Density (BMD) and pelvic fractures. We have conducted a pilot cross-sectional study to establish a method of measuring pelvic BMD and to correlate BMD of the pelvis with BMD at other skeletal sites. Postmenopausal women without a history of pelvic and hip fragility fractures were enrolled. Hip, spine, and pelvic DXA scans were obtained using a Hologic DXA machine. Pelvic BMD was calculated using Hologic Research Software from 3 areas of the pelvis (R1: public symphysis, R2: inferior pubic ramus, and R3: superior pubic ramus), corresponding to common fracture locations. Pelvic BMD was the average of the 3 pelvic sites. Pelvic BMD measurement precision error was calculated using the root mean square method (Recommended by International Society of Clinical Densitometry (ISCD)). Statistical analysis was used to compare BMD at different sites. Alpha error was set at 0.05. Of 73 postmenopausal women who were enrolled in the study (average age 64 years, average 15 years postmenopausal), 3% had chronic kidney disease, 7% had type 2 DM, 3% were on corticosteroids and none were smokers. BMD of femoral neck assessed on pelvic DXA was not significantly different from femoral neck BMD measured on standard DXA (P=0.09). To assess pelvic BMD measurement precision, 15 patients underwent 3 separate pelvic DXA images after repositioning. BMD precision error was 0.011/cm² which is slightly lower than the precision total hip BMD at our center (0.007/cm²). BMD of R1, R2, and R3 pelvic areas were measured as 0.44±0.15, 0.41±0.15, and 0.62±0.19 g/cm², respectively. Notably, BMD of R3 was significantly higher than the other 2 areas (P<0.001, ANOVA). Average BMD (0.49±0.14 g/cm²) of pelvis was significantly lower than BMD of femoral neck (0.72±0.16 g/cm²), total hip (0.86±0.17 g/cm²) and spine (0.97±0.19 g/cm²)(P<0.001). Average BMD of pelvis was significantly lower in participants with osteopenia and osteoporosis of the hip and femoral neck compared to participants with normal BMD in those locations. In summary, we report a precise method of measuring BMD of commonly fractured areas of the pelvis. Pelvis BMD is lower than hip, femoral neck, and spine. Bone density of the pelvis correlates with hip and femoral neck bone density. The results of this pilot study can be used for future studies looking at pelvic low bone density in patients with pelvic fragility fractures which could help identify patients at risk for pelvic fragility fractures and change how osteoporosis is defined based on DXA images.

Diabetes Mellitus and Glucose Metabolism
CLINICAL AND TRANSLATIONAL STUDIES IN DIABETES
FDXR Regulates Iron Metabolism and Glucose Metabolism in Liver.
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MON-635
Iron is an essential cofactor for many proteins that function in electron transport or oxygen transport as heme or iron-sulfur cluster. On the contrary, iron also has the potential to cause oxidative damage if not carefully regulated and when in labial iron excess. Clinical studies show that elevated serum ferritin levels are observed in most patients with type 2 diabetes and non-alcoholic fatty liver disease (NAFLD). In this context, p53 is shown to induces some mitochondrial iron regulatory genes. The role of crosstalk between p53 and iron metabolism has not been sufficiently examined in the pathogenesis of diabetes and NAFLD.

Here, we examined the role of ferredoxin reductase (FDXR), a key mitochondrial regulator for iron metabolism, as p53-inducible gene with focusing on the hepatocyte and liver. We confirmed that p53 induced FDXR expression in HepG2 cells and SKEHP1 cells. Biochemical analysis demonstrated that FDXR regulated ROS levels via iron metabolism. In vivo analysis, high-fat diet activated the p53-FDXR pathway in mice liver. We generated transgene expression in mice liver using adenovirus infection carrying shRNA or CRISPR Cas9 system. Treatment with the FDXR knockdown increased hepatic iron content and aggravated glucose intolerance. Besides, forkhead box protein O1 (FOXO1), a key transcriptional factor that induces phosphoheonopyruvate carboxylase and glucose-6-phosphatase increased ratio of nuclear localization, indicating hepatic gluconeogenesis activation. Consistently, biochemical analysis in HepG2 cells demonstrated that FDXR regulated insulin-dependent FOXO1 nuclear exclusion through oxidative stress.

In conclusion, p533-inducible FDXR regulates iron metabolism and oxidative stress. FDXR inhibits iron accumulation and oxidative stress in liver and links to suppression of hepatic gluconeogenesis via insulin-dependent FOXO1 nuclear exclusion. The results of this study provide important new insights into relationship between iron metabolism and glucose metabolism as well as potentially identify novel therapeutic targets for the treatment of diabetes and NAFLD.

