a systematic review and meta-analysis to evaluate evidence on cardio-metabolic health disparities between Black and White women with PCOS in the US in response to the call for further delineation of these disparities by the International Evidence-based Guideline for the Assessment and Management of PCOS. Databases of MEDLINE, Web of Science, and Scopus were searched (January 1990 to September 2020) to identify observational studies documenting cardio-metabolic risk profile (glucoregulatory, lipid profile, anthropometric, blood pressure status) in Black and White women with PCOS. The primary outcome was fasting glucose. Further, cardiovascular events (stroke, coronary heart disease, heart failure) and mortality rate (cardiovascular death, total mortality) data were evaluated between groups. Studies on children (< 17 yrs.), pregnant or menopausal-aged (> 50 yrs.) women were excluded. Data were pooled by random-effects models and expressed as weighted mean differences and 95% confidence intervals. Eleven studies (n = 2,821; [626 Black; 2,195 White women]) evaluated cardio-metabolic risk profile, yet none reported on cardiovascular events/mortality rate. Black women had comparable fasting glucose (-0.61 [-1.69, 2.92] mg/dL; I^2 = 62.5%), yet exhibited increased fasting insulin (6.76 [4.97, 8.56] μIU/mL; I^2 = 59.0%); homeostatic model assessment of insulin resistance (HOMA-IR; 1.47 [0.86, 2.08]; I^2 = 83.2%); systolic blood pressure (SBP, 3.32 [0.34, 6.30] mmHg; I^2 = 52.0%) and decreased triglyceride (-32.56 [-54.69, -10.42] mg/dL; I^2 = 68.0%) when compared to White women with PCOS (All: P ≤ 0.03). Groups were comparable in total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and diastolic blood pressure (All: P ≥ 0.06). Paucity in the number of studies that evaluated cardiovascular events or mortality limits any conclusions about potential disparities. Overall, Black women with PCOS have a greater tendency for an adverse cardio-metabolic risk profile (increased insulin, HOMA-IR, SBP), despite lower triglyceride levels than White women. Our observations support consideration of these disparities for diagnostic, monitoring, management, and public health practices, and for future guideline recommendations. Heterogeneity among studies warrants future research to address the relative contributions of biological, environmental, socioeconomic, and healthcare factors to the observed disparities. Longitudinal research should address cardiovascular events and mortality rate in Black women with PCOS (www.crd.york.ac.uk/PROSPERO ID, CRD42020183485).

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CARF (CDKN2AIP) Regulates Hepatic Lipid Metabolism and Protects Against Development of Non-Alcoholic Fatty Liver Diseases

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Non-alcoholic fatty liver diseases (NAFLD) is the most common form of liver diseases in the USA with 30–40% of American being affected and about 12% with nonalcoholic steatohepatitis (NASH), a leading cause of end-stage liver diseases. NAFLD has been linked with insulin resistance, type2 diabetes, obesity, and cardiovascular diseases but molecular mechanisms underlying the development of NAFLD and its association with metabolic syndromes are poorly understood. In this study, we explored the role of CARF (collaborator of ARF) also known as CDKN2AIP, a novel gene of ARF-MDM2-p53 pathway in the development of NAFLD. It has been shown that, p53, beyond its tumor suppressor functions, can regulate the cellular glucose and lipid metabolism and its activation has been reported to induce hepatic steatosis in mice. However, as a regulator of p53 pathway, the role of CARF in the lipid metabolism and associated metabolic diseases has not been studied yet. Using high-fat diet (HFD) fed obesity mouse model of NAFLD we found that the expression of CARF along with Sirt1, pAMPK, and pACC was significantly decreased in the HFD induced fatty livers compared to control. Similarly, CARF expression was also down-regulated in palmitate (PA)-treated HepG2 cells, an in vitro model of steatosis. We also observed that shRNA mediated knockdown or lentiviral vector mediated overexpression of CARF induced or reduced the endogenous fat accumulation, respectively, in HepG2 cells, suggesting that CARF expression is negatively regulated in NAFLD. Additionally, we performed RNA seq analysis after CARF silencing in HepG2 cells and demonstrated that silencing of CARF altered the expression of genes regulating hepatic *de novo* lipogenesis, beta-oxidation, and lipid secretion all of which favor the accumulation of fat in the hepatocytes. Furthermore, genes associated with mitochondrial functions such as the TCA cycle and oxidative phosphorylation were also altered which could play a role in the development of NAFLD. Finally, we demonstrated that AAV mediated hepatic overexpression of CARF in HFD fed mouse model significantly reduced the fat accumulation in the liver as evident by H&E staining of liver sections and intrahepatic triglyceride level. Altogether we conclude that CARF plays a vital role in hepatic lipid metabolism and its downregulation perturbs lipid homeostasis leading to hepatic steatosis and the development of NAFLD.

