Women with polycystic ovary syndrome (PCOS) have equivalent lipid profiles but a lower high-molecular-weight (HMW) adiponectin levels compared with controls matched for age, BMI and insulin resistance

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PCOS is the most common endocrine disorder amongst premenopausal women, with a prevalence of 5–10% (1). Insulin resistance (IR) is present in 50–70% (2) of these women. Women with PCOS are more insulin resistant than age- and BMI-matched controls, have a higher incidence of diabetes (3) and have a more deleterious cardiovascular risk profile (4) than women without the condition. As IR makes an important contribution to CVD risk factors, the aim of the present study was to determine whether it is PCOS per se, or the IR component that is impacting most on CVD risk.

Women with PCOS (n 81) and normo-ovulatory control women (n 97) were recruited and a 2 h oral glucose tolerance test performed, with glucose, C-peptide and insulin levels measured at 0 and 2 h. Fasting total plasma cholesterol, HDL, LDL, TAG and total and HMW adiponectin and full blood count were determined. BMI and waist:hip ratio (WHR) were calculated for each volunteer. The extent of IR was assessed by HOMA. Non-normally-distributed data were log transformed. Independent and paired t tests were used to explore differences between the groups. Multiple linear regression explored the predictive relationship between variables. In order to remove the influence and to ascertain the affect of IR more exactly, further pair matching (n 56 PCOS group, n 56 controls) was performed on the basis of age, BMI and IR.

The women with PCOS were more insulin resistant (as assessed by HOMA; P=0.009), had a less-favourable atherogenic index (log(TAG/HDL-cholesterol)³; P=0.020), lower HDL levels (P=0.016) and lower total (P=0.016) and HMW adiponectin (P=0.002) concentrations. The PCOS group also had a higher leucocyte count than the control group (P=0.000). As IR plays a pivotal role in CVD risk further matching was performed. When matched for IR the differences in lipid profile disappeared; however, the PCOS group had a significantly greater WHR and a greater leucocyte count. Interestingly, when matched for IR, HMW adiponectin concentrations remained lower in the PCOS group. Multiple linear regression showed that alanine aminotransferase (ALT) (21.9%) and leucocyte count (23.9%) made the largest significant unique contribution to HMW adiponectin in the PCOS cohort (P=0.050 and P=0.033 respectively). Androgens also contributed to the overall levels of HMW adiponectin in this group, explaining 8% of the variance.

When compared with equally insulin-resistant age- and BMI-matched controls, women with PCOS have an equivalent CVD risk lipid profile; however, differences in WHR, leucocyte count and HMW adiponectin remain. Further work is required to ascertain the cause and impact of lower HMW in women with PCOS and to explore the possible link between IR, HMW adiponectin, low-grade inflammation and the liver in this condition.