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Introduction: Thyroid dysfunction has a great impact on lipids as well as a number of other cardiovascular risk factors. Though the effect of thyroid hormones on plasma cholesterol concentrations are well-recognized, however, there are conflicting reports about the effect of thyroid hormone on the metabolism of plasma triglycerides. We sought to determine the effect of hypertriglyceridemia on patient admitted with hyperthyroidism. **Methods:** We queried the National Inpatient Sample (NIS) databases from 2016 to 2017 for adults aged 18 and above with hypertriglyceridemia as a principle diagnosis with and without hyperthyroidism using ICD-10 codes. Multivariate logistic and linear regression analysis was used accordingly to adjust for confounders. **Results:** There were over 71 million discharges in the combined 2016 and 2017 NIS database. Out of 17,705 hyperthyroidism hospitalizations, 15% had hypertriglyceridemia. Hypertriglyceridemia with hyperthyroidism had a similar odds of inpatient mortality (AOR 0.37, CI 0.06–1.99, P=0.246), acute kidney injury (AOR 1.03, CI 0.706–1.510, P=0.868) and cardiogenic shock (AOR 0.96, CI 0.134–6.72, P=0.963). There was a statistically significant increase in odds of acute respiratory failure (AOR 0.46, CI- 0.21- 0.99, P=0.048) in those hospitalized with hyperthyroidism and hypertriglyceridemia compared to those with hyperthyroidism alone. **Conclusion:** Hypertriglyceridemia is associated with similar outcomes in patient admitted for hyperthyroidism in terms of mortality, acute kidney injury and acute cardiogenic shock with an increased odd of acute respiratory failure. More research is needed to explain the pathophysiologic mechanism underlying the effect of hypertriglyceridemia on hyperthyroidism.

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Improvement in Quality of Life and Psychological Symptoms After Treatment for Primary Aldosteronism

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Background: Primary aldosteronism (PA) is the most common treatable cause of secondary hypertension. In addition to increased cardiovascular risk, patients also suffer from impaired quality of life (QoL) and psychological symptoms. We assessed for changes in QoL and depressive symptoms in a cohort of Asian patients with PA, after surgical and medical therapy. **Methods:** We administered questionnaires to 34 patients with PA, mean age, 51.3 years, 29.4% females, in a prospective observational study from 2017 to 2020. QoL was assessed using RAND-36 and EQ-5D-3L, and depressive symptoms was assessed using Beck Depression Inventory (BDI-II) at baseline, 6 months, and 1 year post-treatment. **Results:** Significant improvement was observed 1 year after treatment in both physical and mental summative scores of RAND-36 from baseline, +3.65 (p = 0.023) and +3.41 (p = 0.033) respectively, as well as four subscale domains (physical functioning, bodily pain, role emotional and mental health). Significant improvement was also seen in EQ-5D dimension of anxiety/depression at 1 year post-treatment. Patients treated with surgery (N=21) had significant improvement in EQ-5D index score post-treatment, and better EQ-5D outcomes compared to medical group (N=13) at 1 year post-treatment. 37.9%, 41.6% and 60.7% of patients had symptoms in the cognitive, affective and somatic domains of the BDI-II respectively. There was significant improvement in the affective domain of BDI after 1 year of treatment. **Conclusion**: Appropriate treatment with surgical and medical therapy improves QoL and psychological symptoms in patients with PA, highlighting the importance of early diagnosis and treatment of this common condition.

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In a Mouse Model of Type 2 Diabetes and Peripheral Artery Disease, Modulation of MirR29a and ADAM12 Reduced Post -Ischemic Skeletal Muscle Injury, Improved Perfusion Recovery and Skeletal Muscle Function

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Diabetes Mellitus (DM) is a major risk factor for developing peripheral arterial disease (PAD) and individuals with DM have worse PAD outcomes but the molecular mechanisms involved are poorly understood. Previously, in a hind limb ischemia (HLI) model of PAD, we identified a disintegrin and metalloproteinase gene 12 (ADAM12) as a key genetic modifier of post-ischemic perfusion recovery. Moreover, we showed that expression of ADAM12 in mouse and human tissue is regulated by miR29a. In non-diabetic mice, miR29a expression is downregulated after HLI that allows increased expression of ADAM12. However, upon HLI in high fat diet feed (HFD) mice, a model of type 2 diabetes, miR29a expression remains elevated that prevents ADAM12 increase and results in poor reperfusion recovery, increased skeletal muscle injury and decreased muscle function. Hence, we hypothesized that inhibition of miR29a or augmenting ADAM12 would improve these functional outcomes.
Mice (male, 26–28 weeks old) were randomized into 3 treatment groups and their hind limbs were treated with saline (grp1), ADAM12 cDNA (grp 2) or mir29a-inhibitor (grp3), through targeted micro-bubble delivery. Mice were

