PCOS patients have a greater risk of having metabolic disorders, such as insulin resistance and cardiovascular diseases, but it is estimated that up to 75% of women remain undiagnosed. Delayed treatment and care can exacerbate comorbid conditions and be detrimental to high risk populations like African American and Hispanic women. We aim to characterize genetic and environmental variables contributing to PCOS and understand its shared etiological features with metabolic disorders. To do this, we developed two algorithms to identify diverse PCOS patients using medical records. The broad algorithm used a combination of PCOS-related billing codes (Code Based) and identified a large dataset (N = 8,340) who exhibited diverse PCOS symptoms, while the strict algorithm required PCOS keywords in addition to billing codes (Regex Based). The strict algorithm identified a smaller cohort of patients (N = 4,593) who exhibited more classically diagnoseable PCOS characteristics according to Rotterdam and NIH criteria. Using both datasets, we tested PCOS case status against 1,853 phenotypes in the medical database using a logistic regression model and identified comorbidity patterns for women of European and African descent. We observed that European descent women consistently had more distinct phenotypes associated with PCOS case status than African American women. Next, we examined the interacting effects of self-reported race on PCOS case status and found four significant phenotypes ($p < 6.25e-4$) in our Regex Based algorithm. African American women with PCOS had greater odds of being diagnosed with "Early onset of delivery" ($p = 1.3e-4$, OR = 1.86), "Hereditary hemolytic anemias" ($p = 1.8e-4$, OR = 0.65), and "Other hereditary hemolytic anemias" ($p = 3.7e-04$, OR = 0.90). Meanwhile, European descent women had greater odds of being diagnosed with "Hypertensive chronic kidney disease" ($p = 1.7e-04$, OR = 0.68). Results show that European and African American women have unique metabolic comorbidity patterns and it may also indicate that clinical PCOS diagnostic standards vary between these groups with possible disparity-causing effects.

Diabetes Mellitus and Glucose Metabolism

DIABETES TECHNOLOGY

Flash Glucose Monitoring Helps Achieve Better Glycemic Control Than Conventional Self-Monitoring of Blood Glucose in Non-Insulin-Treated Type 2 Diabetes: A Randomized Controlled Trial

Eri Wada, MD[1], Takeshi Onoue, MD, PhD[1], Tomoko Kobayashi, MD, PhD,[1], Tomoko Handa, MD[1], Ayaka Hayase, MD[1], Masaaki Ito, MD[1], Mariko Furukawa, MD[1], Takayuki Okuji, MD[1], Norio Okada, MD[1], Shintaro Iwama, MD, PhD[1], Mariko Sugiyama, MD, PhD[1], Taku Tsunekawa, MD, PhD[1], Hiroshi Takagi, MD, PhD[1], Daisuke Hagiwara, MD, PhD[1], Yoshihiro Ito, MD, PhD[1], Hidetaka Suga, MD, PhD[1], Ryoichi Banno, MD, PhD[1], Yachiyo Kuwatsuka, MD, PhD[2], Masahiko Ando, MD, PhD[2], Motomitsu Goto, MD, PHD[1], Hiroshi Arima, MD,PHD[1].

[1]Department of Endocrinology and Diabetes, Nagoya University Graduate School of Medicine, Nagoya, Japan, [2]Department of Advanced Medicine, Nagoya University Hospital, Nagoya, Japan.

SAT-648

Background and aims: Flash glucose monitoring (FGM) is a novel system with which glucose levels are monitored and has been reported to improve glucose variability and glycemic control in type 1 and type 2 diabetes patients treated with insulin. The present study aimed to evaluate the effects of FGM and conventional self-monitoring of blood glucose (SMBG) on glycemic control in patients with non-insulin-treated type 2 diabetes. **Reseach design and Methods:** In this 24-week, multicenter, open-label, randomized (1:1), parallel group study, non-insulin-treated type 2 diabetic patients at 5 hospitals in Japan were randomly assigned to the FGM (n = 49) or SMBG (n = 51) groups and were provided FGM or SMBG devices for 12 weeks. The primary outcome was change in glycated hemoglobin (HbA1c) level. This trial is registered with UMIN-CTR (UMIN000026452). **Results:** Forty-eight participants in the FGM group and 45 in the SMBG group completed the study. The mean HbA1c levels were 7·83% (SD 0·25) in the FGM group and 7·84% (SD 0·27) in the SMBG group at baseline, and the values were reduced in both FGM (−0·43%; 95% confidence interval [CI], −0·57 to −0·28; $p < 0·0001$) and SMBG groups (−0·30%; 95% CI −0·48 to −0·13; $p = 0·001$) at 12 weeks. On the other hand, HbA1c was significantly decreased from baseline values in the FGM group, but not in the SMBG group at 24 weeks (FGM: −0·46%, 95% CI −0·59 to −0·32, $p < 0·0001$; SMBG: −0·17%, 95% CI −0·05 to 0·11, $p = 0·124$); a significant between-group difference was also observed (difference −0·29%, 95% CI −0·54 to −0·05; $p = 0·022$). Diabetes Treatment Satisfaction Questionnaire score was significantly improved, and the mean glucose levels, standard deviation of glucose, mean amplitude of glycemic excursions, and duration of hyperglycemia were significantly decreased in the FGM group compared with the SMBG group. **Conclusions:** Glycemic control was better with FGM than with SMBG after cessation of glucose monitoring in non-insulin-treated type 2 diabetic patients.

