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.