178 nonfunctioning adenomas). Twenty-three patients, 17 with Cushing disease, (CD), mean age: 31 y, 13 female and 4 male) and 6 patients with SCA (mean age: 47 y, 5 female and 1 male) had positive ACTH confirmed by immunohistochemical analysis. Clinical characteristics: in the CD group, 53% had hypertension (9/17), 42% diabetes (7/17), 100% dyslipidemia, BMI was 30.7 kg/m^2. Among SCA group, 67% hypertension, 50% diabetes, 50% dyslipidemia, BMI was 28 kg/m^2. All patients were evaluated with basal ACTH and DHEAS before surgery. Patients with SCA underwent desmopressin test and were compared to CD. Dexamethasone suppression test (DST 1 mg) and 24-hour free urinary test was performed in patients with CD and in two patients with SCA. Response to desmopressin test was considered positive when increase in cortisol was above 20% and in ACTH of 35% using chemiluminescence assay (Immulite 2000).
Results: Among CD group, the median (med) basal ACTH was 75.9 pg/mL (30.9 to 211), the med basal cortisol was 22.5 µg/dL (14.5 to 43.5), the med DHEAS was 170 µg/dL (33 to 465), the med 24h urinary free cortisol of 454.5 µg/24 h (149 to 1673) and med basal cortisol after DST 1mg of 15.4 µg/dL (4.7 to 31.5). Among SCA, med basal ACTH was 19.4 pg/mL (9.5 to 65.5), the med basal cortisol was 9.5 µg/dL (7.8 to 16.4) and the med DHEAS was 104.5 µg/dL (82 to 127). Only 4 patients with CD had macroadenomas. All of them responded with ACTH increase (med increase of 98%, 31.6 to 377%), and only 4 did not respond to cortisol increase (med increase of 54.4%, 0 to 167%). All patients with SCA had macroadenomas. Only one patient did not respond to ACTH increase (med increase of 123.5%, 9.5 to 1522%, 9.5 to 1522%), and 3 patients did not respond to cortisol increase (med increase of 17.9%, 0 to 234%).
Discussion: SCA are invasive tumors, with high recurrence and tests predicting their occurrence
are missing. We hypothesized that as ACTH is present in the adenoma a response to desmopressin test could exist (like CRH).
Conclusion:
The desmopressin test can be a useful tool in the evaluation of SCA and can predict pathological phenotype in preoperative tumors.

Diabetes Mellitus and Glucose Metabolism

CLINICAL AND TRANSLATIONAL STUDIES IN DIABETES

The Relationship Between Glucose Control & Cognitive Function in People with Diabetes After a Lacunar Stroke

Tali Cukierman-Yaffe, Dr., MD, MSC[1], Leslie McClure, Prof., PhD[2], Thomas Risoli, MSC[3], Jackie Bosch, Prof., PhD[4], Hertzel C. Gerstein, MS,MD,MSC,FRCPC[5], Oscar Benavente, Prof., MD[6].

[1]Endocrinology Institute, Sheba Medical Center, Epidemiology department, Sackler School of Medicine, Herceg institute on aging Tel-Aviv university, Tel-Aviv, Israel, [2]Department of Epidemiology & Biostatistics Dornsife School of Public Health, Drexel University, Philadelphia, PA, USA, [3]Duke CTSI Biostatistics, Epidemiology and Research Design (BERD) Methods Core Duke University School of Medicine,Durham, NC, Durham, NC, USA, [4]School of Rehabilitation Sciences, McMaster University, Hamilton, Canada, Hamilton, ON, Canada, [5]McMaster University, Hamilton, ON, Canada, [6]Department of Medicine, Division of Neurology, University of British Columbia, Vancouver, Vancouver, BC, Canada.

