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Influence of Race/Ethnicity on Cardiovascular Risk Factors in Polycystic Ovary Syndrome, the Dallas Heart Study

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Abstract

Objective—Polycystic ovarian syndrome (PCOS) is estimated to affect up to 20% of women. PCOS is associated with insulin resistance and cardiovascular (CV) risk factors. We aimed to evaluate the impact of race/ethnicity on the prevalence of CV risk factors and subclinical predictors of CV events.

Design—Cross-sectional analysis of data collected by the Dallas Heart Study, an urban, population-based cohort oversampled for blacks.

Patients—A previously described cohort of women with PCOS and control subjects of the same racial/ethnic group, matched for age and body mass index.

Measurements—Hormonal and clinical measures associated with PCOS and CV risk factors.

Results—The study included 117 women with PCOS and 204 controls. Women with PCOS had significant differences across racial/ethnic groups in the prevalence of hypertension, hypercholesterolemia, hypertriglyceridemia, and impaired fasting glucose ($P<.05$). Controls showed significant racial/ethnic differences in the prevalence of hypertension and impaired fasting glucose ($P<.05$). The odds of hypertension were significantly greater among women with PCOS than controls after adjusting for race/ethnicity (odds ratio, 1.50 [95% CI, 1.03–2.30]; $P=.04$). However, we did not see an interaction of race/ethnicity that significantly changed CV risk factor prevalence between PCOS and controls. In addition, subclinical measures of CV disease were not different between women with PCOS vs controls, even among hypertensive women.

Conclusions—Race/ethnicity affects the prevalence of CV risk factors for women with and without PCOS. However, race/ethnicity does not interact with PCOS to additionally increase CV risk factor prevalence or subclinical CV disease.

Keywords

androgen excess disorder; chronic anovulation; hyperandrogenism; insulin resistance

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Introduction

Polycystic ovarian syndrome (PCOS) is a disorder estimated to affect at least 6% to 8% of women worldwide using the criteria of hyperandrogenism and chronic anovulation or up to 19% to 20% with current consensus criteria that includes polycystic ovarian morphology (1–4). PCOS is associated with insulin resistance and a higher prevalence of cardiovascular (CV) risk factors, including hypertension, dyslipidemia, and obesity (5,6). Despite the higher prevalence of CV risk factors and subclinical markers of CV disease in women with PCOS, studies have failed to show a consistent association between PCOS and increased CV events or mortality (6). Thus, a better understanding is needed regarding the relationship between CV risk factors and CV disease in PCOS. The role of race/ethnicity could be one such factor that attenuates or increases the impact of CV risk factors.

Although the prevalence of PCOS and its defining features may be similar across countries, the prevalence of CV risk factors in PCOS varies among different racial/ethnic groups. In the United States, fasting insulin or insulin resistance was greater in black women than in white women with PCOS in 1 study (7) but not in 2 others (8,9). Hispanics had higher insulin concentrations and decreased insulin sensitivity in 2 separate studies that compared Mexican-Americans or Caribbean-Hispanic women with PCOS to whites in the United States (10,11) and in a third study comparing Hispanics with Italian and Japanese women with PCOS (12). Many studies of racial/ethnic differences lack comparisons with matched controls without PCOS or are limited by small control groups (8–30 participants) (11,12).

Because of known racial/ethnic differences in CV risk factors, uncertainty exists about whether observed differences in CV risk factors for women with PCOS are largely attributable to race/ethnicity itself (13,14) or whether an interaction between PCOS and race/ethnicity accelerates or increases the development of CV risk factors. An example of known interactions between race/ethnicity and risk factor status is the significantly accelerated risk of conversion to type 2 diabetes mellitus after prior gestational diabetes in Hispanic women compared with non-Hispanic women. Whereas Hispanic women with impaired glucose tolerance have rates of conversion to diabetes mellitus of approximately 2% to 5% per year, Hispanic women with prior gestational diabetes and postpartum impaired glucose tolerance may have a conversion rate of 16% per year (and 80% by 5 years) (15). To date, only 1 epidemiologic cohort was large enough to stratify by race/ethnicity (16), and no significant differences across race/ethnicity were observed between PCOS and controls regarding the prevalence of CV risk factors. However, that study was limited by its reliance on clinical database codes to diagnose PCOS, which might have excluded a large proportion of the sample with PCOS (age-stratified PCOS prevalence rates ranged from 0.8%–2.7%).

