Metabolic dysfunction in obese Hispanic women with polycystic ovary syndrome

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STUDY QUESTION: Are certain ethnic groups with polycystic ovary syndrome (PCOS) at increased risk of metabolic disorders?

SUMMARY ANSWER: Obese Hispanic women with PCOS are at increased risk of metabolic disorders compared with age- and BMI-matched obese non-Hispanic white women with PCOS in the USA.

WHAT IS KNOWN ALREADY: Ethnic differences in body composition and metabolic risk are well established. PCOS is a common disorder in women of reproductive age and is associated with high rates of insulin resistance, glucose intolerance and dyslipidemia.

STUDY DESIGN, SIZE, DURATION: A cross-sectional observational study was performed at an Academic Medical Center on 60 women of reproductive age with PCOS.

PARTICIPANTS/MATERIALS, SETTING, METHODS: Blood was obtained after fasting from 17 Hispanic, 22 non-Hispanic black and 21 non-Hispanic white women with PCOS who were similar in age and BMI. Anthropometric parameters, insulin, lipid and lipoprotein levels (measured by nuclear magnetic resonance) were compared between the three groups.

MAIN RESULTS AND THE ROLE OF CHANCE: Age and BMI did not differ between the groups. Hispanic women with PCOS had a greater degree of abdominal obesity, insulin resistance and dyslipidemia compared with non-Hispanic white women. The differences in HDL, HOMA-IR, VLDL size and LDL particle number persisted after adjustment for WHR while differences in LDL particle size and HDL particle size did not persist after adjustment for WHR.

LIMITATIONS, REASON FOR CAUTION: The sample size for the three groups was small but the findings were still significant. The women were mostly obese so the ethnic differences in metabolic disorders may not apply to non-obese women with PCOS.

WIDER IMPLICATIONS OF THE FINDINGS: Independent of BMI, obese, reproductive age, Hispanic women with PCOS in the USA had a greater degree of abdominal obesity, insulin resistance and dyslipidemia. Hispanic women with PCOS may benefit from more focused management of metabolic parameters.

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Key words: waist-to-hip ratio / dyslipidemia / high-density lipoprotein particle size / low-density lipoprotein particle number / nuclear magnetic resonance
**Introduction**

Dyslipidemia is one of the most frequent metabolic abnormalities in women with polycystic ovary syndrome (PCOS). Women with PCOS have been shown to have higher triglyceride and low-density lipoprotein (LDL) cholesterol and lower high-density lipoprotein (HDL) cholesterol compared with control women of similar ethnicity, age and BMI (Wild et al., 1985; Conway et al., 1992; Talbott et al., 1998; Legro et al., 2001). In addition, using the nuclear magnetic resonance (NMR) technique, our group has demonstrated that women of reproductive age with PCOS have a more atherogenic lipoprotein profile consisting of higher very-low-density lipoprotein (VLDL) and LDL particle number and significantly lower HDL size and borderline lower LDL size compared with control women of similar age and BMI (Sidhwani et al., 2011). Other investigators using different techniques have reported similar findings, although NMR is the gold standard technique for assessment of lipoprotein particle number and size (Dejager et al., 2011). Similar findings, although NMR is the gold standard technique for assessment of lipoprotein particle number and size (Dejager et al., 2011; Pirwany et al., 2001; Berneis et al., 2007). These adverse alterations are not always fully apparent on conventional lipid assay (Garvey et al., 2003) but are strongly associated with insulin resistance (Garvey et al., 2003) and cardiovascular disease (Gardner et al., 1996; Lamarche et al., 1997; Blake et al., 2002; Kuller et al., 2002). These atherogenic alterations are also likely related to increased accumulation of intra-abdominal fat (Nieves et al., 2003).

Ethnic differences in insulin sensitivity and body composition are well recognized. Greater degrees of insulin resistance and abdominal obesity have been reported among Hispanic Americans compared with other ethnicities in the USA (Haffner et al., 1986; Haffner et al., 1991; Park et al., 2003). Hispanic women have been shown to have lower insulin sensitivity (Ho et al., 2002) and higher prevalence of metabolic syndrome (St-Onge et al., 2004), type 2 diabetes (DM2) and cardiovascular disease risk factors compared with non-Hispanic white women (Winkleby et al., 1998). There is a suggestion that the prevalence of PCOS is higher among Hispanic women compared with women of other ethnicities although the prevalence in this study was determined by self-report (Goodarzi et al., 2005). Furthermore, Hispanic women with PCOS have been shown to have higher degree of insulin resistance compared with other ethnic groups (Dunaif et al., 1993) although this finding has not been universal (Welt et al., 2006). If metabolic dysregulation is more severe among Hispanic women with PCOS compared with other ethnic groups, these women will benefit from more intense monitoring of metabolic parameters.

