

syndrome (PCOS). Early risk factors and subclinical CVD includes atherogenic dyslipidemia, obesity, insulin resistance, blood pressure, atherosclerosis and impaired cardiac function. Androgen exposure is associated with onset of adiposity, impaired insulin-glucose and lipid metabolism, and cardiac dysfunction. The mechanisms of increased risk of CVD and cardiac dysfunction in PCOS related to hyperandrogenemia, AR and estrogen receptor (ER) activation remain unclear. **Aim:** The aim of this study was to investigate the effect of androgen treatment on cardiac AR and ER activation, fatty acid metabolism and cardiac function in a PCOS-prone rodent model. **Methods:** A PCOS-prone rodent model at 6 wks of age with obesity, apoB-remnant lipemia and insulin resistance, and controls were treated with testosterone for 12 weeks. Cardiac function was assessed using transthoracic doppler echocardiography (M-Mode 2D-imaging), lipogenic, AR, ER and other metabolic gene and protein expression were assessed using RTPCR and SDS-PAGE western blot. **Results:** PCOS-prone animals exhibited left ventricular (LV) hypertrophy, with increased LV mass to body weight (551.6 ± 38.85 mg vs 999 ± 96.17 mg, $p < 0.05$), LV posterior wall diastolic diameter and LV internal diastolic diameter compared to controls. Isovolumetric relaxation time (IVRT) was prolonged (15.91 ± 1.591 msec vs 23.75 ± 0.722 msec, $p < 0.05$). Mild systolic dysfunction was evidenced by increased isovolumetric contraction time (IVCT; 22.5 ± 1.348 msec vs 28.96 ± 1.248 msec, $p < 0.05$) and decreased % ejection fraction and % fractional shortening in PCOS-prone compared to controls. T treatment increased LV mass, IVCT and IVRT in controls but did not exacerbate cardiac function in PCOS-prone animals. T treatment increased cardiac protein expression of PPAR- α in PCOS-prone and controls, and T increased ACC in controls. AR protein expression tended to be reduced, and ER- α was reduced in both T treated control and PCOS-prone animals. **Conclusions:** The PCOS-prone rodent model demonstrates early cardiac LV hypertrophy and diastolic-systolic dysfunction and T treatment alters fatty acid metabolism, and AR and ER activation are associated with altered cardiac morphology and function in the PCOS-prone and control conditions.

Cardiovascular Endocrinology

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Association Between Thyroid Hormones and Lipids Stratified by Race and Sex

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It has been well-established that thyroid hormones play a role in cholesterol and lipoprotein metabolism. However, there is limited data assessing the variability in the association between thyroid hormones and lipids across sex and race. We hypothesized that thyroid dysfunction is associated with changes in lipids and lipoproteins with no substantial variability in this association between races and sex. The electronic medical record of a large county hospital in Dallas, TX was queried to obtain data on all patients who had lipid panels and thyroid function tests checked on the same day from 1/1/2013 to 1/1/2018. The results were

stratified into hypothyroid (TSH greater than 4.5 mcIU/L and Free T4 less than 0.8 ng/dL), hyperthyroid (TSH less than 0.5 mcIU/L and Free T4 greater than 1.8 ng/dL) and normal (TSH between 0.5 and 4.5 mcIU/L, Free T4 between 0.8 and 1.8 ng/dL). Results consistent with subclinical thyroid disease were excluded from further analysis. There were 25,290 unique results for thyroid hormones and lipid panels checked on the same day. The results were further stratified by race and sex, and the relationship between thyroid function and lipids was assessed. The correlation coefficient (r) was compared between sexes within each race for the following variables: TSH vs HDL-C, TSH vs LDL-C, TSH vs Total Cholesterol, TSH vs triglycerides, FT4 vs HDL-C, FT4 vs LDL-C, FT4 vs Total Cholesterol, and FT4 vs triglycerides. Among black males with hypothyroidism, there was a notably stronger correlation when compared to black females in the relationship between TSH vs LDL-C, and TSH vs Total Cholesterol. Specifically, the correlation coefficient of TSH vs LDL-C among Black males with hypothyroidism was 0.582, compared to 0.133 among Black females with hypothyroidism ($P = 0.0053$). Furthermore, the correlation coefficient of TSH vs Total Cholesterol among Black males was 0.567 compared to 0.184 among Black females ($P = 0.016$). In contrast, no difference in any of the relationships between thyroid and lipids was demonstrated between sexes amongst Whites, Asians, and Hispanics. Overall, we found differences in Black patients compared to patients of other races with regards to the association between thyroid and lipids. Specifically, it was found that Black males with hypothyroidism had a stronger positive correlation in TSH vs LDL-C and TSH vs Total Cholesterol than Black females. This type of difference between sexes was not found amongst any other race. These findings suggest that thyroid dysfunction is associated with changes in lipids, and the way these changes manifest may vary depending on the race and sex. This further highlights the importance of checking lipid panels in patients with thyroid dysfunction. Further research is needed to more clearly characterize the variation that is seen in thyroid and lipid function amongst races.

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Black Women Have a Worse Cardio-Metabolic Risk Profile Compared to White Women with Polycystic Ovary Syndrome in the United States: A Systematic Review and Meta-Analysis

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Health disparities may influence cardio-metabolic risk in women with polycystic ovary syndrome (PCOS). The magnitude and direction of differences in cardio-metabolic risk between Black and White women with PCOS remain uncertain due to inconsistent reports. We conducted