Here, we evaluated the impact of race/ethnicity on the prevalence of CV risk factors in a previously described population-based cohort of more than 300 women with PCOS and matched controls from the Dallas Heart Study (DHS) (4), which was specifically designed to study the impact of race/ethnicity on CV health in a population-based cohort oversampled for blacks. To determine whether race/ethnicity was an effect modifier in PCOS, we sought to identify interactions between race/ethnicity, PCOS, and its associated CV risk factors. As a secondary aim, we evaluated prospectively collected, detailed measurements of subclinical

CV disease to determine whether increases in risk factor prevalence were associated with increases in subclinical predictors of CV events.

Methods

The study protocol was approved by the University of Texas Southwestern Medical Center Institutional Review Board, and all participants provided written informed consent to enroll in the study.

Study Sample

The DHS was designed to develop population estimates of biologic and social variables underlying ethnic differences in CV health and explore mechanisms through detailed phenotyping. Recruited participants were a probability sample of Dallas County adults, oversampled for blacks (n=6,101), as previously described (17). Participants were enrolled and completed studies from July 2000 through January 2002.

Data Collection

Participants completed a structured survey with trained interviewers and provided blood samples (17). A nested cohort in the DHS consisted of premenopausal women 35 to 49 years old, who provided additional information about their reproductive health (4). Race/ethnicity was self-identified as non-Hispanic black, non-Hispanic white, Hispanic, or other, in accordance with the categories used in the Third National Health and Nutrition Examination Survey (18). Oral estrogen and statin use were ascertained by questionnaire and by review of reported medications at the time of the structured survey.

Measurements

Details for the immunoassay of total testosterone, sex hormone–binding globulin, and insulin and for measurements of glucose and of total and lipoprotein cholesterol concentrations were previously reported (19). Insulin sensitivity was estimated by the homeostasis model assessment (HOMA-IR; HOMA Calculator version 2.2) (20). Calculated free testosterone was derived using equations previously described (19). Magnetic resonance imaging (MRI) was performed to assess ovarian size and morphology (4) and to obtain measurements of aortic wall thickness, left ventricular (LV) mass, and aortic plaque (4,21). Electron-beam computed tomographic scans were performed to measure coronary artery calcium (4). Measurements of lean mass, fat mass, and percent body fat were derived from dual-energy x-ray absorptiometry (22).

Variable Definitions

As previously described (4), PCOS was defined by Rotterdam consensus criteria using a combination of survey information (eg, cycle length, distribution of hair growth), elevated total testosterone, and measurements of ovarian morphology (23). Specific survey variables included 1) length of menstrual cycle >45 days from ages 20 to 30 years when not on birth control pills, pregnant, or breastfeeding and 2) hyperandrogenism, defined as treatment for “unwanted or excessive hair growth on your face, back, chest, arms or thighs”; these criteria were validated previously and used in another population-based cohort study (24,25). The

control group was randomly selected from women in the DHS without PCOS, matched for race/ethnicity, age (±1 year), and body mass index (BMI) (±1 kg/m^2) for each woman with PCOS. Hypertension was defined as an average systolic blood pressure ≥140 mm Hg, diastolic blood pressure ≥90 mm Hg, or the use of antihypertensive medication (26). Diabetes mellitus was defined as a fasting serum glucose level ≥7 mmol/L (126 mg/dL), nonfasting serum glucose ≥11.1 mmol/L (200 mg/dL), or the use of any glucose-lowering medication. Impaired fasting glucose was defined as a glucose level ≥5.55 mmol/L (100 mg/dL) in individuals without diabetes mellitus. Hypercholesterolemia was defined as fasting calculated low-density lipoprotein (LDL) ≥4.14 mmol/L (160 mg/dL), nonfasting direct LDL ≥4.14 mmol/L (160 mg/dL), total cholesterol ≥6.22 mmol/L (240 mg/dL) when a direct LDL was not available, or the use of statins. Hypertriglyceridemia was defined as triglycerides ≥1.70 mmol/L (150 mg/dL), and low high-density lipoprotein (HDL) was defined as <1.30 mmol/L (50 mg/dL). Metabolic syndrome was defined according to the National Cholesterol Education Program Adult Treatment Panel (at least 3 of 5 criteria): 1) low HDL cholesterol, 2) hypertriglyceridemia, 3) impaired fasting glucose, 4) hypertension or systolic blood pressure >130 mm Hg or diastolic blood pressure >85 mm Hg, and 5) waist circumference >88 cm (27). Oral estrogen use was defined as use of oral contraceptives or hormone replacement therapy; status was determined by survey response and by review of medications.

