test for unpaired comparisons; $p<0,002$). Self-control comparison of PACAP-38-IR of 17 migraineurs in the ictal and interictal periods showed significant elevation during the attack (Student's *t*-test for paired comparisons; $p<0,002$).

It is concluded, that in animals the elevated PACAP-IR in the systemic circulation and/or in the TNC induced by PACAP release from both the peripheral and central terminals of the trigeminal pseudounipolar neurones.

The reduced concentration of PACAP-38 in the interictal period might be due to energy deficit. After a trigger the peptide can release from the sensory nerve terminals. The level of PACAP starts to significantly increase in the systemic circulation and induces vasodilatation, neuronal activation, sensitization, which are responsible for the initiation of pathomechanism of primary headache, like migraine.

The crucial role of PACAP in the activation mechanisms of TS is assumed. The nervous system specific examinations of PACAP can provide new perspectives to identify a new target in the therapy of migraine.

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