Tuesday, Nov. 5

7:30 Coffee and light breakfast

8:00 - 10:30 Symposium III. See and hear - PACAP in the visual and auditory systems. Session Chairs: Dora Reglodi, MD/PhD/DSc (Univ Pécs) and Tomoya Nakamachi, PhD (Univ Toyama)

8:00 Dora Reglodi, MD/PhD/DSc (Univ Pécs) Introduction - Protective effects of PACAP and VIP peptides in the sensory organs

8:10 Luis Pérez de Sevilla, PhD (Univ California, Los Angeles) Characterization of the VIP-1 amacrine cell in the mouse retina

8:40 Tamas Atlaš, PhD (Univ Pécs) Retinoprotective role of endogenous PACAP in inflammation

9:05 Alexandra Vaczy, MSc (Univ Pécs) The protective role of PAC1 receptor in endotoxin-induced retinal inflammation

9:30 Balazs Daniel Fulop, MD (Univ Pécs) Hearing impairment associated changes in the auditory pathway of PACAP-deficient mice

10:00-11:00 Coffee Break

11:00 - 1 Symposium IV. New insights on the mechanisms involved in the effects of PACAP on neuronal cell death and plasticity. Session Chair: David Vaudry (Univ Rouen, INSERM U1239)

11:00 David Vaudry, PhD (Univ Rouen, INSERM U1239) Prenatal exposure to neuropeptide PACAP prevents brain oxidative damages and apoptosis in fetal alcohol syndrome (FAS) mouse model

11:30 Atsuko Hayata-Takano, PhD (Osaka Univ) Analysis of PACAP signaling for functional synapse formation in the hippocampal neurons

12:00 Christina Van, PhD (Yale Univ) PAC1 maintains neurons and modulates inflammation in a model of multiple sclerosis and optic neuritis

12:30 Dora Reglodi, MD/PhD/DSc (Univ Pécs) PACAP deficiency in mice and humans – from a potential model of accelerated aging to human conditions

1-2 Lunch break

ABSTRACTS

Our data show in VPAC1/- mice an altered feeding behavior with higher numbers of feeding bouts during the dark cycle (p<0.009), lower TEE (total energy expenditure) values during both the dark (p=0.025) and light cycle (p=0.042), and lower O2 consumption during the dark (p=0.029) as well as the light phase (p=0.044). Furthermore, VPAC1/- mice had significantly lower CO2 values during the dark phase (p=0.016 and light phase p=0.029), and lower levels of released H2O (dark phase, p=0.018).

In addition, VPAC1/- mice had significant higher plasma levels of C-1 during fasting (p=0.035) and post-prandial conditions (p=0.0287), gluconate during postprandial conditions (p=0.0006), YY during fasting (p=0.0001) as well as postprandial conditions (p=0.0002), and lower postprandial leptin levels (p=0.001). Also, VPAC1/- mice had significant lower plasma glucose levels (p=0.0299).

A glucose clearance test revealed no significant differences between VPAC1/- and WT mice at 0, 30, 60, and 120 minutes post-glucose infusion; whereas, the insulin tolerance test, demonstrated significantly lower glucose levels in VPAC1/- mice at 60 minutes (p=0.0419) and at 120 minutes (p=0.0152). No significant differences were found in body phenotype, in the amounts of food consumed, in physical activity and insulin levels.

These data reveal significant metabolic differences between VPAC1/- and their WT littermates in feeding behavior, but without the circadian alterations recorded in VPAC2/- and VIP/- mice, and also in TEE, respirometry indices, plasma metabolic hormone and glucose homeostasis, thus supporting a key role for VPACR1 in the regulation of body metabolism, and diabetic control.

PACAP – SWEAT SECRETION AND SWEAT GLAND
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Sweat secretion is the major function of eccrine sweat glands and its process is disturbed, serious skin problems will be occurred. To investigate the effect of PACAP on eccrine sweat secretion, we first studied the distribution and localization of PACAP and its receptor in the sweat glands and secondly physiological function of PACAP on sweat secretion. Moreover, we made an in vitro study to clarify a signal transduction mechanism by PACAP on sweat secretion by use of immortalized sweat gland cell line NCLS-SC3. As a result, PACR-like immunoreactivity was expressed in the secretory cells of both mouse and human sweat glands. PACAP-like immunoreactivity was found in nerve fibers surrounding the eccrine sweat glands. Moreover, PACAP significantly promoted sweat secretion after intradermal injection, but this was inhibited with PACAP6-38 treatment. However, VIP, an agonist of VPAC1R and VPAC2R, failed to induce sweat secretion. It was confirmed that PACAP acts on PAC1-R in the NCLS-SC3 cells to increase intracellular calcium concentrations. It is also confirmed that water channel protein, aquaporin 5 (AQP5), is expressed in the NCLS-SC3 cells and PACAP stimulates AQP5 translocation from the cytoplasm to cell membrane. These results may indicate that PACAP plays a crucial role in sweat secretion via throug PAC1R located in eccrine sweat glands. If the sweat secretion is disturbed, serious skin problems can arise, so PACAP may protect paridrosis and stimulate epidermal cell regeneration and repair as occurred in the cornea.

12 Shun Sasaki1, Michio Yamashita2, Hirohiko Sueki1 and Seiji Shioda2

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