Statistical Analyses

Statistical analyses were performed using SAS version 9.2 (SAS Institute Inc). To determine if there was a significant difference among continuous variables by racial/ethnic group within a diagnostic category of PCOS or controls, the Kruskal-Wallis test was used to compare the medians based on ranks. The Bonferroni-Dunn test for multiple comparisons was performed to identify significant pairwise comparisons within a group of PCOS or controls. The Cochran Mantel Haentzel χ^2 test was used to identify racial/ethnic differences in the prevalence of risk factors within the group of PCOS or controls and to determine if the odds ratios (ORs) for the prevalence of risk factors between PCOS and controls was >1 after controlling for race/ethnicity. The Breslow-Day test of homogeneity was used to identify significant differences in the OR for CV risk factors between PCOS and controls. The Fisher exact test was used to calculate differences between PCOS and controls in prevalence of risk factors. To adjust insulin levels by measurements of body size, log-transformed insulin levels were modeled separately by BMI, lean mass, and percent body fat using a generalized linear model with comparison of the least-squares means. *P* values <.05 were considered statistically significant. Comparisons of aortic wall thickness and LV mass were restricted to blacks and whites because of the small sample size of available data for Hispanics; analysis was performed using the Wilcoxon rank sum test.

Results

The study cohort included 117 women with PCOS and 204 age- and BMI-matched controls. Participant characteristics, stratified by race/ethnicity, are shown in Table 1.

Racial and Ethnic Differences Among Women With PCOS

For the women with PCOS, we observed significant differences across racial/ethnic groups in BMI, systolic blood pressure, total cholesterol, and fasting insulin. Post-hoc analysis showed that black women with PCOS had significantly higher BMI, systolic blood pressure, and fasting insulin and lower total cholesterol than white women with PCOS. After adjusting for BMI, lean mass, or percent body fat, these racial/ethnic differences in log-transformed insulin persisted (Table 1). Also, a higher proportion of Hispanic women had impaired fasting glucose compared with black women. The prevalence of hypertension, hypercholesterolemia, hypertriglyceridemia, and impaired fasting glucose among the women with PCOS showed significant differences across the same ethnic groups.

Racial and Ethnic Differences Among Control Women

To dissect the contributions of PCOS itself and ethnicity, we performed a similar analysis with the control groups. We observed significant differences across racial/ethnic groups in BMI, waist:hip ratio, total testosterone, systolic and diastolic blood pressure, triglycerides, fasting insulin, and HOMA-IR. Post-hoc analysis showed that black women in the control group had significantly higher BMI, waist:hip ratio, systolic and diastolic blood pressure, and fasting insulin and significantly lower triglycerides than white women. Hispanic women had significantly lower total testosterone and higher fasting insulin and HOMA-IR than white women. After adjusting for BMI, lean mass, or percent body fat, these racial/ethnic differences in log-transformed insulin persisted (Table 1). Black women had significantly higher systolic and diastolic blood pressure and lower triglycerides than Hispanic women. This finding corresponded with significant differences across racial/ethnic groups in the prevalence of only hypertension and impaired fasting glucose in the control group.

