Kiss1 (~35%) and NKB (~40%) compared to saline-treated animals; the abundance of Dyn mRNA did not differ between treatments. These data demonstrate that PVN UCN2 cells are activated during metabolic stress and that UCN2 is sufficient to suppress LH secretion and the expression of genes involved in stimulating LH pulses. These data support the hypothesis that UCN2 released from neurons in the PVN impairs KNDy cell function and LH secretion during acute stress.

Adrenal

TRANSLATIONAL STUDIES ON ADRENOCORTICAL FUNCTION IN HEALTH AND DISEASE

Luteinizing Hormone/Human Chorionic Gonadotropin Receptor Protein Expression in Adrenocortical Progenitor Cells, Aldosterone Producing Cell Clusters and Adrenal Adenomas Derived from Postmenopausal Women

Effie Tsomos, MD[1], Regina Belokovskaya, DO[1], Jose Sanchez Escobar, MD[2], Shen Yao, MD[1], Kazutaka Nanba, MD[3], Kenneth Haines, MD[1], William E. Rainey, MS PHD[4], Alex Kirschenbaum, MD[1], Alice C. Levine, MD[1].
[1]Icahn School of Medicine at Mount Sinai, New York, NY, USA, [2]Lenox Hill Hospital, New York, NY, USA, [3]National Hospital Organization Kyoto Medical Center, Kyoto, Japan, [4]University of Michigan, Ann Arbor, MI, USA.

OR19-02

Objective/Background
Adrenal pathologies are more common in women than men. Embryologically the adrenals and gonads develop from the adrenogenital ridge with differential migration and differentiation. We hypothesized that in adult females there are adrenocortical progenitor cells that express the LH/hCG-R and proliferate in response to elevated LH. Indeed, several case reports demonstrated LH/hCG-R expression in adrenal secretory tumors in postmenopausal and pregnant females. In aging adults, nests of cells known as aldosterone-producing cell clusters (APCCs) that may be precursors to aldosterone producing adenomas are frequently detected. We retrospectively studied the immunohistochemical expression of LH/hCG-R in normal adrenals, adrenal adenomas and APCCs in archival specimens derived from post-menopausal
women.
Methods
Archival specimens from adrenal adenomas derived from 23 women >55 years of age were examined. Clinical data was obtained in a blinded fashion and hormonal data was available in 9/23 cases; 6/9 were secreting cortisol and 3/9 adenomas were secreting aldosterone. In addition, 6 samples derived from a repository of normal adrenal tissues from deceased kidney donors (1 male, and 5 postmenopausal females) were studied. All specimens were immunostained for LH/hCG-R and the adrenal stem cell marker DLK1 that facilitates the maintenance of an undifferentiated phenotype. The normal adrenal tissues were also stained for aldosterone synthase (CYP11B2) to detect APCCs. The slides were reviewed and graded by a pathologist in a blinded fashion.
Results
Expression of LH/hCG-R was demonstrated in both normal and adenomatous tissues in all 23 specimens. The staining in adenomas was heterogeneous, with clusters of densely stained LH/hCG-R positive cells in all specimens. There were less densely stained clusters in normal adjacent adrenocortical tissue that was most prominent in the subcapsular, zona glomerulosa region, an area where the putative adrenal cortical stem cells are found as well as the zona reticularis. Double staining for the stem cell marker DLK1 and LH/hCG-R confirmed that these cells represent adrenocortical progenitor cells. CYP 11B2 immunohistochemistry of normal adrenals demonstrated cell foci dipping from the capsule into the zona fasciculata classified as APCCs that co-expressed cytoplasmic LH/hCGR.
Conclusion
Adrenal adenomas and APCCs derived from postmenopausal women exhibited heterogeneous but strong immunohistochemical expression of LH/hCG-R in all samples. Interestingly, DLK1-positive adrenocortical stem cells in the subcapsular zone also expressed LH/hCG-R. These data may provide insights into the female predominance of adrenal pathologies, particularly in postmenopausal women with high LH levels. The LH/hCG-R may be a viable target for treatment of adrenal adenomas in postmenopausal women.

Neuroendocrinology and Pituitary

CASE REPORTS IN SECRETORY PITUITARY PATHOLOGIES, THEIR TREATMENTS AND OUTCOMES

New Diagnosis of Acromegaly with DKA as Initial Presentation

Rajani Gundluru, MD[1], Ritika Verma, MD[1], Hariharan Regunath, MD[1], Michael J. Gardner, MD[2], Kiet Huynh, BS in biology[1].
[1]UNIVERSITY OF MISSOURI-COLUMBIA, Columbia, MO, USA, [2]University of Missouri, Columbia, MO, USA.

SAT-251

Background: While diabetes mellitus from growth hormone related insulin resistance is not uncommon in GH secreting tumors, initial presentation with diabetic ketoacidosis is rare.
Clinical Case: 31 YO male with no significant past medical history presented with c/o fatigue, 40 lb weight loss, polyuria, polydipsia. Clinical features of acromegaly with frontal bossing, protruding jaw, large hands and feet, thick spade like fingers, hammer toes, high arches and thickened fat pads on both feet were noted. Initial labs were consistent with DKA with anion of 31 mmol/L (0-20), blood glucose 241 mg/dl, bicarb 12 mmol/L (22-29), serum betahydroxy butyrate > 8 mmol/L (0-0.29), urine positive for glucose, ketones, protein. Patient was initially treated with IV insulin per DKA protocol, transitioned to subcutaneous insulin.
MRI brain showed 2.1x1.3x2.1 cm pituitary macro adenoma. Labs showed elevated IGF1 LC/MS, S 1094 ng/ml (54-310), IGFBP3 14 mcg/ml (3.5-7), Z score IGF MS Mayo > 3 (-2 to +2), normal FSH 7.1 m unit/ml (1.5-12.4), normal LH 3.4 m unit/ml (1.7-8.6), normal prolactin 6 ng/