

Polycystic Ovarian Syndrome Conference

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Lipoprotein cardiovascular risk assessment and reduction in PCOS

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P^{COS} patients have increased cardiovascular risk according to a consensus statement by the Androgen Excess and Polycystic Ovary Syndrome (AE-PCOS) Society. Women with PCOS with obesity, cigarette smoking, dyslipidemia, hypertension, impaired glucose tolerance and subclinical vascular disease are at risk. Those with metabolic syndrome and or type-2 diabetes mellitus are at high risk for CVD. Lifestyle management is recommended for primary CVD prevention, targeting low-density and non-high-density lipoprotein cholesterol and adding insulin-sensitizing and other drugs if dyslipidemia or other risk factors persist. Lipid and lipoprotein abnormalities in PCOS consist of: High triglycerides and low HDL-C (ratio greater than 3:1 in insulin resistant states), Small LDL particles are prevalent; LDL-C may be high, but is often (misleadingly) low or normal. These numbers from the standard lipid panel represent serum concentrations of fats. Underlying these abnormalities lie the pathophysiology of this (and other) insulin resistant states: Hyper-production of antherogenic beta lipoproteins from the liver (delayed clearance also plays a role). HDL particles are often "dysfunctional" in IRS states like PCOS. HDL-C tells you nothing about HDL function! Correction of this abnormal "lipoprotein trafficking" is the goal to reducing CVD risk in PCOS not simply targeting the lipid abnormalities.

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Testosterone and SHBG levels in post-menarcheal teenage girls in relation to known cardiovascular risk markers: Evidence from two large European birth cohorts

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L ow and high testosterone is associated with higher CVD risk in men and postmenopausal women respectively. Several studies Linked PCOS to cardiovascular disease risk through high testosterone and insulin. We investigated the relationship between testosterone and SHBG levels and known cardiovascular risk markers in post-menarcheal teenage girls in two well characterized European birth cohorts; ALSPAC (Avon Longitudinal Study of Parents and Children) and NFBC-86 (North Finland Birth Cohort-1986). Serum testosterone was assayed using mass spectrometry at age 15-16 years. Serum sex hormone binding globulin (SHBG) was assayed using commercial kits. Total testosterone and SHBG were correlated with known cardiovascular markers; blood pressure, anthropometric measures, body fat composition (ALSPAC only), serum lipid profile, high sensitivity C-reactive protein and insulin in 1081 and 1312 girls from the ALSPAC and NFBC-86 cohorts respectively. After adjusting for age of menarche and BMI, total testosterone correlated with HDL-cholesterol and negatively with hs-CRP in the ALPSAC cohort. In the NFBC-86, total testosterone correlated with total cholesterol, HDL-cholesterol in the ALSPAC group. However it correlated negatively with insulin and hs-CRP. In the NFBC-86, SHBG correlated with total cholesterol, LDL-cholesterol in the ALSPAC group. However it correlated negatively with insulin and hs-CRP. In the NFBC-86, SHBG correlated with total cholesterol (LDL and HDL) and hs-CRP. However it negatively correlated with a favorable cardio-vascular risk markers profiles. There is confirmation from both European cohorts and evidence of population differences. The negative relationship between SHBG and insulin is evident in teenage years.

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