1. Soh SB, Aw TC. Laboratory Testing in Thyroid Conditions - Pitfalls and Clinical Utility. *Ann Lab Med*. 2019;39(1):3-14. doi:10.3343/alm.2019.39.1.3
2. Spencer C., PhD. Assay of Thyroid Hormones and Related Substances

Adipose Tissue, Appetite, and Obesity

NEURAL MECHANISMS OF OBESITY

Development of a Protocol for Stellate and Celiac Ganglia Dissection for Characterization of Pituitary Adenylate Cyclase Activating Polypeptide (PACAP) Protein and Receptor Expression in Male and Female Mice Following Cold Acclimation

Parleen K. Pandher, BSc (Hons), Ekaterina Filatov, BHSc, Sarah L. Gray, BSc, PhD.
UNBC, Prince George, BC, Canada.

SAT-601

Pituitary adenylate cyclase-activating polypeptide (PACAP) is being studied to understand the endocrine regulation of energy balance and has been shown to be important in the regulation of the stress response (1,2). Specifically, PACAP has been shown to regulate thermogenesis, an energy burning process regulated by the sympathetic nervous system that contributes to achieving energy homeostasis in response to cold stress and overfeeding. PACAP is expressed in the sympathetic nervous system and is required at the adrenomedullary synapse to maintain epinephrine secretion from the adrenal medulla in response to physiological stress (3). Across the branches of the sympathetic nervous system, PACAP receptor expression is most well characterized in the superior cervical ganglia (SCG) (4). However, a detailed characterization of PACAP and its receptors has not been performed in ganglia whose postganglionic fibres innervate adipose tissues (stellate and celiac ganglia) in response to thermogenic stress. We hypothesized that PACAP is produced by preganglionic neurons innervating the stellate and celiac ganglia, and act on PACAP receptors expressed on the post-ganglionic neurons, and this expression will be upregulated in response to chronic cold stress. Due to their small and amorphous shape, we have developed a protocol to reliably isolate the stellate and celiac ganglia and validate their identity through the presence of tyrosine hydroxylase mRNA, using adrenal and SCG samples as positive controls. PACAP receptor expression (VPAC1, VPAC2, PAC1) was examined in the ganglia utilizing real-time PCR, and PACAP protein was visualized in the ganglia of transgenic mice that express eGFP under the control of the PACAP promoter (PACAP-eGFP mice) (5). This research demonstrates the expression of PACAP receptors in ganglia whose postganglionic fibres innervate adipose tissue, enhancing our understanding of PACAP's role in the SNS, and its contribution to the regulation of adaptive thermogenesis. **References**: (1) Gray et al., Pacap: Regulator of the stress response. In: Fink G, ed. Stress: Physiology, biochemistry, and pathology. 2019:279-291. (2) Mustafa, Adv Pharmacol. San Diego, Calif:445-457. (3) Eiden et al., Pflungers Arch. 2018 Jan;470(1):79-88. (4) Braas et al., J Biol Chem. 1999 Sep 24;274(39):27702-27710. (5) Condro et al., J Comp Neurol. 2016 Dec 15; 524(18):3827-3848.

Adrenal

ADRENAL CASE REPORTS I

Unilateral Adrenalectomy Can Induce Control in ARMC5 Mutations Patients

Maria João Ferreira, MD[1], José Luis Castedo, MD[1], Maria Manuel Costa, MD[2], Davide M. Carvalho, MD,PhD[3].
[1]Department of Endocrinology, Diabetes and Metabolism of Centro Hospitalar Universitário São João, Porto, Portugal, [2]Department of Endocrinology, Diabetes and Metabolism of Hospital Garcia da Orta, Porto, Portugal, [3]Department of Endocrinology, Diabetes and Metabolism of Centro Hospitalar Universitário São João; Faculty of Medicine, University of Porto, Portugal, Sao Mamede Infesta, Portugal.

SAT-197

Primary macronodular hyperplasia (PMAH) is a rare cause of endogenous Cushing's syndrome characterized by functioning adrenal macronodules and variable cortisol secretion. ARMC5 is the most frequent gene responsible for PMAH. Genetic mutations including inactivating germline mutations in armadillo repeat-containing 5 (ARMC5) gene have been identified. The occurrence of several other non adrenal tumors (meningioma, breast, colon, thyroid and parathyroid) has also been associated with PBMAH, suggesting a possible role of ARMC5 for the development of other neoplasias. The best treatment to this condition is not established. Clinical case: 64-year-old man, referred due to bilateral adrenal incidentalomas on a CT scan with characteristics of adenoma/hyperplasia. He had type-2 diabetes, hypertension and dyslipidemia for about 4 years. He was being treated with 4 different anti-hypertensive medications, gliclazide and simvastatin. Physical examination revealed thin and dry skin, obesity with centripetal fat distribution, multiple ecchymosis and facial erythrosis. He had a son and a daughter. No known familial relevant diseases. His laboratory workup revealed ACTH-independent hypercortisolism: failure to suppress on the overnight dexametasone suppression test - cortisol 27.6ug/dL- and on the low-dose dexamethasone test- cortisol 24ug/dL- associated to morning ACTH <1ng/L. He underwent stimulation tests with tetracosactide, LHRH, TRH, vasopressin, metoclopramide, glucagon anddeambulation test, which were overall negative. It was decided to proceed to bilateral adrenalectomy. Due to surgical complications, only right adenalectomy was performed. Histology revealed adrenal nodular hyperplasia. Molecular study in DNA extracted from peripheral blood leucocyte and in the adrenal gland revealed the presence in heterozygosity of the pathogenic mutation c.1379T>C in the ARMC5 gene. The patient underwent a cerebral CT scan which showed a meningioma in the posterior left temporal convexity. Genetic testing was also performed on the daughter and son of the patient, which revealed the same mutation. They were also tested for hypercortisolism and underwent adrenal and cerebral CT scan, which showed no abnormality. The adrenal CT scan of the daughter showed enlargement of the left adrenal gland, with aspects suggestive of nodular hyperplasia. Her blood tests revealed no sign of hypercortisolism - overnight dexametasone test- cortisol 1.0 ug/dL, morning ACTH 19.1 ng/L. The patient is currently with cortisol hypersecretion controlled. Conclusion: All patients with PMAH should be tested for ARMC5 mutations and if they are found, family screening is mandatory in this autosomal