wild-type (WT) mice as follows: fear conditioning training followed 24hr later by extinction training (new context), then 24hr later by extinction recall training. Percentage of time freezing was used to assess conditioned fear response. We measured BDNF gene expression in brain regions after completion of extinction recall training. Results: As expected, fear conditioning (learning) behavior was similar in HZ and WT mice. However, HZ mice showed a significant deficit in the early phase of fear extinction learning compared to WT. There was no difference in extinction recall between genotypes. Alterations in BDNF gene expression in the prefrontal cortex and amygdala was associated with deficit in fear extinction. Conclusion: Mice with a downregulation of Prkar1a gene demonstrate intact fear conditioning but impaired fear extinction learning, consistent with prior studies that report that PKA inhibition is necessary to facilitate extinction learning. Prkar1a-/- mice provide a valuable model to investigate impaired fear extinction to identify mechanisms for therapeutic targets for anxiety and trauma-related disorders.

Adrenal
ADRENAL CASE REPORTS II
Adrenal Cortical Carcinoma in a Male Patient Who Presented with Classic Signs and Symptoms of Cushing's Syndrome
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SUN-156
Adrenal Cortical Carcinoma in a Male Patient who Presented with Classic Signs and Symptoms of Cushing's Syndrome
Background: Adrenal Cortical Cancer (ACC) is a rare malignancy with an annual incidence of 1-2 per million population. ACC is generally considered a highly malignant tumor and account for 0.05-2% of all cancer Clinical case: A 32-year old male, newly diagnosed diabetic and hypertensive for 8 years, presented with left-sided sporadic, vague, non-radiating flank pain. Within a month, patient started to develop weight gain associated with Cushingoide features, buffalo hump, elevated blood sugars, hypertension, and eventually developed purple striae in the abdomen associated with easy bruisingness. Ultrasound was done which showed bilateral nephrolithiasis. Further work-up with CT-stonogram was done which showed bilateral nephrolithiasis and a 55x37x60mm heterogeneous mass above the left kidney in the area of the left adrenal gland. Further imaging with whole abdominal CT scan was done which revealed stable size of the mass in the region of the left adrenal gland with heterogeneous attenuation and enhancement. Initial hormonal work-ups done showed abnormal 1mg dexamethasone suppression test (874nmol/L N: 172-497nmol/L), but with normal aldosterone (73.22, N: <90pg/ml), renin (8.60pg/mL, N: 5-40pg/mL) and 24-hour urine metanephrines (31 mcg/24 hours N: 115-695mcg/24 hours). Serum K and blood glucose was managed according and patient was cleared to undergo surgery. Patient underwent left unilateral laparoscopic adrenalectomy which revealed a 7cm soft, friable left adrenal mass with minimal hematoma on the antero-inferior portion of the adrenal gland, with active bleeding. Histopathology of the left adrenal mass revealed high-grade adrenal neoplasm, consistent with adrenocortical carcinoma. Postoperatively, patient did not have complications of hypo nor hyperglycemia, hypotension and serum potassium was stable. Hydrocortisone was given postoperatively in tapering doses. Repeat cortisol were done 24 and 48 hours post op which showed normal results (4.02ug/dL and 5.37 ug/dL respectively, N: 5-25 ug/dL). Patient was referred to Oncology service for Chemotherapy and was eventually discharged stable with home medications for his Diabetes mellitus. On follow-up, there was noted improvement on the signs and symptoms of Cushing’s syndrome, with no hypokalemia and better blood glucose control. Conclusion: Majority of adrenal mass are benign but a high index of suspicion for malignancy should always be part of the workup. The acuteness of symptoms and size of mass are important determinants of malignancy. Prior to adrenalectomy, perioperative endocrine therapy is a must. Adrenocortical carcinoma should be managed by a multidisciplinary specialist team including an endocrinologist, oncologist, surgeon, radiologist, and a histopathologist.