Racial and Ethnic Differences in OR for CV Risk Factors and Measurements of Subclinical CV Diseases

The ORs for CV risk factors are shown in Table 2. The OR for hypertension was significantly greater among women with PCOS than controls after adjusting for race/ethnicity (OR, 1.50 [95% CI, 1.03–2.30]; P=.04). ORs were significantly different by race/ethnicity for hypertriglyceridemia, which precluded calculation of a group OR for women with PCOS vs controls. Analyzed separately by race/ethnicity, only white women with PCOS had significantly increased odds of hypertriglyceridemia and hypertension compared with controls (OR, 2.81 [95% CI, 1.13–7.00]; P=.02; OR, 5.06 [95% CI, 1.09–23.6]; P=.02, respectively).

To determine whether the greater prevalence of hypertension in PCOS could be associated with increases in subclinical measures of CV disease, we compared measurements of LV mass and aortic wall thickness in PCOS vs controls. No differences were noted between women with PCOS vs controls in any of these measures (Table 3). In the subgroup of black women with hypertension, we observed no difference in LV mass/fat-free mass when comparing women with PCOS (n=23) vs controls (n=37); median values were 2.86 g/kg (interquartile range [IQR], 2.82–3.14 g/kg) and 3.09 g/kg (IQR, 2.78–3.55 g/kg), respectively (P=.29). Likewise, aortic wall thickness was not different in this subgroup of black women with hypertension when comparing PCOS (n=18) vs controls (n=28); median

values were 1.56 mm (IQR, 1.43–1.72 mm) and 1.59 mm (IQR, 1.49–1.80 mm), respectively (*P*=.45). Analysis of women with hypertension was restricted to only black women because of an insufficient number of white and Hispanic subjects with hypertension. Evaluating other measures of atherosclerosis, we observed no difference in the presence of coronary artery calcium or of aortic plaque between PCOS and controls in blacks or whites (Table 3).

Evaluation of Confounding Medications

Finally, we reviewed oral estrogen and statin use to determine whether these treatments could explain the differences in prevalence of hypertension and hypertriglyceridemia. No difference in the use of oral estrogen was noted between PCOS and controls among blacks (1.6% vs 5.3%; *P*=.42), whites (3.3% vs 13.5%; *P*=.25), or Hispanics (4.6% vs 2.6%; *P*>.99). The Cochran Mantel Haentzel χ^2 and Breslow-Day tests showed no significant differences (*P*=.10 and *P*=.42, respectively), nor was there any difference in oral estrogen use across racial/ethnic groups when analyzing women with PCOS (*P*=.09) separately from controls (*P*=.74). No difference in the use of statins was noted between PCOS and controls among blacks (1.6% vs 1.8%; *P*=.45) or whites (6.7% vs 3.9%; *P*=.33); statins were not used by Hispanic women in this study. Again, the Cochran Mantel Haentzel χ^2 and Breslow-Day tests showed no significant differences (*P*=.71 and *P*=.67, respectively), nor was there any difference in statin use across racial/ethnic groups when analyzing women with PCOS (*P*=.25) separately from controls (*P*=.31).

Discussion

To determine whether PCOS had an additive or synergistic effect on CV risk factors that varied by race/ethnicity, we examined CV risk factors and subclinical CV disease in a population-based cohort of women with PCOS and compared them with age- and BMI-matched control subjects of the same racial/ethnic group. We observed previously known differences between blacks and whites with PCOS regarding higher blood pressure, BMI, and fasting insulin among blacks. We also observed known differences across racial/ethnic groups for all women in the prevalence of hypertension, impaired fasting glucose, and triglycerides. Importantly, we did not observe an interaction of race/ethnicity that significantly changed CV risk factor prevalence between PCOS and controls. The CV risk factor associated with PCOS across all racial/ethnic groups was hypertension; however, no difference was noted in the subsequent development of subclinical CV disease, either by race/ethnicity or between PCOS and controls.