MON-636

Background & Objective Both lacunar strokes and diabetes are risk factors for dementia and cognitive dysfunction. Thus, elucidating modifiable risk factors for cognitive dysfunction in people with type 2 diabetes who experienced a lacunar infarct has large public health implications. One such factor may be glycemic status, as measured by glycosylated hemoglobin (A1C). Thus, the aim of this study was to assess the relationship between A1C and cognitive function in people with diabetes after a lacunar stroke. **Research Design & Methods** The effect of baseline and follow-up A1C on the baseline and the change in Cognitive Assessment Screening Instrument (CASI) score over time among participants with a median of 2 cognitive assessments (range 1-5) was examined in of 942 individuals with diabetes and a lacunar stroke who participated in the Secondary Prevention of Small Subcortical Strokes (SPS3) trial (ClinicalTrials.gov number, NCT00059306). **Results** Every 1 % higher baseline A1C was associated with a 0.06 lower standardized CASI z-score (95% CI -0.101, -0.018). Higher baseline A1C values were associated with lower CASI z-score over time (p for interaction=0.037). A 1% increase in A1C over time, corresponded with a CASI score decrease of 0.021 (95% CI -0.0043, -0.038) during follow-up. All these remained statistically significant after adjustment for age, sex, education, race, depression, hypertension, hyperlipidemia, BMI, CVD, OSA, diabetic retinopathy, nephropathy insulin use and White Matter Abnormalities. **Conclusion** This analysis of 942 individuals with diabetes after a lacunar stroke demonstrates a relationship between A1C and change in cognitive scores over time. Intervention studies are needed in order to delineate if better glucose control could slow the rate of cognitive decline in this high risk population.

Pediatric Endocrinology

PEDIATRIC OBESITY, THYROID, AND CANCER

Plasma Insulin Measured with a Sensitive Immunoassay May Establish the Diagnosis of Congenital Hyperinsulinism Without Further Testing.

Julie Siersbæk, Graduate Student, Annette R. Larsen, M.Sc., PhD student, Mads Nybo, MD, PhD, Henrik Boye Thybo Christesen, MD, PhD, Professor.
Odense University Hospital, Odense, Denmark.

MON-111

Plasma insulin measured with a sensitive immunoassay may establish the diagnosis of congenital hyperinsulinism without further testing.

Abstract
Background: The diagnosis of congenital hyperinsulinism (CHI) is often hampered by a plasma insulin (p-insulin) detection limit of 2-3 mU/L (14-21 pmol/L) by RIA methods.

Objective: To evaluate the diagnostic performance of a sensitive immunoassay for p-insulin and to find the optimal p-insulin cut-off for CHI versus other conditions with hypoglycaemia.
Design: Single centre retrospective cohort study.
Methods: Diagnostic tests with no medication, no i.v. glucose and under fasting conditions were performed in children with a clinical diagnosis of CHI. P-insulin concentrations determined at simultaneous p-glucose concentrations at least <3.2 mmol/L (57.5 mg/dL) were included in the analysis (n=61).
The diagnosis of CHI was either clinical (n=61) or by gold standard criteria: hypoketotic hypoglycaemia plus disease-causing genetic mutations and/or diffuse, focal or atypical pancreatic histopathology (n=57). Samples from 15 children with idiopathic ketotic hypoglycaemia (IKH, diagnosis by exclusion, p-ketones >1.5 mmol/L during hypoglycaemia) were used as controls.
P-insulin was measured by the high-sensitive assay (Cobas e411 immunoassay analyzer); lower detection limit 1.4 pmol/L (0.2 mU/L); normal range 18-173 pmol/L (2.57-24.7 mU/L). Concentrations <18 pmol/L were considered suppressed; ≥18 pmol/L un-suppressed.
Receiver operating characteristics (ROC) curves with determination of area under the curve (AUC) values were performed for the diagnostic performance of p-insulin in the diagnosis of CHI. *Results:* In the 61 samples from CHI patients, the median (range) p-insulin was un-suppressed in all diagnostic samples [90; 20-758 pmol/L (12.9; 2.9-109.1 mU/L)], while p-insulin was suppressed in all 15 samples from IKH patients [1.5; 1.5-9 pmol/L (0.21; 0.21-1.3 mU/L)]. The ROC AUC was 1.0 (95%CI. 1.0-1.0) for the diagnosis of CHI defined both by the clinic and by gold standard. The optimal p-insulin cut-off was 14.5 pmol/L (2.1 mU/L) or 12.5 pmol/L (1.8 mU/L), for CHI patients by use of a simultaneous p-glucose cut-off of <3.2 mmol/L (57.5 mg/dL; n=61), or 3.0 mmol/L (55 mg/dL; n=49), respectively.
Conclusions: The sensitive insulin assay performed excellent in diagnosing CHI with a ROC AUC of 1.0. The use of a p-insulin cut-off of 13 pmol/L (1.86 mU/L) during a diagnostic hypoketotic hypoglycaemia test may establish the diagnosis of CHI without further diagnostic testing.