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Carotid Intima Media Thickness in Graves Disease: Comparing Overt Hyperthyroid and Remission State

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Background/Objective: Hyperthyroid has been associated with increased cardiovascular event. Carotidintima media thickness (CIMT) is oftenly measured to evaluate the risk of cardiovascularevent. The aim of this study is to measure CIMT in Graves' disease and to comparebetween subjects in overt hyperthyroidism and

remission state. **Methods:** Our study was a cross-sectional study performed at the outpatient endocrinologyclinic of Dr. Cipto Mangunkusumo Hospital, a tertiary care hospital in Jakarta, Indonesia. Graves' disease subjects were recruited, of whom then grouped into overthyperthyroidism (clinical signs and symptoms of hyperthyroidism, low THS, highthyroxine levels, treatment naïve of within 3 months of treatments) and remission state(no clinical signs and symptoms of hyperthyroidism, normal THs and thyroxine levels,without any anti thyroid drugs for at least 6 months). CIMT measurements wereperformed by trained physician on both right and left artery carotid arteries using anultrasound equipped with software that automatically measured the CIMT. We alsomeasured lipid profile, fasting blood glucose, and ECG. **Results:** We recruited 49 Graves' disease subjects, of whom 32 and 17 subjects werein overt hyperthyroidism and remission state respectively. Median CIMT in overthyperthyroidism and remission state were 0,473 mm and 0,488 mm respectively, p:0,109. Among clinical and laboratory risk factors, only age which had an independentcorrelation with CIMT in Graves disease. (r: 0,371; p:<0,0001). **Discussion:** Our is the first study that measured CIMT among subjects with Graves'disease in remission and overt hyperthyroidism state, of which we observed nodifferences. This might be due to the fact that the atherosclerosis risk factors were notdistributed evenly on both group, of which subjects were older in the remission group. Ithas been reported that there are increasing CIMT along with aging (0,003-0,010 mm peryear). Furthermore, in remission state we need to take metabolic and physical changesinto consideration, such as increasing weight as much as 2,5% from prior weight alongwith increasing total cholesterol and LDL-cholesterol which both can affect CIMT levels. **Conclusions:** There are no significant differences in CIMT between overt hyperthyroid andremission state in Graves' disease. **Keywords:** carotid intima media thickness, Graves' disease, overt hyperthyroid, remission.

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Carotid Intima Media Thickness in Young Peruvians With Onset of Type 2 Diabetes Mellitus, an Early Marker of Vascular Compromise

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Objective: To evaluate the relationship between the thickness of the carotid intima media (GIMT) with clinical and laboratory parameters in young people to identify asymptomatic endothelial dysfunction and prevent future vascular complications. **Methods:** This is a cross-sectional study of 81 adolescents from 10 to 18 years of age distributed in three groups: (i) 27 with onset of type 2 diabetes (DM2), (ii) 22 were non-diabetic obese; and (iii) 32 with normal weight, non-diabetic BMI from a National Reference Hospital in Lima, Peru. Laboratory evaluation consisted of fasting glucose levels, HbA1C, lipid profile, us-CRP, and Doppler ultrasound for measurement of the common carotid artery (right and left, both?). **Results:** In total 81 participants, 27 of them with a previous diagnosis of type 2 diabetes mellitus, of which 22 obese patients without the onset of type 2 diabetes mellitus and 32 normal weight without onset of type 2 diabetes mellitus, the median for age was 20, 19.5 and 20 years respectively, with a predominance of the female sex in the three groups; the median time of illness in years for the study group was 6 years (IQR 3–8) with DM2; Regarding treatment, metformin was the main drug used (20 patients) followed by sulfonylureas (glimepiride and glyburide, 9 patients), insulin (7 patients) and DPP4 inhibitors (vildagliptin and sitagliptin, 06 patients); Differences were found between the groups of patients with DM2, obese patients without DM2, and normal weight subjects without DM2, this difference being stronger in terms of body mass index (26.29, 31.35, 23.73 kg / m2, respectively); abdominal girth (91, 97.25, 78 cm, respectively); fasting blood glucose (126, 87.5, 94 mg / dl, respectively); glycosylated hemoglobin (7.77, 4.85, 4.97%, respectively), all of these with a p: 0.001; diastolic blood pressure (74, 68, 64 mmHg, respectively); and triglycerides (112, 112.5 and 65.5 mg / dl, respectively); The median IMT ± iqr was 0.430 ± 0.08 mm in adolescents with DM2; 0.420 ± 0.03 mm, in non-diabetic obese adolescents; and 0.405 mm ± 0.02 mm, in non-diabetic adolescents with normal weight. In general, lean, non-diabetic adolescents had a lower IMT than adolescents with DM2 ($p = 0.003$) and obese adolescents ($p = 0.006$). **Conclusions:** Adolescents with DM2 had a higher median IMT compared to lean, non-diabetic adolescents that reflect the onset of early vascular damage due to DM 2.

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Effect of Chronic Kidney Disease on Outcome of Adult Patient Admitted With Hyperthyroidism: Analysis of the National Inpatient Sample 2016–2017

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Introduction: Kidney and thyroid function and dysfunction are interrelated through several mechanisms. Thyroid hormones can also have significant impact on kidney disease so it is important to consider the physiological association of thyroid dysfunction in relation to chronic kidney disease (CKD). Research shows that hyperthyroidism is usually not associated with CKD but is known to accelerate it. We sought to determine the effect of chronic kidney disease 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 hyperthyroidism as a principle diagnosis with and without hypertriglyceridemia 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