Our results were consistent with the other reports of black women with PCOS having higher fasting insulin (7) and a higher prevalence of hypertension (16). We did not observe an increased prevalence of impaired fasting glucose in women with PCOS compared with controls, unlike prior studies (28,29), but this difference might be attributable to the high BMI of our sample and higher prevalence of impaired fasting glucose in our controls. We also did not observe higher fasting insulin, glucose, or HOMA-IR among Hispanic women with PCOS compared with black or white women with PCOS as in other studies (10–12), which might derive from the smaller sample of Hispanic women. In contrast, we did observe

the expected higher fasting insulin and HOMA-IR among Hispanic women without PCOS compared with black or white women without PCOS. Other studies have also found that racial/ethnic differences in insulin sensitivity were not significant after adjusting for BMI (9) or obesity (30). Among control subjects, racial/ethnic differences observed in CV risk factors were similar to prior studies that reported greater BMI and prevalence of hypertension in blacks (31,32), higher insulin and insulin resistance in blacks and Hispanics (33), and increased triglycerides in Hispanics (34).

In this analysis of a larger population-based sample, we specifically sought to determine whether the OR for a CV risk factor in PCOS vs controls changed across racial/ethnic groups and whether prevalence of these risk factors were significantly increased or modified by race/ethnicity with PCOS. The only significant change in the OR for a CV risk factor among race/ethnicities was observed in the prevalence of hypertriglyceridemia, with a greater OR among whites with PCOS vs controls. Adjusted for race/ethnicity, the OR for hypertension was significantly increased in PCOS vs controls across all racial/ethnic groups. If anything, whites with PCOS appeared to have a greater OR for hypertension compared with white controls. Even though these data suggest that PCOS increases the prevalence of hypertension in PCOS compared with controls, we did not see an increase in subclinical CV disease at an earlier age. Although race/ethnicity increases the prevalence of CV risk factors among nonwhite women with and without PCOS, it does not interact with PCOS to increase CV risk factor prevalence or subclinical CV disease beyond known racial/ethnic differences. However, these findings do not obviate screening for hypertension and other CV risk factors in women with PCOS across all races/ethnicities.

The greatest strength of this study is the large, multiethnic, population-based sample designed to identify racial/ethnic differences through detailed measurements of CV risk factors and subclinical measures of CV disease. Matching control subjects by both age and BMI and recruiting from the same population sample served to limit the influence of major confounders. Other studies have compared women with PCOS of different races/ethnicities but recruited subjects living in different countries (8,12,16,30). Although we used the Rotterdam criteria to diagnose PCOS, the majority of women (86%) demonstrated hyperandrogenism (4), which is associated with a greater prevalence of CV risk factors (35). In the current study, the sample was not large enough to evaluate whether using the PCOS diagnostic criteria of only hyperandrogenism and oligomenorrhea would have changed our analysis of racial/ethnic differences. We have previously reported that restricting case selection using these 2 criteria did not change the analysis of coronary artery calcium and aortic plaque between PCOS and controls (4).

A novel aspect of this study is the measurement of aortic plaque and aortic wall thickness by MRI. To our knowledge, such measures have not been previously published in evaluating the effect of race/ethnicity in PCOS. Increasing mean arterial wall thickness predicts a greater risk of fatal and nonfatal cardiovascular events in the DHS cohort (36). Additionally, aortic plaque prevalence by MRI in asymptomatic individuals from the Framingham Heart Study was significantly correlated with the Framingham Coronary Risk Score (37). Although long-term outcome data are lacking for women with PCOS that correlates premenopausal MRI measurement of aortic plaque and aortic wall thickness with postmenopausal outcomes, the

absence of ethnic/racial differences in these MRI measurements in our PCOS cohort further decreases the likelihood that racial/ethnic differences in CV risk factors increases the development of CV disease in PCOS.

Although most risk factors were unchanged across subgroups and did not vary by PCOS status, smaller differences in risk factors might have been obscured by the obesity of the entire cohort (9,30) and by the sample size. The smaller number of Hispanic women might have limited the ability to detect differences previously reported. To our knowledge, this is the largest, prospectively characterized, multi-ethnic cohort that could evaluate these comparisons in a single sample. Furthermore, the influence of ethnicity on CV risk factors and disease might increase after menopause. Although PCOS is associated with a higher prevalence of CV risk factors, increased risk of CV events or CV-related death has not been observed in younger or middle-aged patients (38,39). This analysis also was limited by the cross-sectional design, and our measures of subclinical CV disease might lack the sensitivity to detect small differences in atherosclerotic burden. Prospectively following a larger multiethnic population-based sample would best evaluate whether race/ethnicity, specific features of PCOS, or genetics or other biomarkers predict accelerated risk of diabetes, atherosclerosis, and CV disease over time.