In this study, we examined differences in body composition, insulin sensitivity and lipid and lipoprotein profile (measured by NMR) between groups of obese women with PCOS in the following racial/ethnic groups in the USA: Hispanic, non-Hispanic white and non-Hispanic black. Identification of groups of women with PCOS at higher risk for metabolic and cardiovascular disease is important since these groups may require closer metabolic monitoring.

**Materials and Methods**

**Subjects**

Sixty women of reproductive age with PCOS were recruited for the study. Of these women, 17 were Hispanic, 22 were non-Hispanic blacks and 21 were non-Hispanic whites. Women with PCOS were recruited from advertisement at the University of Illinois (IL, USA) or from endocrinology or reproductive endocrinology clinics at the University of Illinois who agreed to participate in the research. These women were recruited as part of our original study to assess differences in lipid and lipoprotein profile between PCOS and control women (Sidhwani et al., 2011). Eligible women were between 18 and 40 years of age who were free of chronic disease, including diabetes and hypertension, and reported a history of menstrual irregularity and clinical hyperandrogenism such as hirsutism, acne or androgenic alopecia. The diagnosis of PCOS was confirmed based on the National Institutes of Health (NIH) criteria and defined by the presence of oligomenorrhea (<6 menses per year) and clinical and biochemical hyperandrogenism (Zawadzki and Dunaif, 1992). Biochemical hyperandrogenism was established based on elevated total or bioavailable testosterone levels. Levels were considered to be elevated if they were above the normal range in our assay (Moran et al., 2015). Thyroid hormone abnormalities, hyperprolactinemia and non-classical congenital hyperplasia due to 21-hydroxylase deficiency were excluded by appropriate laboratory testing in all women with PCOS. All women with PCOS underwent a history and physical exam by a physician investigator that included detailed questions regarding their reproductive function and symptoms related to hyperandrogenism. All women reported oligomenorrhea as defined by <6 menstrual cycles per year since menarche. Additionally, in order to qualify for participation in the study all women had reported clinical symptoms consistent with hyperandrogenism and had elevated androgen levels. We do not report hirsutism scores, such as Ferriman Gallwey scoring system, since even though all of our patients complained of skin manifestations of hyperandrogenism, including hirsutism, cosmetic removal of hair was common among women and interfered with the accuracy of this determination. None of the women with PCOS had taken any oral contraceptive, other forms of hormonal contraception or fertility treatments in at least 3 months prior to their participation nor had they received progesterone for at least 1 month prior to their participation in the study. None of the women had ever taken any insulin-sensitizing agents or metformin.

Women were excluded from participation if they were pregnant or lactating, had any chronic disease including diabetes, hypertension, psychiatric disorder or any surgical procedure on their ovaries or uterus. None of the subjects were receiving any medication for treatment of dyslipidemia, diabetes or hypertension. Women were asked to complete standard questionnaires regarding alcohol and tobacco use and exercise habits. English was the primary language of all Hispanic participants who were mostly second or third generation of Central American background. Hispanic women were well acculturated into American lifestyle including dietary habits.

**Ethical approval**

The study was approved by the institutional review board at the University of Illinois and all subjects provided written informed consent prior to the participation in the study.

**Data collection**

All women were studied at the clinical research center at University of Illinois and underwent a history and physical exam by a physician investigator that included a detailed menstrual and medical history as well as assessment for hirsutism and other signs of hyperandrogenism and insulin resistance. Standardized forms were used to obtain medical history including information on exercise habits, alcohol and tobacco use. Height, weight and waist measurements were determined on all subjects. Blood pressure was determined as an average of three measurements following 30 min of rest at the clinical research center. A morning blood sample was obtained after an overnight fast from all subjects for measurements of total testosterone, sex hormone-binding globulin (SHBG), lipid and lipoprotein profile. A 2-h oral glucose
Laboratory methods