Bone and Mineral Metabolism

PARATHYROID HORMONE TRANSLATIONAL AND CLINICAL ASPECTS

Neonatal Hypocalcemic Seizures in Offspring of a Mother with Familial Hypocalciuric Hypercalcemia Type 1 (FHH1)

Fadil M. Hannan, DPhil[1], Poonam Dharmaraj, MB ChB[2], Caroline M. Gorvin, DPhil[3], Astha Soni, MB BS[2], Nick D. Nelhans, MB ChB[4], Mie Kronborg Olesen, MSc[5], Hannah Boon, MSc[6], Treena Cranston, MSc[6], Rajesh V. Thakker, MD[1].

[1]University of Oxford, Oxford, United Kingdom, [2]Alder Hey Childrens Hospital, Liverpool, United Kingdom, [3]University of Birmingham, Birmingham, United Kingdom, [4]Wrexham Maelor Hospital, Wrexham, United Kingdom, [5]University of Liverpool, Liverpool, United Kingdom, [6]Oxford University Hospitals NHS Foundation Trust, Oxford, United Kingdom.

SAT-404

Background: Familial hypocalciuric hypercalcemia type 1 (FHH1) is caused by loss-of-function mutations of the calcium-sensing receptor (CaSR), and considered to be a benign condition associated with mild-to-moderate hypercalcemia (1). However, the children of parents with FHH1 can develop a variety of disorders of calcium homeostasis in infancy.

Objective: To further characterise the range of calcitropic phenotypes in the children of a mother with FHH1.
Methods: We assessed a three generation FHH kindred by clinical, biochemical and mutational analysis following informed consent.
Results: The kindred comprised a hypercalcemic male, his daughter who had hypercalcemia and hypocalciuria, and her four children, of whom two had asymptomatic hypercalcemia, one was normocalcemic, and one suffered from transient hypocalcemic seizures during infancy. The hypocalcemic infant had a serum calcium of 1.57 mmol/L (normal, 2.0-2.8) and PTH of 2.2 pmol/L (normal, 1.0-9.3) as a consequence of maternal hypercalcemia, and required treatment with I-V calcium gluconate infusions. Mutational analysis identified a novel heterozygous p.Ser448Pro CaSR variant in the hypercalcemic family members, but not in the children with hypocalcemia or normocalcemia. Three-dimensional modelling using a reported crystal structure of the dimeric CaSR showed the mutated Ser448 residue to be located in the CaSR extracellular domain, and predicted the p.Ser448Pro variant to disrupt a hydrogen bond interaction across the extracellular CaSR dimer interface. The variant Pro448 CaSR, when expressed in HEK293 cells, was shown to significantly impair CaSR-mediated intracellular calcium mobilisation and mitogen-activated protein kinase (MAPK) responses following stimulation with extracellular calcium, thereby demonstrating it to represent a loss-of-function mutation.
Conclusion: These studies have identified a novel loss-of-function CaSR mutation which caused asymptomatic hypercalcemia in a mother and her children who had inherited the mutation. However, one child who did not inherit the mutation developed transient neonatal hypocalcemic seizures as a consequence of maternal hypercalcemia. These findings highlight the importance of assessing serum calcium and undertaking CaSR mutational analysis in the newborn offspring of a mother with FHH1.
Reference: (1) Hannan FM, Kallay E, Chang W, Brandi ML, Thakker RV. The calcium-sensing receptor in physiology and in calcitropic and noncalcitropic diseases. *Nat Rev Endocrinol*. 2018; 15(1): 33-51.