In conclusion, despite observing known differences across racial/ethnic groups for CV risk factors within the groups of women with PCOS and within the groups of controls without PCOS, race/ethnicity was not an effect modifier further increasing the prevalence of CV risk factors in black or Hispanic women with PCOS. PCOS significantly increased the risk for hypertension across all racial/ethnic groups; however, no difference was noted in the subsequent development of subclinical CV disease. The greater prevalence of other CV risk factors in black and Hispanic women with PCOS was similar to women without PCOS in those ethnic groups. Nevertheless, race/ethnicity should be considered in the management of PCOS, with more attention to screening and treatment of hypertension in blacks and whites and impaired fasting glucose in Hispanics.

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Abbreviations

BMI	body mass index
CV	cardiovascular
DHS	Dallas Heart Study
HDL	high-density lipoprotein
HOMA-IR	homeostasis model of insulin resistance

IQR	interquartile range
LDL	low-density lipoprotein
LV	left ventricular
MRI	magnetic resonance imaging
OR	odds ratio
PCOS	polycystic ovarian syndrome

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Table 1

Characteristics of Women With PCOS and Controls Matched for Ethnicity, Age, and Body Mass Index

	Black		White		Hispanic		*P* Value[c]	
Characteristic[a,b]	**PCOS (n=62)**	**Control (n=113)**	**PCOS (n=32)**	**Control (n= 54)**	**PCOS (n=23)**	**Control (n= 37)**	**PCOS**	**Control**
Age, y	41 (37–42)	40 (37–43)	41 (37–43)	40 (38–42)	39 (37–41)	39 (36–41)	.32	.12
BMI, kg/m^2	32.3 (27.6–38.0)[d]	32.3 (29.1–37.7)[e]	28.2 (23.4–32.6)[d]	27.5 (23.4–31.5)[e]	31.7 (25.9–34.0)	30.3 (26.2–33.3)	.02	<.001
Waist:hip ratio	0.87 (0.81–0.90)	0.87 (0.82–0.90)[e]	0.83 (0.80–0.89)	0.83 (0.77–0.89)[e]	0.86 (0.83–0.89)	0.85 (0.81–0.90)	.47	.006
History of tobacco use, No. (%)	21 (33.9)	49 (43.4)	18 (56.3)	25 (46.3)	8 (34.8)	8 (21.6)	.09	.02
Total testosterone, ng/mL	0.8 (0.7–1.0)	0.6 (0.5–0.7)	0.8 (0.6–1.0)	0.6 (0.5–0.8)[f]	0.8 (0.6–0.9)	0.5 (0.4–0.7)[f]	.53	.046
Calculated free testosterone, pmol/L	20.2 (16.3–30.2)	12.8 (8.2–21.4)	23.7 (10.4–34.6)	11.5 (7.0–20.0)	20.9 (11.4–32.7)	13.2 (6.9–19.6)	.81	.70
Sex hormone-binding globulin, nmol/L	117 (84–162)	154 (91–201)	101 (76–171)	167 (101–236)	108 (81–171)	107 (87–184)	.81	.19
Blood pressure								
Systolic blood pressure, mm Hg	127 (116–138)[d]	123 (113–133)[e,g]	117 (109–130)[d]	118 (110–127)[e]	117 (108–131)	114 (107–122)[g]	.009	.001
Diastolic blood pressure, mm Hg	81 (75–86)	80 (74–87)[e,g]	78 (73–82)	76 (73–81)[e]	73 (70–86)	74 (70–80)[g]	.15	.001
Hypertension prevalence, No. (%)	23 (37.1)	31 (27.4)	6 (18.8)	2 (3.7)	3 (13.0)	4 (10.8)	.03	<.001
Cholesterol								
Total cholesterol, mg/dL	163 (145–188)[d]	161 (142–187)	190 (149–211)[d]	175 (154–190)	171 (159–217)	166 (148–180)	.04	.22
Low-density lipoprotein cholesterol, mg/dL	97 (78–114)	93 (72–120)	107 (83–135)	105 (85–117)	112 (86–135)	95 (83–105)	.09	.33
High-density lipoprotein cholesterol, mg/dL	51 (45–59)	48 (43–58)	51 (41–60)	53 (42–62)	51 (39–55)	49 (42–54)	.59	.35
Hypercholesterolemia prevalence, No. (%)	1 (1.6)	9 (8.0)	4 (12.5)	5 (9.3)	3 (13.0)	1 (2.7)	.045	.40
Triglycerides								
Triglycerides, mg/dL	75 (57–95)	72 (58–102)[e,g]	79 (54–179)	84 (57–116)[e]	97 (74–127)	118 (75–157)[g]	.13	.001
Hypertriglyceridemia prevalence, No. (%)	3 (4.8)	9 (7.9)	10 (31.3)	5 (9.3)	2 (8.7)	9 (23.1)	.002	.05
Glucose metabolism								
Fasting glucose, mg/dL	92 (82–101)	88 (82–96)	89 (80–96)	91 (83–101)	99 (88–110)	93 (87–102)	.06	.08
Fasting insulin, μU/mL	15.6 (9.4–22.0)[d]	14.6 (9.1–22.4)[e]	10.6 (5.8–18.1)[d]	9.7 (5.6–12.1)[e–f]	15.5 (10.7–21.5)	16.4 (8.4–21.7)[f]	.04	<.001
Log insulin, BMI-adjusted[h]	2.67 (2.52–2.82)	2.52 (2.41–2.62)	2.42 (2.20–2.63)	2.39 (2.24–2.55)	2.71 (2.46–2.95)	2.75 (2.57–2.92)	.002[i]	<.001[i]
Log insulin, % body fat-adjusted[h]	2.74 (2.57–2.90)	2.59 (2.48–2.70)	2.33 (2.10–2.56)	2.29 (2.13–2.45)	2.64 (2.37–2.91)	2.66 (2.48–2.85)	.004[i]	<.001[i]
Log insulin, lean mass adjusted[h]	2.64 (2.47–2.80)	2.49 (2.38–2.61)	2.35 (2.13–2.58)	2.31 (2.15–2.47)	2.87 (2.60–3.14)	2.91 (2.72–3.11)	.003[i]	<.001[i]