All laboratory evaluations with the exception of lipoprotein profile and insulin were performed at Quest Diagnostics. Total testosterone was measured by turbulent flow liquid chromatography mass spectrometry (ThermoFisher Scientific, Franklin, MA, USA; and Applied Biosystem-MDS Sciei, Foster City, CA, USA) that has an assay sensitivity of 0.034 nmol/l and no cross reactivity with 30 testosterone-related compounds. Bioavailable testosterone was calculated based on constants for the binding of testosterone to SHBG and albumin. SHBG was measured by chemiluminescent immunometric assay (Siemens Immulite 2500; Deerfield, IL, USA) and albumin was measured by spectrophotometry. Total and HDL cholesterol and triglyceride were determined by spectrophotometry. The intra- and inter-assay coefficients of variation were 1.1 and 1.8% for total cholesterol, 2.1 and 2.9% for HDL and 1.1 and 1.9% for triglyceride, respectively. The LDL cholesterol was calculated using the Friedewald equation (Friedewald et al., 1972). Plasma glucose was collected in a fluoride/oxalate tube and analyzed using spectrophotometry. The intra- and inter-assay coefficients of variation for this assay were 1.1 and 1.5%, respectively. Insulin was measured by a chemiluminescent sandwich immunoassay (Siemens Immulite 2000 from Flanders, NJ, USA) measuring to as low as 1.4 pmol/l. The inter- and intra-assay coefficients of variation for this assay were 4 and 5%, respectively. Lipoproteins were analyzed using NMR technology by LipoScience (Raleigh, NC, USA). The intra- and inter-assay coefficients of variation were 1.4 and 3.1% for VLDL particle number, 2.4 and 2.1% for LDL particle number, 1.2 and 1.5% for HDL particle number, 0.8 and 1.8% for VLDL size, 0.5 and 0.4% for LDL size and 0.5 and 0.6% for HDL size, respectively (Jeyarajah et al., 2006).

Table I: Comparison of clinical and laboratory characteristics of obese women with polycystic ovary syndrome.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Hispanic (n = 17)</th>
<th>Non-Hispanic Black (n = 22)</th>
<th>Non-Hispanic Whites (n = 21)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>27 ± 4</td>
<td>28 ± 6</td>
<td>29 ± 7</td>
<td>0.52</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>36.0 ± 7.3</td>
<td>36.8 ± 10.6</td>
<td>33.8 ± 10.9</td>
<td>0.60</td>
</tr>
<tr>
<td>WHR</td>
<td>0.84 ± 0.07</td>
<td>0.80 ± 0.07</td>
<td>0.77 ± 0.07</td>
<td>0.02</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>117 ± 12</td>
<td>118 ± 12</td>
<td>115 ± 12</td>
<td>0.8</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>71 ± 7</td>
<td>72 ± 7</td>
<td>71 ± 10</td>
<td>0.7</td>
</tr>
<tr>
<td>Testosterone (ng/dl)</td>
<td>56 ± 19</td>
<td>64 ± 30</td>
<td>62 ± 30</td>
<td>0.41</td>
</tr>
<tr>
<td>Bioavailable T (ng/dl)</td>
<td>18 ± 7</td>
<td>22 ± 15</td>
<td>15 ± 5</td>
<td>0.08</td>
</tr>
<tr>
<td>DHEAS (µg/dl)</td>
<td>191 ± 113</td>
<td>161 ± 100</td>
<td>190 ± 100</td>
<td>0.52</td>
</tr>
<tr>
<td>SHBG (nmol/l)</td>
<td>21 ± 12</td>
<td>21 ± 10</td>
<td>35 ± 24</td>
<td>0.01</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>3.6 ± 2.1</td>
<td>3.0 ± 3.4</td>
<td>1.8 ± 1.5</td>
<td>0.01; 0.05‡</td>
</tr>
<tr>
<td>G0 (mg/dl)</td>
<td>82 ± 9</td>
<td>80 ± 7</td>
<td>86 ± 4</td>
<td>0.07</td>
</tr>
<tr>
<td>I0 (µU/ml)</td>
<td>17 ± 10</td>
<td>15 ± 1</td>
<td>9 ± 7</td>
<td>0.04; 0.06‡</td>
</tr>
<tr>
<td>G2h (mg/dl)</td>
<td>135 ± 40</td>
<td>120 ± 73</td>
<td>121 ± 29</td>
<td>0.41</td>
</tr>
<tr>
<td>I2h (µU/ml)</td>
<td>163 ± 651</td>
<td>102 ± 73</td>
<td>70 ± 49</td>
<td>0.002; 0.01‡</td>
</tr>
</tbody>
</table>

Data are presented as mean ± SD and analyzed by general linear model with group comparisons by Bonferroni.

