necrosis factor in the VAT and a trend (evident by effect size analysis only) for an increase in SAT and PCAT and 3) significant increase in PRAT and a trend toward increase in the macrophage marker CD68 expression in VAT. Among the thermogenic gene markers, the expression of UCP1 was significantly increased in VAT and PCAT with a trend for an increase in PRAT. The expression of UCP2 and PPAR gamma co-activator 1 beta (PPARGC1B) were also significantly elevated in VAT with a trend for an increase in SAT. These findings are indicative of depot-specific differences in prenatal T-induced inflammatory status with effects being pronounced in VAT compared to other depots. The increases in thermogenic markers in adipose depots do not support our hypothesis but rather are reflective of a compensatory response to promote adipose depot insulin sensitivity and may have a bearing on function of organs in the proximity of respective depots. These findings are likely of translational significance in metabolic disorders associated with hyperandrogenic state.

Tumor Biology
ENDOCRINE NEOPLASIA CASE REPORTS II
Primary Neuroendocrine Tumor of the Central Nervous System, a Case Report and Literature Review
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MON-919
Introduction
Neuroendocrine Neoplasms are rare, with an incidence of 5 to 100,000 inhabitants, constituting 1% of all malignancies, presenting high survival rates in general, even in metastatic diseases. However, in those poorly differentiated, as in the following case, survival is around 4% in 5 years. We will describe a case of primary neuroendocrine tumor in the brain, of which is uncommon in the literature. Clinical case
A 26 years women was referred to the ER of Santa Casa de São Paulo, in January 2019, to be evaluated by neurosurgery, due to progressive left hemiparesis and headache for 3 months, which got worse in 4 days. On CT scan, there was a 6 x 6 cm solid-cystic, expansive, lesion in the right frontal lobe, with perilesional edema and contralateral midline 1.3cm deviation and subfalcine herniation. Thus, the tumor was resected soon, with anatomopathological analysis showing poorly differentiated tumor of cells with scarce cytoplasm, hyperchromatic nuclei and high mitotic activity. The patient was once again submitted to emergency neurosurgery, with microsurgical resection. The pathology was identical to the previous one. We proceed with hormonal evaluation, regarding to Medullar Thyroid carcinoma, Gastrinoma, Insulinoma, Pheochromocytoma, Carcinoid tumor and others. Imaging exams were also performed to investigate other primary sites: no changes in CT scan of the chest and abdomen and PET CT FDG. However, this one showed recurrence of the intracranial lesion, with three sites of involvement, all hypermetabolic: one of 4.1 x 2.9 cm (SUV 4.9) and another of 3.9 x 3.3 cm (SUV 8, 4) in the right frontoparietal region and medial nodule to the right thalamus of 1.2 cm (SUV 6.1).

Patient currently maintain left hemiparesis, frequent pain, taking carbamazepine due to epileptic seizures, and considerable weight loss. She has an important limitation of daily activities and basic self-care, with 50% Karnofsky scale. Due to relapse, palliative radiotherapy was initiated in the region of the tumors.

Conclusion
The patient had a poor outcome in relation to cancer, with little possibility of treatment due to poor tumor differentiation and poor performance status.

Adipose Tissue, Appetite, and Obesity
RARE CAUSES AND CONDITIONS OF OBESITY: PRADER WILLI SYNDROME, LIPODYSTROPHY
Identification of NASH Using Data from NHANES III
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SUN-606
Nonalcoholic steatohepatitis (NASH) is a serious liver condition marked by hepatic steatosis (HS), cell damage and inflammation. Patients with NASH are at risk for developing cirrhosis and hepatic cancer. Currently, the definitive method of diagnosing NASH is by liver biopsy. To avoid the costs and risks associated with biopsy procedures, there has been considerable effort to develop a non-invasive method of identifying patients with NASH. However, none of these methods has become accepted as a “gold standard.” Our objective was to compare three non-invasive methods of identifying NASH by using data from NHANES III (1988-1994) to determine variables associated with published formulas to identify NASH. We used ultrasound data to identify subjects with moderate - severe HS. Among those with HS, we identified the NASH population using either the HAIR score, the NASH liver fat score, or the Gholam score. The HAIR score was developed in a sample of obese patients, based on hypertension, insulin resistance and alanine transaminase (ALT) levels, and had an AUROC of 0.9, a sensitivity of 0.8, and a specificity of 0.89. The NASH liver fat score was developed in a Finnish population undergoing gastric bypass, and validated in an Italian population of liver biopsy patients. This score incorporates metabolic syndrome, type 2 diabetes, serum insulin, AST, and ALT. In the Finnish and Italian populations, respectively,
Diabetes Mellitus and Glucose Metabolism

TYPE 1 DIABETES MELLITUS

Transient Neonatal Diabetes Mellitus Triggered by EIF2AK3 and PTF1A Mutation

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SAT-681

Background: Neonatal diabetes mellitus (NDM) occurs within the first 6 months of life. Advances in molecular genetics have identified various causative genes. Mutations in EIF2AK3 cause Wolcott-Rallison syndrome characterized by NDM, multiple epiphyseal dysplasia and growth retardation. PTF1A is associated with the development of pancreas and cerebellum. Both EIF2AK3 and PTF1A mutations are causative genes for permanent NDM with spontaneous and autosomal recessive inheritance. We report a neonate with transient NDM with both EIF2AK3 and PTF1A variants confirmed by Sanger sequencing where each parent found to be a heterozygous carrier of each mutation. Case presentation: A two-day old boy was transferred from a local hospital due to hyperglycemia (blood glucose of 385 mg/dL) and glycosuria. Serum c-peptide (0.06 ng/mL) and insulin (0.64 μU/mL) were low. The patient did not present signs of ketoacidosis and was screened negative for pancreatic autoantibodies. The patient did not have any family history of diabetes. Molecular genetic analysis was performed and continuous infusion of intravenous insulin with pre-prandial bolus was started.

Diabetes Mellitus and Glucose Metabolism

ISLETS, LIVERS, PLACENTA, AND VASCULATURE — THE MULTITISSUE IMPACT OF DIABETES

Circadian Regulation of Chromatin State Mediates Pancreatic Islet Incretin Response

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OR14-02

Circadian Regulation of Chromatin State Mediates Pancreatic Incretin Response

The circadian clock is programmed by an autoregulatory transcription feedback loop present in brain and peripheral tissues that coordinates metabolism with nutritional state and the sleep-wake cycle. Epidemiologic and genetic studies indicate circadian disruption as a risk factor in the development of diabetes. We have demonstrated that conditional ablation of the β cell clock in adult life leads to hypoinsulinemic diabetes, and through mRNA-sequencing in mouse and human islets we revealed clock control of gene networks involved in insulin secretion, nutrient sensing, and exocytosis. A remaining question is: How does the core molecular clock modulate time-of-day dependent chromatin state to regulate pancreatic islet response to glucose and insulin secretagogues? Here we report that loss of the pancreatic β cell molecular clock results in closed chromatin at cAMP-responsive gene regulatory elements and dysregulated cAMP-dependent coregulator recruitment following cAMP agonism, consistent with a role for the molecular clock in mediating cell response to environmental stimuli. Further, tandem analyses of ATAC- and ChIP-sequencing in synchronized islets revealed dynamic chromatin accessibility across the 24-hour cycle at genes regulating insulin secretion and at genomic regions.