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Characteristic[a,b]	Black		White		Hispanic		*P* Value[c]	
	PCOS (n=62)	Control (n=113)	PCOS (n=32)	Control (n= 54)	PCOS (n=23)	Control (n= 37)	PCOS	Control
Homeostasis model of insulin resistance	3.3 (2.1–5.0)[j]	3.1 (1.8–5.0)	2.6 (1.1–4.1)	2.1 (1.2–3.7)[f]	3.8 (2.9–5.4)[j]	3.9 (1.7–5.0)[f]	.08	.005
Impaired fasting glucose prevalence, No. (%)	4/57 (7.0)[j]	5/103 (4.9)	0 (0)	0 (0)	4/21 (19.1)[j]	2/34 (5.9)	.03	.02
Type 2 diabetes mellitus prevalence, No. (%)	5 (8.1)	10 (8.9)	4 (12.5)	6 (11.1)	2 (8.7)	3 (8.1)	.79	.86
Metabolic syndrome prevalence, No. (%)	23 (37.1)	41 (36.0)	11 (34.4)	12 (22.2)	9 (39.1)	13 (33.3)	.94	.19

Abbreviations: BMI, body mass index; PCOS, polycystic ovarian syndrome.

[a] Results are presented as median (interquartile range) unless otherwise indicated.

[b] The conversion factors from conventional or metric units to Système International units are as follows: low-density lipoprotein, high-density lipoprotein, and total cholesterol, multiply by 0.0259 (mmol/L); triglycerides, multiply by 0.0113 (mmol/L,); glucose, multiply by 0.0555 (mmol/L); insulin, multiply by 6.945 (pmol/L); total testosterone, multiply by 0.0347 (nmol/L).