WHR, waist-to-hip ratio; SBP, systolic blood pressure; DBP, diastolic blood pressure; Bioavailable T, bioavailable testosterone; DHEAS, dehydroepiandrosterone sulfate; SHBG, sex hormone-binding globulin; HOMA-IR, homeostasis model assessment of insulin resistance; G0, fasting glucose; G2, 2-hr glucose after 75 g oral glucose load. Bioavailable T, HOMA-IR, Fasting insulin (I0) and 2-h insulin (I2h) were log-transformed prior to the analyses.

‡ P < 0.05 compared with non-Hispanic whites.

P < 0.01 compared with non-Hispanic whites.

After adjustment for WHR.

Statistical analyses

The homeostasis model assessment of insulin resistance (HOMA-IR) was calculated according to the following formula: [fasting glucose (mmol/l) × fasting insulin (µU/ml)]/22.5 (Matthews et al., 1985). Continuous variables were presented as mean and standard deviation (SD). Bioavailable testosterone, fasting and 2 h insulin, and HOMA-IR were natural log-transformed prior to all analyses because of skewed distributions. All other variables were normally distributed based on their histogram. Continuous variables were compared using general linear model for the overall comparison followed by Bonferroni correction for comparisons of differences between ethnic groups. These analyses were repeated after adjustment for waist-to-hip ratio (WHR). Categorical variables were compared using chi-square statistics. Analyses were performed using the 18.0 PC version of software for the Statistical Package for the Social Sciences (SPSS, Inc., Chicago, IL, USA). A P ≤ 0.05 was considered significant.

Results

Baseline clinical and laboratory characteristics of women with PCOS in each group are summarized in Table I. There were no differences in age or BMI among the three groups of women. Hispanic women had a higher WHR compared with non-Hispanic white women. There were no differences in blood pressure between the three groups. Very few women in each group smoked (three Hispanic, one non-Hispanic black and three non-Hispanic white) or consumed more than three alcoholic beverages per week (one Hispanic, one non-Hispanic black and three non-Hispanic white); differences that were not significant between groups (P = 0.6 and P = 0.2, respectively, data not shown). Thirty-eight percent of non-Hispanic whites, 33% of non-Hispanic...
blacks and 28% of Hispanic women reported routine exercise of at least 30 min, three times per week; differences that were not significant between groups (\( P = 0.70, \) data not shown).

Total and bioavailable testosterone and dehydroepiandrosterone sulfate (DHEAS) were not different between the three groups (Table I) but SHBG levels were significantly higher in non-Hispanic white compared with Hispanic (Table I) and non-Hispanic black women (Table I). Women were excluded from the study if they had a prior medical history of DM2. None of the subjects in the study were found to have DM2 or impaired fasting glucose based on fasting blood glucose. However, two Hispanic women and one non-Hispanic black woman had DM2 based on the 2-h glucose value and five Hispanic women, five non-Hispanic black women and four non-Hispanic white women had impaired glucose tolerance (IGT) based on 2-h glucose value. The number of women with DM2 or IGT did not differ significantly between groups. Hispanic women had higher HOMA-IR and fasting insulin levels compared with non-Hispanic white women (Table I). Similarly 2-h insulin levels were higher in Hispanic women compared with non-Hispanic white women (Table I). The differences in HOMA-IR and fasting insulin levels between Hispanic and non-Hispanic white women with PCOS became borderline significant after adjustment for WHR (Table I). However, the difference in 2-h insulin remained significant even after adjustment for WHR (Table I).

Non-Hispanic white women had significantly higher HDL cholesterol compared with Hispanic women (Fig. 1). There were no differences between groups in LDL cholesterol (\( P = 0.80 \) data not shown). There

