increased proliferation and activin signalling, respectively. These findings suggest that the balance between cellular proliferation and differentiation might be altered in the absence of FSTL3. Thus, we conclude that FSTL3 function, at least partly through the inhibition of activin action, is necessary for normal placental circulation and development.

Diabetes Mellitus and Glucose Metabolism

LIPIDS, OBESITY AND METABOLIC DISEASE

Ethnic Differences in the Relationship Between Uric Acid Clearance and Insulin Sensitivity

Abdul-Latif Armiyaw, BA[1], Andin Fosam, BS[1], Camila Sarcone, BS[1], Ahimsa Aradhya, N/A[1], Mary Walter, PHD[1], Antoinette Rabel, MSN, FNP[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-651

Background: Hyperuricemia is associated with gout, type-2 diabetes, hypertriglyceridemia, and other insulin resistant (IR) states. Impaired clearance of uric acid (UA) has been proposed to play a role in hyperuricemia in IR. In a small study, urinary UA clearance was negatively associated with IR. African-Americans (AA) are more insulin resistant than Caucasians (CA). However, plasma UA levels are lower in AA. Ethnic differences underlying the relationship between UA clearance and insulin sensitivity (Si) remain unknown.
Objective: To compare the relationships between UA production, clearance, and Si in AA and CA.
Design: In a cross-sectional study, AA (n = 40; age 43 ± 10 years; BMI 41.3 ± 9.7 kg/m^2) and CA (n = 88; age 44 ± 13 years; BMI 32.9 ± 8.2 kg/m^2) subjects underwent an intravenous glucose tolerance test (IVGTT) to derive Si using the Minimal Model. Plasma UA and creatinine (Cr), were measured in the NIH Department of Laboratory Medicine. Urinary UA and Cr levels in spot urine samples were measured using a colorimetric assay. Fractional Excretion (FE) of UA and urine urate-to-creatinine ratio (UUCR), a measure of uric acid production, were calculated.
Results: AA had a significantly higher BMI ($p < 0.0001$), percent body fat (45.8 ± 8.9 vs. 39.7 ± 11.5 %, $p = 0.0007$), A1C (5.8 ± 0.4 vs. 5.5 ± 0.3 %, $p = 0.0006$), diastolic BP (74 ± 9 vs. 71 ± 8 mm of Hg, $p = 0.02$), and lower Si (2.36 ± 2.7 vs. 4.43 ± 3.4 min−1·μU·ml−1, $p = 0.0004$) compared to CA. Stepwise multiple regression analysis was performed with independent variables A1c, fasting glucose, fasting insulin, triglycerides, and Si. Among these, Si was a significant predictor of FE of UA in CA ($r = 0.33$, $p=0.001$), but not AA ($r = - 0.02$, $p=0.92$).There was also a negative association between Si and plasma UA in CA ($r = 0.48$, $p < 0.0001$), but not in AA ($r = 0.06$, $p = 0.70$). There was no association between Si and UA production in both groups (AA: $p = 0.33$, CA: $p = 0.69$).
Conclusion: These findings suggest that reduced insulin sensitivity may not play a major role in the pathogenesis of hyperuricemia in AA in insulin-resistant states.

Adrenal

ADRENAL CASE REPORTS II

A Complex Case of Adrenal Insufficiency Associated with NLRP1 Gene Mutation in a Patient with Myopathy and Mitochondrial Cytopathy

Mehvish Khan, MD[1], Masuma Khawaja, MD[1], Mohammad Adnan Ul Haq, MD[1], Humza Quadir, MD[1], Brian C. Jameson, DO[2].
[1]The Wright Center for Graduate Medical Education, Scranton, PA, USA, [2]Geisinger Health System, Mountain Top, PA, USA.

SUN-155

A Complex Case of Adrenal Insufficiency Associated with NLRP1 Gene Mutation in a Patient
with Myopathy and Mitochondrial Cytopathy
Most cases of Addison's disease are due to an autoimmune response, with the most commonly
associated genes belonging to human leukocyte antigen (HLA) complex. Genome wide
association studies have shown a significant association of variants of Nuclear Localization
Leucine-Rich-Repeat Protein 1 (NLRP1) with Addison's disease. NLRP1 protein is involved in
the assembly of inflammasome which promotes the secretion of interleukin-1β, interleukin-18
and downstream inflammatory responses to regulate inflammation. With underlying myopathy
and mitochondrial disease, coexisting adrenal insufficiency may be challenging to identify.
A 36-year-old female presented for evaluation of fatigue, myalgia, and dyspnea for several years.
She carried a diagnosis of asthma, myopathy, gastroparesis requiring a gastric stimulator, and
recently diagnosed adrenal insufficiency secondary to long term fluticasone use. Beside low
blood pressure of 91/64 millimetres of mercury, physical exam was unremarkable. Lab findings
were significant for dehydroepiandrosterone-sulfate (DHEAS) of 7.0 micrograms per deciliter
(mcg/dL), Adrenocorticotropic hormone (ACTH) of 7.2 picograms per milliliter and cortisol of
1.4 mcg/dL. Adrenal insufficiency was confirmed with cosyntropin stimulation test.
Methacholine challenge test showed worsening asthma. She was managed with empiric stress
dose steroids when indicated. Muscle fatigue progressed despite taking ubiquinol, B100 and
carnitine. She was further evaluated with muscle biopsy that showed type two fiber atrophy.
Muscle coenzyme *Q*10 was 0.08 mcg/dL, and citrate synthase was 50% of normal, insufficient for
electron transport complex I. Whole exome sequencing showed mutations in NLRP1 in addition
to Myosin Heavy Chain 2 (MYH2) and Sodium voltage-gated Channel alpha subunit 4 (SCN4a)
both of which are associated with myopathy. She was then started on a short-acting
glucocorticoid regimen.
While her adrenal insufficiency was initially thought to be secondary to inhaled steroids,
subsequent mutation analysis suggested that she was prone to autoimmunity. This case illustrates