Diabetes Mellitus and Glucose Metabolism

DIABETES TECHNOLOGY

Results of a Preclinical Pilot Study Evaluating 24-Hour Subcutaneous Infusion of the GLP-1 Analogue Liraglutide Delivered via the H-Patch Wearable Device

Jessica W. Barnes, PhD, Erick Lucera, MBA.
Valeritas, Inc., Bridgewater, NJ, USA.

SAT-635

Nonalcoholic fatty liver disease (NAFLD) affects an estimated 30% of Americans, is the most common cause of chronic liver disease in the US, and a leading cause of liver-related morbidity/mortality worldwide[1]. Currently, there are no FDA-approved drugs specifically tailored to NAFLD or its big brother, non-alcoholic steatohepatitis (NASH). Glucagon-like peptide 1 (GLP-1) is an incretin peptide hormone, secreted in the distal ileum and proximal colon by L cells. Besides stimulating the pancreas to cause beta cell proliferation and enhance insulin biosynthesis, GLP-1 interacts with receptors in other parts of the GI tract, lung, kidney and CNS. Thus, GLP-1's metabolic functions include delayed gastric emptying, appetite suppression, enhanced liver glucose uptake, peripheral insulin sensitivity, as well as glucose-dependent insulin secretion while inhibiting the release of glucagon from α-cells. While GLP-1 receptor agonists such as exenatide and liraglutide have been approved for type 2 diabetes, a meta-analysis of several studies has shown promise in patients with NASH. Effects including decreased serum ALT levels, improvement in hepatic fat content and fibrosis, and weight loss making GLP-1 therapeutics potentially attractive for use in patients with NASH and metabolic syndrome[2-5]. While subcutaneous injection is an avenue for administration, continuous infusion offers many benefits for native GLP-1 and GLP-1 analogues. We investigated the pharmacokinetics (PK) of a GLP-1 analogue (liraglutide) delivered over a single 24h period using the wearable h-Patch™ subcutaneous infusion device in dogs. Liraglutide (1800ug) was infused at a static rate over 24h delivered with PK evaluated at time points to 72h from the start of infusion. Liraglutide levels were detectable in blood detected within 30m of the beginning of infusion, peaked above the upper level of quantitation of the LC/MS/MS analysis (316ng/ml upper level of quantitation), and gradually decreased with quantifiable levels still detected 48h after completion of h-Patch™ infusion. The h-Patch™ provides a simple all-in-one delivery device with no exposed needle, no programming or infusion set required, additionally it can be configured to deliver two payload in separate reservoirs offering the potential option of an insulin/GLP-1 infusion combination. These preliminary encouraging results of liraglutide infusion via the h-Patch warrant further investigation in a variety of indications including diabetes, NASH, and obesity as a monotherapy and in combination with other complementary therapeutics. 1. Liver Int. 2017; 37(S1):97-103. 2. Lancet. 2016; 387(10019):679-690. 3. J Gastroenterol Hepatol. 2016; 31(1):23-31. 4. Clin Ther. 2007; 29(1):139-153. 5. Aliment Pharmacol Ther. 2013; 37(2):234-242.

Diabetes Mellitus and Glucose Metabolism

CLINICAL STUDIES IN OBESITY, DIABETES RISK, AND CARDIOVASCULAR OUTCOMES

Predictive Ability of Lipoprotein Insulin Resistance (LPIR) Score in South Asians: A Comparison of Surrogate Indices of Insulin Sensitivity/Resistance

Andin Fosam, BS[1], Shivraj Grewal, BS[1], Abdul-Latif Armiyaw, BA[1], Camila Sarcone, BS[1], Antoinette Rabel, MSN, FNP[1], Sungyoung Auh, PhD[1], Ranganath Muniyappa, MD PhD[2].
[1]National Institutes of Health, Bethesda, MD, USA, [2]National Institutes of Health Clinical Center Endocrine Fellowship Program, Bethesda, MD, USA.

SAT-624

South Asians (SA) are at higher risk for developing insulin resistance (IR) and type 2 diabetes. Consequently, identifying IR in this population is important. Lack of