Figure 1  Ethnic differences in lipid and lipoprotein profile among obese women with polycystic ovary syndrome. Data are presented as mean ± SE. All analyses are by general linear model with Bonferroni correction for between group differences. LDL-PN, low-density lipoprotein particle number; VLDL-S, very low-density lipoprotein size; HDL-S, high-density lipoprotein size; LDL-S, low-density lipoprotein size; HDL, high-density lipoprotein. *\( P < 0.05 \) compared with non-Hispanic white; †\( P < 0.01 \) compared with non-Hispanic white; ‡\( P = 0.06 \) compared with non-Hispanic white; §\( P < 0.05 \) after adjustment for WHR compared with non-Hispanic white.
were no differences between groups in triglyceride levels (Fig. 1). LDL particle number (LDL-PN) was highest in Hispanic (1386 ± 514 nmol/l) compared with non-Hispanic black (1146 ± 458 nmol/l) and non-Hispanic white women (936 ± 290 nmol/l) and this difference achieved statistical significance between Hispanic and non-Hispanic white women (Fig. 1). The difference in LDL-PN between Hispanic and non-Hispanic white women persisted after adjustment for WHR (Fig. 1). VLDL-size (VLDL-S) was highest in Hispanic women (52 ± 8 nm) compared with non-Hispanic black (48 ± 7 nm) and non-Hispanic white women (45 ± 5 nm) and this difference achieved statistical significance between Hispanic and non-Hispanic white women (Fig. 1). LDL-size (LDL-S) was lowest in Hispanic (20.5 ± 0.7 nm) compared with non-Hispanic black (20.9 ± 0.9 nm) and non-Hispanic white women (21.3 ± 0.8 nm) and this difference achieved statistical significance between Hispanic and non-Hispanic white women (Fig. 1). The differences in VLDL-S and LDL-S did not persist after adjustment for WHR (Fig. 1). HDL-size (HDL-S) was lowest in Hispanic (8.8 ± 0.3 nm) compared with non-Hispanic black (9.0 ± 0.4 nm) and non-Hispanic white women (9.3 ± 0.4 nm) and this difference achieved statistical significance between Hispanic and similarly obese non-Hispanic white women (Fig. 1). The difference for HDL-S between Hispanic and non-Hispanic white women remained significant even after adjustment for WHR (Table I).

Discussion

Our results demonstrate that, independent of BMI, there are ethnic differences in abdominal obesity, insulin sensitivity and lipid and lipoprotein levels among young obese women of reproductive age with PCOS in the USA. Obese Hispanic women with PCOS had the highest WHR and this difference achieved statistical significance in comparison to obese non-Hispanic white women. Consistent with this finding, obese Hispanic women with PCOS had a greater degree of insulin resistance as determined by higher HOMA-IR, fasting and 2 h insulin and lower SHBG; differences that achieved statistical significance in comparison to obese non-Hispanic white women. In addition to higher degree of insulin resistance, obese Hispanic women with PCOS had the lowest HDL cholesterol, highest LDL-PN and VLDL-S and lowest LDL-S and HDL-S; differences that became significant between Hispanic and similarly obese non-Hispanic white women. Many of these differences did not persist after adjustment for WHR suggesting that abdominal obesity predisposes to these adverse alterations in insulin sensitivity and lipid and lipoprotein parameters. However, the increase in LDL-PN and the decrease in HDL-S in Hispanic women persisted even after adjustment for abdominal obesity suggesting that additional unmeasured factors are responsible. These changes in lipid and lipoprotein profile are highly atherogenic and predispose to cardiovascular disease (Gardner et al., 1996; Lamarche et al., 1997; Garvey et al., 2003).

Lower HDL cholesterol levels have been reported in women with PCOS compared with reproductivey normal women (Wild et al., 1985; Conway et al., 1992; Talbott et al., 1998) and the finding from this study indicates that the levels are further reduced in obese Hispanic women with this condition. We have previously shown that women with PCOS have a higher LDL particle number and smaller, more dense LDL and smaller HDL particles compared with ethnicity, age and BMI-matched control women (Sidhwani et al., 2011). This study demonstrates a further significant increase in LDL particle number and a reduction in HDL and LDL particle size in Hispanic women with PCOS compared with non-Hispanic white women with PCOS of similar BMI. HDL cholesterol is atheroprotective, primarily by its role in reverse cholesterol transport that involves removal of cholesterol from macrophages in the vessel wall back to the liver (Rader, 2006). Smaller HDL particles are less effective in reverse cholesterol transport and hence are less atheroprotective (Garvey et al., 2003; El Harchaoui et al., 2009). Prospective studies of large cohorts have demonstrated that increased LDL particle number, especially of dense small particles, is a strong predictor of development of cardiovascular disease independent of LDL concentration (Gardner et al., 1996). The increase in LDL-PN and the decrease in HDL-S and in obese Hispanic women appear to be independent of abdominal obesity and place these women at increased risk for cardiovascular disorders compared with other ethnic groups.