treated at -3 days and -1 pre-surgery, followed by post-surgery weekly boosting. HLI was achieved by unilateral ligation and excision of the femoral artery of the left hind limb. The right hind limb served as non-ischemic control. Gene expression analysis in the hind limbs 3 days post HLI showed decreased miR29a expression in normal chow fed B6, but elevated miR29a expression in HFD (B6 vs HFD; 0.5730±0.01 vs.1.02 ± 0.06, n=3–4, p= 0.001). Treatment with miR29a inhibitor decreased miR29a expression in HFD and increased ADAM12 expression compared to control untreated HFD mice (miR29a INH vs Control HFD: 0.70±0.06 vs 1.02±0.06, n= 4–5, p= 0.004) ADAM12 expression (miR29A INH vs Control: HFD 208.62±24.52 vs 11.75±4.94, n= 3–4 P<0.01). Although ADAM12 cDNA improved ADAM12 expression, miR29a inhibition increased ADAM12 expression to a greater extent (HFD vs ADAM12 vs miR29aINH; 11.75±4.94 vs 20.71±2.98 vs 208.62±24.52, n3-4, p=< 0.001). Accordingly, miR29a inhibition and ADAM12 augmentation decreased skeletal muscle injury assessed by the number of centralized nuclei/muscle fibre (Control vs ADAM12 vs miR29aINH: 0.252±0.043, vs 0.139±0.041 vs 0.040±0.012 n=4, p= 0.05), and improved skeletal muscle function assessed as maximum muscle contraction (Control vs ADAM12 vs miR29aINH: 0.17±0.06 vs 0.26±0.06, vs 0.54±0.08, n=6–7, p<0.01). It also improved perfusion recovery, (% ischemic to non-ischemic limb, control vs ADAM12 vs miR29aINH: 42.52±5.35, vs 58.45±4.87, vs 97.59±6.14, n= 5–10, p<0.01). Thus, our results show augmentation of ADAM12 and Inhibition of MiR29a improves outcomes in experimental PAD in diabetic mice but inhibiting miR29a is a more effective strategy.
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Insulin Resistance Is Associated With Impaired HDL Function and Atherogenic Modification of LDL in Polycystic Ovarian Syndrome

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Background and Aims: Polycystic ovarian syndrome (PCOS) is associated with increased risk of cardiovascular disease (CVD). The aim of this study was to assess the association between PCOS and markers of HDL functionality and atherogenic LDL modification. **Methods:** This is a cross-sectional study of 104 women with PCOS [median (IQR); age 29 (24–36) years, and BMI 32.9 (25.7–38.5) kg/m^2] and 103 BMI-matched healthy participants (age 34 (27–37) years, and BMI 31.1 (27.6–35.5) kg/m^2). PCOS was defined using the NIH criteria. Measurement of lipid profile and glycaemic blood parameters were undertaken. Patients with PCOS were divided into tertiles of insulin resistance assessed using the homeostatic model assessment (HOMA-IR). Cholesterol efflux capacity (CEC), and paraoxonase-1 (PON1) activity were measured as markers of HDL functionality. Oxidized LDL (OxLDL), lipoprotein-associated phospholipase A2 (LpPLA2), oxidized phoshopholipids on apolipoprotein B-100 (OxPL-apoB) and apolipoprotein(a) (OxPL-apo(a)), and glycated apoB were used as markers of atherogenic modification of LDL. **Results:** Patients with PCOS in the upper tertile of insulin resistance had impaired HDL functionality compared to the lower tertile and controls, with lower CEC [13.7 (12.4–14.6) vs 14.9 (13.6–17.0), *P*=0.003; and 14.5 (13.0–16.0) %, *P*=0.063 respectively] and PON1 activity [77.2 (48.2–129.2) vs 112.9 (54.0–175.4), *P*=0.043; and 131.6 (89.5–195.1) nmol/ml/min, *P*<0.001 respectively]. Markers of atherogenic modification of LDL were also increased in the upper tertile compared to the lower tertile and controls, with higher levels of OxLDL [91.6 (58.8–120.9) vs 67.2 (20.1–86.3), *P*=0.016; and 74.8 (47.6–89.5) ng/ml, *P*=0.013 respectively], LpPLA2 [1.66 (1.48–1.84) vs 1.48 (1.39–1.60), *P*=0.004; and 1.53 (1.37–1.70) μg/ml, *P*=0.015 respectively], small-dense LDL cholesterol (sdLDL) [24.8 (16.8–35.0) vs 15.3 (11.3–20.1), *P*<0.001; and 20.9 (14.6–29.0) mg/dl, *P*<0.001 respectively], and glycated apoB [4.02 (3.63–4.33) vs 3.51 (3.27–3.70), *P*<0.001; and 3.48 (3.20–3.96), *P*<0.001 respectively]. Both BMI and insulin resistance were associated with adverse lipoprotein modification, correlating positively with OxLDL, LpPLA2, sdLDL, and glycated apoB (Spearman's ρ=0.244–0.325 and Spearman's ρ=0.254–0.377 respectively, all *P*<0.050); and negatively with CEC (Spearman's ρ=-0.244 and
Spearman's ρ=0.254 respectively, both *P*<0.050). OxPL-apoB, OxPL-apo(a), and lipoprotein(a) did not differ between PCOS and controls. **Conclusions:** Insulin resistance is a key determinant of decreased HDL functionality and increased oxidative modification and glycation of LDL in PCOS, which is likely to contribute to the increased CVD risk.

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Interaction Between Wnt/β-catenin and ACTH Signaling Pathways and Paracrine Regulation in Aldosterone Producing Adenoma

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Primary aldosteronism (PA) is the most frequent form of secondary arterial hypertension and is caused in the majority of cases by an aldosterone producing adenoma (APA) or bilateral adrenal hyperplasia. Different somatic mutations have been identified in APA and in other aldosterone producing structures, which can be distinct within the same adrenal, suggesting multiple mechanisms underlying