of the metabolic syndrome (MetS) (2). These effects are thought to be mediated by the glucocorticoid receptor, a nuclear receptor showing affinity for both glucocorticoids and mineralocorticoids. In this study, we examined plasma concentrations of glucocorticoids and mineralocorticoids in women with or without the MetS. In addition, we assessed the ability of these steroids to predict fat accumulation and features of the MetS.

Methods: In a sample of 49 women (age 47 ± 4.99 years; BMI 26.4 ± 4.70 kg/m^2), plasma concentrations of cortisol, 11-deoxycortisol, cortisone, aldosterone, corticosterone and 11-deoxycorticosterone were analyzed by electrospray ionization-liquid chromatography-tandem mass spectroscopy (ESI-LC-MS/MS). Metabolic parameters were assessed to establish the presence of the MetS using NCEP-III criteria. Subcutaneous and visceral adipocyte cell size was measured by histomorphometry.

Results: We found HDL-triglycerides to be positively associated with levels of 11-deoxycorticosterone, 11-deoxycortisol, corticosterone, cortisone and cortisol ($p<0.05$ for all). 11-deoxycorticosterone concentration was also negatively associated with waist circumference (-0.294, $p<0.05$), LDL-cholesterol and LDL-triglyceride content (-0.264 and -0.362, $p<0.05$) whereas cortisone level was positively associated with fasting glucose (0.3, $p<0.05$). Our model including mineralocorticoids predicted systolic blood pressure (R^2=0.303), while the one including glucocorticoids predicted HDL-cholesterol (R^2=0.495). In addition, as expected, we found that women with the MetS were characterized by significantly higher percentage body fat and displayed subcutaneous and visceral adipocyte hypertrophy ($p<0.05$). Interestingly, women with the MetS also showed a trend for lower plasma cortisol concentrations ($p=0.07$).

Conclusion: Our data suggest that glucocorticoids and mineralocorticoids are associated with individual components of the MetS in women.

(1) Tchernof et al.(2013), Physiol Rev, 93(1);
(2) Constantinopoulos et al., (2015), Eur J Endocrinol, 172(1)

Tumor Biology

NOVEL REGULATORS OF BREAST CANCER PROGRESSION

Small Heterodimer Partner Modulates Antigen Presenting Myeloid Cells to Impair Regulatory T Cell Expansion, Promoting Anti-Tumor Immunity in Models of Breast Cancer.

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Immune checkpoint blockade has had underwhelming responses in breast cancer, in part due to the highly immune suppressive microenvironment. As a result, breast cancer continues to be the second most common cancer-related mortality amongst women, providing strong rationale for the development of new therapeutic approaches. Elevated circulating cholesterol is a poor prognostic, while breast cancer patients taking cholesterol-lowering drugs display increased time to recurrence. We and others have previously demonstrated that cholesterol metabolites mediate these effects by promoting breast cancer growth and metastasis, in part by suppressing the immune system. Therefore, given the demonstrated importance of cholesterol and its metabolites in breast cancer pathophysiology and immunology, we hypothesized that proteins involved in the regulation of cholesterol homeostasis would influence cancer progression. Through informatics analysis of breast tumors, we found that elevated expression of Small Heterodimer Partner (SHP; NR0B2) was a favorable prognostic. Antigen presenting cells such as macrophages and dendritic cells were found to express SHP, and manipulation of SHP altered the expression of genes involved in cross-talk with T cells. Intriguingly, when activated T cells were co-cultured with macrophages overexpressing SHP, there was a decrease in the expansion of regulatory T cells (Tregs) and *vice versa* in the absence of SHP. Adoptive transfer studies confirmed that loss of SHP resulted in immune suppressive Tregs. We hypothesized that myeloid cell-expressed SHP would promote immune surveillance and tumor clearance. In support of this hypothesis, tumors in the MMTV-PyMT model of mammary cancer grew at an accelerated rate in SHP-knockout mice. Tumors from these mice had significantly more Tregs and fewer effector T cells. Furthermore, orthotopic mammary tumor grafts grew at an increased rate in mice lacking SHP expression in myeloid cells (SHPfl/fl;LysMCre), compared to controls. A small molecule agonist of SHP impaired primary and metastatic tumor growth, and significantly enhanced the efficacy of immune checkpoint blockade in murine models of mammary cancer. Therefore, SHP represents a potential target to decrease suppressive Tregs, thereby allowing for immune-clearance of tumors.

Neuroendocrinology and Pituitary

CASE REPORTS IN SECRETORY PITUITARY PATHOLOGIES, THEIR TREATMENTS AND OUTCOMES

Surprising Transformation of a Microprolactinoma to a Macroprolactinoma

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Background: Microprolactinomas are typically benign tumors that rarely become macroprolactinomas. We present a rare case of a microprolactinoma that, after discontinuation of dopamine agonist (DA) therapy, transformed into a macroprolactinoma over a period of 6 years.**Clinical Case:** A 16-year-old woman initially presented for evaluation of galactorrhea without menstrual irregularities and was found to have elevated prolactin (68 ng/ml, normal range: 0-20), and a 4 mm pituitary microadenoma on MRI imaging.