An additional finding of this study is the higher levels of SHBG among non-Hispanic white women with PCOS compared with Hispanic and non-Hispanic black women of similar BMI. Despite differences in SHBG, bioavailable testosterone levels did not differ between the groups. SHBG is an independent predictor of DM2 among all ethnicities (Chen et al., 2012) and reduced SHBG level is a good surrogate for insulin resistance (Jayagopal et al., 2004; Ding et al., 2009). Lower SHBG levels among non-white women indicate that these women are more insulin resistant compared with non-Hispanic white women and are consistent with the results of HOMA-IR and fasting and 2-h insulin that also indicate a higher degree of insulin resistance in Hispanic women. Interestingly, among a large group of obese premenopausal women without PCOS who had participated in the Diabetes Prevention Program, SHBG levels were not different between Hispanics, non-Hispanic white or black women (Kim et al., 2013). In this study unlike ours, waist circumference did not differ between the ethnic/racial groups. Abdominal obesity consisting of increased subcutaneous and visceral depots has been shown to be a feature of PCOS, independent of overall obesity (Yildirim et al., 2003; Carmina et al., 2007; Huang et al., 2012), and our data indicate that Hispanic women with this condition are more severely affected. There are data for strong associations between waist circumference and risk for insulin resistance and DM2 in Hispanic populations (Mamtani et al., 2013). Our group and others have shown that among both diabetic as well as non-diabetic cohorts, the lipoprotein abnormalities, such as increase in VLDL and LDL particle number and reduction in LDL and HDL size, correlate best with visceral fat rather than overall adiposity (Sam et al., 2008; Fox et al., 2009).

Our study has a number of limitations. The sample size in each group was small and our findings require confirmation in larger studies. However, despite these limitations, there were significant differences in metabolic parameters between the three ethnic/racial groups of similar age and BMI. The differences in insulin resistance and lipid and lipoprotein parameters between Hispanic and non-Hispanic women with PCOS might be independent of PCOS and related to ethnic differences in these measures (Haffner et al., 1986; Haffner et al., 1991; Park et al., 2003). Hispanics have been shown to have a greater degree of insulin resistance and metabolic and cardiovascular risk factors independent of gender (Winkleby et al., 1998; Ho et al., 2002; St-Onge et al., 2004). An additional limitation is inclusion of mostly obese women thus the observed ethnic differences in metabolic parameters and lipid and lipoprotein profile may not apply to normal weight or overweight women with PCOS. We did not obtain a dietary history from our subjects and it is possible that dietary differences could account for the differences in metabolic parameters although women were similar in
terms of exercise habits and their alcohol and tobacco use. Additionally, our data in Hispanic women may only apply to the Hispanic population in the USA and likely will not be reflective of the native population throughout Latin America who are different in terms of lifestyle, dietary habits, prevalence of obesity, metabolic dysfunction and even ethnic makeup (Daviglus et al., 2012). However, several aspects of our study are novel. To our knowledge simultaneous assessment of metabolic function in women with PCOS belonging to the three main ethnic groups in the USA is lacking especially since women in our study were similar in terms of age and BMI and were diagnosed with PCOS based on NIH criteria. Previous assessment of lipoprotein profile in women has not included the gold standard technique of NMR that provides a much more accurate, detailed and simultaneous assessment of VLDL, LDL and HDL particle size and number.

In summary, data on ethnic differences in insulin sensitivity and metabolic disorders in PCOS are sparse (Dunaif et al., 1993; Kauffman et al., 2002) and contradictory (Welt et al., 2006). Our study is novel since we were able to simultaneously study women with PCOS from three ethnic/racial groups in the USA and compare their metabolic and cardiovascular risk factors. Our findings on lipoprotein parameters are also novel since we utilized the gold standard technique of NMR to obtain information on lipoprotein particle number and size, which are superior predictors of atherosclerosis in comparison to measures obtained from conventional lipid assays (Gardner et al., 1996; Lamarche et al., 1997). Our results indicate that obese reproductive age Hispanic women with PCOS have higher degrees of abdominal obesity, insulin resistance and lipid and lipoprotein abnormalities compared with non-Hispanic white women with this condition in the USA. These findings require confirmation in larger studies but indicate that obese reproductive age Hispanic women with PCOS in the USA are at high risk for metabolic complications and may benefit from more focused monitoring of metabolic parameters.

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Authors’ roles

All authors provided substantial contributions to conception and design, acquisition and/or analysis and interpretation of data, drafting and revising of the manuscript and provided final approval of the version to be published.

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Conflict of interest

None declared.

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