Pediatric Endocrinology
PEDIATRIC PUBERTY, TRANSGENDER HEALTH, AND GENERAL ENDOCRINE
Usefulness of a LHRH Test with Low Dose of Triptorelin Pamoate in the Diagnosis of Precocious Puberty in Girls
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SUN-063
Objective: to determine diagnosis value of a new LHRH test for diagnosis or precocious puberty (PP) correlated with clinical and paraclinical pubertal changes. Methods: 79 girls under age 10 years old were referred to our laboratory with diagnosis of precocious puberty went thought a physical exam and bone age /pelvic US review to classify them clinically in probably PP or unlikely PP. A LHRH test was performed with measurement of at least 3 times including baseline measurement of gonadotrophins (LH / FSH) and
Thyroid
THYROID DISORDERS CASE REPORTS II
Pembrolizumab-Induced Thyroiditis with Negative Thyroid Peroxidase Antibody
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SAT-481
Introduction: Immune checkpoint inhibitors (ICI) have re-formed oncology treatment through its immunomodulatory effect on T-lymphocytes to target metastatic and locally advanced cancers but have been known to produce immune-related adverse events (irAEs). Thyroiditis is a well-documented endocrinopathy occurring in patients receiving ICI; however, a Thyroid Peroxidase Antibody (TPO Ab) negative case of ICI induced thyroiditis suggests that its pathogenesis is independent of antibody mediated thyroid destruction and more associated with an alternative immunoregulatory mechanism. Case Description: A 60-year-old Caucasian, male with a 37-year smoking history and lung adenocarcinoma with metastasis to the brain was referred to Endocrinology clinic for evaluation of suppressed thyroid stimulating hormone (TSH) level. Patient was treated with a four-month course of IV Pembrolizumab every three weeks. TSH was <0.015 (NL 0.465-4.680 IU/mL) four weeks before being seen at the office. TSH level was normal 2.359 before starting immunotherapy. Patient reported occasional anxiety and heat intolerance, but did not experience other hyperthyroid symptoms. Physical examination in office demonstrated no significant thyromegaly, nodules, or tenderness. Vital signs were normal. Thyroid function tests obtained during the office visit were consistent with subclinical hypothyroidism. TSH was mildly elevated 7.545 with normal Free T4 of 0.94 (NL 0.78 - 2.19 ng/dL) and normal Free T3 level of 3.61 (NL 2.77 - 5.27 pg/mL). TPO antibodies were negative. Four weeks later, patient developed overt hypothyroidism; TSH level was higher 12.437 with low Free T4 of 0.71. Patient was then complaining of fatigue and cold intolerance. A diagnosis of drug-induced thyroiditis from Pembrolizumab was made. The patient was prescribed levothyroxine 75 mcg daily and followed closely. Discussion: While literature exists documenting the rare side effect profile of ICI endocrinopathies, few studies illustrate the implications and correlations of TPO Ab negative findings in ICI induced thyroiditis. The role of thyroid autoantibodies in the presumed antibody mediated pathogenesis of thyroid abnormalities is unclear and warrants further longitudinal studies to determine its function in these patients. This case report hopes to both identify the deficit of pathophysiological knowledge contextualizing irAEs while encouraging current healthcare practitioners to continue close monitoring of patients receiving ICI.

Tumor Biology
TUMOR BIOLOGY: DIAGNOSTICS, THERAPIES, ENDOCRINE NEOPLASIAS, AND HORMONE DEPENDENT TUMORS
Distinct DNA Methylation Signature in Neuroendocrine Tumors of Different Primary Sites and Hereditary Predisposition
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SUN-115
Objective
There is scant data of the genome-wide methylome alterations in neuroendocrine tumors (NET). Thus, the goal of this study was to compare the DNA methylation signature of NETs with respect to various primary sites and inherited genetic predisposition syndromes including von Hippel-Lindau (VHL) and multiple endocrine neoplasia type 1 (MEN1).

Methods
 Genome-wide DNA methylation analysis of 96 NETs (primary and metastatic) was performed by using the Illumina Infinium EPIC Array. Principal component analysis (PCA) and unsupervised clustering analyses were performed to identify distinct methylome signatures. The methylation status of genetic drivers such as APC were assessed by primary site.

Results
A total of 835,424 CpGs methylation sites were quantified. Hypermethylated CpG sites were detected more frequently in sporadic vs. MEN1-related vs. VHL-related NETs, respectively (p < 0.001 for all comparisons), while hypomethylated CpGs sites were more common in VHL-related NETs vs. sporadic and MEN1-related NETs (p<0.001 for both comparisons). Small-intestinal NETs (SINETs) had the most differences at CpGs with the highest number of hyper- and hypomethylated CpG sites, followed by duodenal NETs.