Thyroid

BENIGN THYROID DISEASE AND HEALTH DISPARITIES IN THYROID II

The Impact from AJCC 8Th Edition Staging System on Thyroid Cancer Outcomes by Race And Ethnicity

Juan Antonio Santamaria-Barria, MD[1], Amanda N. Graff-Baker, MD[2], Shu-Ching Chang, PhD[3], Adam Khader, MD[1], Anthony J. Scholer, MD[1], Mary Kledzik-Garland, MD[1], Melanie Goldfarb, MD[1].
[1]John Wayne Cancer Institute at Providence Saint John's Health Center, Santa Monica, CA, USA, [2]Kaiser Permanente San Jose Medical Center, San Jose, CA, USA, [3]Medical Data Research Center at Providence St. Joseph Health, Portland, OR, USA.

SUN-426

Background. Previous studies have demonstrated racial and ethnic outcome disparities among differentiated thyroid cancer (DTC) patients. However, the impact of the 8th edition of the American Joint Committee on Cancer staging system (AJCC8) on these disparities is unknown.
Methods. DTC patients with sufficient tumor and survival data were identified in the National Cancer Database from 2004-2013. The 7th edition of the staging system (AJCC7) and AJCC8 criteria were compared. Multivariable logistic regression was used to evaluate the association between AJCC7 to AJCC8 staging change and race and ethnicity. Cox-proportional hazards regression was then used to evaluate the association between AJCC7 to AJCC8 staging change and overall survival.
Results. Of 33,323 DTC patients, 76.7% were White/Non-Hispanics, 7.6% Blacks, 6.7% Hispanics, 5.4% Asian/Pacific-Islanders, and 3.6% Native-American/Other. Most were female (77%) with papillary DTC (90%). After adjusting for demographic, tumor, and treatment characteristics, Hispanics and Asian/Pacific-Islanders were 27% and 12% less likely to be AJCC7 to AJCC8 downstaged than White/Non-Hispanics (OR=0.73, 95%CI: 0.66-0.81; and OR=0.88, 95%CI: 0.79-0.99, respectively); Blacks had no significant downstaging difference compared to White/Non-Hispanics (OR=0.99, 95% CI: 0.90-1.09, p=0.79). Although AJCC8 was a better survival prognosticator than AJCC7, Cox-proportional hazards regression showed that all AJCC7 to AJCC8 downstaged patients had an increased risk of death compared to patients with unchanged staging, regardless of race and ethnicity: White/Non-Hispanics (HR=2.64, 95%CI: 2.34-2.98), Blacks (HR=1.77, 95%CI: 1.23-2.54), Hispanic (HR=3.27, 95%CI: 2.05-5.22), Asian/Pacific-Islanders (HR=2.31, 95%CI: 1.35-3.98), and Native-American/Other (HR=5.26, 95%CI: 2.10-13.19). However, based on two way interaction, the magnitude of negative change in survival from downstaging was only different between White/Non-Hispanics and Blacks (HR=2.64 vs. HR=1.77, respectively; p=0.04).
Conclusions. Racial and ethnic outcome disparities persist with AJCC8. The proportion of downstaged DTC patients with AJCC8 varies by race and ethnicity, with the least impact found in Hispanics and Asian/Pacific-Islanders. Downstaged patients across all racial and ethnic groups had a decreased survival than those with unchanged stage, with the least impact in Blacks. These disparities should be taken into account when counseling patients about their prognosis with the new AJCC8.

Pediatric Endocrinology

PEDIATRIC ENDOCRINE CASE REPORTS I

A De Novo 1p13.2 Deletion Related to Short Stature, Hypothyroidism and Mild Developmental Delay.

Elpis Athina Vlachopapadopoulou, MD, MSc[1], Eirini Dikaiakou, MD[1], Ioannis Panagiotopoulos, MD[1], Ioannis Papoulidis, B.Sc, PhD[2], Emannouil Manolakos, B.Sc, PhD[2], Stefanos Michalacos, MD, PhD[1].
[1]Department of Endocrinology, Growth and Development "P.&A. Kyriakou" Children's Hospital, Athens, Greece, [2]ATG Clinical Laboratory Genetics, Athens, Greece.

SAT-055

Background: Chromosomal deletions may lead to variable phenotypic alterations, depending on which loci and genes are deleted. We present the case of a boy with a de