Neuroendocrinology and Pituitary
NEUROENDOCRINOLOGY AND PITUITARY
A Pilot Study: Comparing Prolactin Measurements Between Two Different Immunoassays
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MON-281
Abstract: Hyperprolactinemia from a prolactin-secreting pituitary tumor is the most common endocrine disorder of the hypothalamic-pituitary axis. As suggested in the 2011 Endocrine Society Guidelines on Diagnosis and Treatment of Hyperprolactinemia, macroprolactin level should be assessed in patients with asymptomatic hyperprolactinemia. However, as discussed in prior studies comparing the performance of common prolactin immunoassays in a reference population of both males and females with and without known hyperprolactinemia or macroprolactinemia, there has been poor harmonization between assays and variable reactivity towards macroprolactin, resulting in significantly different normal ranges for total and monomeric prolactin between manufacturers. The goal of our analysis is to assess the concordance of the Roche and Siemens prolactin immunoassays using cases in which prolactin and macroprolactin testing was ordered on clinical indication. We hope to educate clinicians regarding potential variability between assays that may not be fully accounted for by using established, assay-specific reference ranges. We reviewed patients 18 years and older from any gender who underwent evaluation of prolactin levels as clinically indicated and had elevated serum prolactin on a Roche assay with a subsequent normal prolactin on a Siemens assay. Seven
out of 18 patients had an elevated prolactin on the Roche assay and a normal prolactin on the subsequent Siemens assay that also tests for the presence of macroprolactin. The reasons for testing prolactin in the 7 patients were: secondary hypogonadism (4), pituitary microadenoma (1), oligomenorrhea (1) and baseline labs in a transgenic female starting estrogen (1). Of the 7 cases we observed with discordant Roche and Siemens prolactin results, one of our 2 female patients and one of our 4 male patients would have shown concordant hyperprolactinemia results on both assays if the Siemens reference range was narrowed to align with published studies. This study demonstrates significant analytical discordance between prolactin immunoassays, leading to variable clinical interpretation regarding the presence of hyperprolactinemia. We suggest using a single prolactin immunoassay for routine measurement of prolactin as well as investigation of macroprolactin measurement to ensure comparable reactivity towards all forms of prolactin. References: (1) Vallette-Kasic et al., J Clin Endocrinol Metab. 2002 Feb;87(2):581-8. (2) Gibney et al., J Clin Endocrinol Metab. 2005 Jul;90(7):3927-32. (3) Luisa et al., Clinical chemistry 2008 Sept; 54:10 1673-1681 (4) Shlomo et al., J Clin Endocrinol Metab, February 2011, 96(2):273-288

Adrenal
ADRENAL CASE REPORTS I

Clinical Dilemmas of Adrenal Incidentaloma, Is Change in HU Clinically Significant?
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SAT-200
Introduction
There has been a recent increase in the incidence of adrenal incidentaloma (AI), defined as an adrenal mass that’s unintentionally discovered on imaging obtained for an indication other than suspected adrenal pathology, mainly because of increased availability and improved quality of imaging modalities. Differentiation based on malignant vs benign and secretory vs non secretory becomes the most important question on finding such mass. Another important question is long term follow up of these AI. We present a case that highlights the importance of follow up due to the potential of AI to increase in size and density over 3 year.

Case
A 58 years old male with a history of ESRD secondary to hypertension, controlled hypertension, CAD s/p CABG was seen for a right 1.9 cm AI (found in 11/2016 on noncontrast CT abdomen) with low attenuation of approximately 10 Hounsfield units (HU).
He was asymptomatic, normal vitals, and an unremarkable physical exam. Hormonal work up was negative for hyperaldosteronism and Cushing syndrome. About 1 year later, repeat CT abdomen reported an unchanged right, 2.0 x 1.7 cm adrenal mass, 22 HU. Hormonal work-up was not completed and patient was lost to follow up.

Two years later, repeat CT abdomen reported increase size of AI to 2.7 cm and increase in attenuation of 24 HU precontrast which increased to 55 HU on the immediate postcontrast exam and 55 HU on 13-minute delayed images. Hormonal work up was negative for Cushing syndrome and hyperaldosteronism. Plasma metanephrine was 0.50 with elevated plasma normetanephrine of 3.85.
In light of being a renal transplant patient and due to increase in size, change in HU, and significantly elevated normetanephrine levels patient was referred for right adrenalectomy.

Discussion
Change in adrenal mass size is known to be a significant predictor of malignant potential. An unenhanced attenuation value <10 HU is characteristic of a lipid-rich adenoma, can predict benign adrenal adenoma with 98% specificity, and has a very low likelihood of a pheochromocytoma. European Society of Endocrinology recommends if the AI is homogenous, < 4 cm, with a density ≤ 10 UH, no imaging follow up is recommended and biochemical testing for ruling out pheochromocytoma is indicated in adrenal tumors with an unenhanced attenuation value >10HU. However, the AACE/AAES guidelines recommends AI ≥ 1 cm and less than 4 cm, repeat imaging with noncontrast CT should be performed at 3–6 months and annually for 1-2 years and if the mass grows or becomes hormonally active then adrenalectomy should be performed.

Conclusion
There are no prospective studies of the optimal frequency and duration of follow up for AI. Considering the increasing incidence of AI, further studies on the clinical significance of change in HU, such as risk of developing pheochromocytoma, along with standardized international guidelines would be helpful for clinicians in managing patients with AI.

Neuroendocrinology and Pituitary
RESEARCH ADVANCES IN PITUITARY TUMORS

Inadequate High Mitochondrial ATP-Synthesis Explains “Non-Fatty-Liver” in Patients with Acromegaly
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Background
Patients with active acromegaly exhibit low hepatocellular lipid content (HCL) despite pronounced insulin resistance.

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