[c] *P* values for the Kruskal-Wallis test comparing medians among ethnic groups within a diagnostic category (PCOS or control) or the likelihood ratio χ^2 from the Cochran Mantel Haentzel χ^2 test comparing ethnic differences in the prevalence of risk factors within a diagnostic category (PCOS or control).

[d] $P<.05$ when comparing blacks and whites with PCOS.

[e] $P<.05$ when comparing black and white controls.

[f] $P<.05$ when comparing white and Hispanic controls.

[g] $P<.05$ when comparing black and Hispanic controls.

[h] Results presented as least-squares mean (95% CI).

[i] Generalized linear model, significance of ethnicity adjusted for body size variable.

[j] $P<.05$ when comparing blacks and Hispanics with PCOS.

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Table 2

Influence of Ethnicity on ORs for Cardiovascular Risk Factors Between PCOS and Controls

	Hypertension		Impaired Fasting Glucose		Type 2 Diabetes Mellitus		Hypercholesterolemia		Hypertriglyceridemia		Metabolic Syndrome	
Racial/ Ethnic Group	**OR (95% CI)**	***P* Value**	**OR (95% CI)**	***P* Value**	**OR (95% CI)**	***P* Value**	**OR (95% CI)**	***P* Value**	**OR (95% CI)**	***P* Value**	**OR (95% CI)**	***P* Value**
All women	1.50 (1.03–2.30)	.04	1.98 (0.74–5.28)	.16	1.01 (0.50–2.04)	.98	0.92 (0.40–2.14)	.85	…[a]	…	1.23 (0.89–1.68)	.21
Black	1.35 (0.87–2.10)	.19	1.45 (0.40–5.17)	.57	0.91 (0.33–2.55)	.86	0.20 (0.03–1.56)	.08	0.78 (0.21–2.91)	.71	0.95 (0.75–1.20)	.65
White	5.06 (1.09–23.6)	.02	…[b]	…	1.13 (0.34–3.69)	.85	1.35 (0.39–4.66)	.64	2.81 (1.13–7.00)	.02	0.84 (0.63–1.13)	.22
Hispanic	1.21 (0.30–4.91)	.79	3.24 (0.65–16.16)	.13	1.07 (0.19–5.94)	.94	4.83 (0.53–43.67)	.11	0.32 (0.08–1.33)	.08	0.88 (0.59–1.30)	.50

Abbreviations: PCOS, polycystic ovarian syndrome; OR, odds ratio.

[a] Breslow-Day test for difference in ORs among racial/ethnic groups was significant (P=.02). ORs must be interpreted separately by racial/ethnic group.

[b] Could not be calculated (no cases of impaired fasting glucose among whites in PCOS or control groups).

Table 3

Subclinical Measures of Cardiovascular Disease Among Women With PCOS and Matched Controls

	Black			White		
Characteristic	**PCOS**	**Control**	***P* Value**	**PCOS**	**Control**	***P* Value**
Left ventricular mass/fat free mass, median (IQR), g/kg[b,c]	2.88 (2.68–3.20)	2.85 (2.56–3.27)	.44[a]	2.77 (2.37–2.95)	2.86 (2.50–3.04)	.40[a]
Aortic wall thickness, median (IQR), mm[d]	1.61 (1.46–1.69)	1.56 (1.44–1.72)	.58[a]	1.69 (1.51–1.74)	1.50 (1.38–1.71)	.07[a]
Coronary artery calcium present, No. (%)	4/58 (6.9)	8/108 (7.4)	.25[e]	0/32 (0)	1/50 (2.0)	.61[e]
Aortic plaque present, No. (%)	14/53 (26.4)	30/89 (33.7)	.36[f]	6/28 (21.4)	19/51 (37.3)	.15[f]

Abbreviations: IQR, interquartile range; PCOS, polycystic ovarian syndrome.

[a] Wilcoxon 2-sample test.

[b] Adjusted for fat-free mass.

[c] Blacks with PCOS, n=59; black controls, n=102; whites with PCOS, n=29; white controls, n=53.

[d] Blacks with PCOS, n=53; black controls, n=88; whites with PCOS, n=28; white controls, n=51.

[e] Fisher exact test.

[f] $\chi 2$ test.