GYNECOLOGY

Racial and ethnic differences in the polycystic ovary syndrome metabolic phenotype

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BACKGROUND: Women with polycystic ovarian syndrome have a high prevalence of metabolic syndrome and type 2 diabetes mellitus. Blacks and Hispanics have a high morbidity and mortality due to cardiovascular disease and diabetes mellitus in the general population. Since metabolic syndrome is a risk factor for development of type 2 diabetes and cardiovascular disease, understanding any racial and ethnic differences in metabolic syndrome among women with polycystic ovarian syndrome is important for prevention strategies. However, data regarding racial/ethnic differences in metabolic phenotype among women with polycystic ovary syndrome are inconsistent.

OBJECTIVE: We sought to determine if there are racial/ethnic differences in insulin resistance, metabolic syndrome, and hyperandrogenemia in women with polycystic ovarian syndrome.

STUDY DESIGN: We conducted secondary data analysis of a prospective multicenter, double-blind controlled clinical trial, the Pregnancy in Polycystic Ovary Syndrome II study, conducted in 11 academic health centers. Data on 702 women with polycystic ovarian syndrome aged 18-40 years who met modified Rotterdam criteria for the syndrome and wished to conceive were included in the study. Women were grouped into racial/ethnic categories: non-Hispanic whites, non-Hispanic blacks, and Hispanic. The main outcomes were the prevalence of insulin resistance, metabolic syndrome, and hyperandrogenemia in the different racial/ethnic groups.

RESULTS: Body mass index (35.1 $\pm$ 9.8 vs 35.7 $\pm$ 7.9 vs 36.4 $\pm$ 7.9 kg/m^2) and waist circumference (106.5 $\pm$ 21.6 vs 104.9 $\pm$ 16.4 vs 108.7 $\pm$ 7.3 cm) did not differ significantly between non-Hispanic white, non-Hispanic black, and Hispanic women. Hispanic women with polycystic ovarian syndrome had a significantly higher prevalence of hirsutism (93.8% vs 86.8%), abnormal free androgen index (75.8% vs 56.5%), abnormal homeostasis model assessment (52.3% vs 38.4%), and hyperglycemia (14.8% vs 6.5%), as well as lower sex hormone binding globulin compared to non-Hispanic whites. Non-Hispanic black women had a significantly lower prevalence of metabolic syndrome (24.5% vs 42.2%) compared with Hispanic women, and lower serum triglyceride levels compared to both Hispanics and non-Hispanic whites (85.7 $\pm$ 37.3 vs 130.2 $\pm$ 57.0 vs 120.1 $\pm$ 60.5 mg/dL, $P < .01$), with a markedly lower prevalence of hypertriglyceridemia (5.1% vs 28.3% vs 30.5%, $P < .01$) compared to the other 2 groups.

CONCLUSION: Hispanic women with polycystic ovarian syndrome have the most severe phenotype, both in terms of hyperandrogenism and metabolic criteria. Non-Hispanic black women have an overall milder polycystic ovarian syndrome phenotype than Hispanics and in some respects, than non-Hispanic white women.

Key words: ethnicity, metabolism, phenotype, polycystic ovary syndrome, race, sex steroids

Introduction

Polycystic ovary syndrome (PCOS) has significant public health importance with a high prevalence of metabolic syndrome and diabetes and potential long-term health consequence of cardiovascular disease (CVD). In the general population, blacks and Hispanics have a high morbidity and mortality due to CVD and type 2 diabetes mellitus (T2DM).[1,2] Since metabolic syndrome is a known risk factor for progression to CVD and T2DM,[3,4] understanding the prevalence of metabolic syndrome by race and ethnicity in women with PCOS is important in targeting the relevant population(s) for early prevention and treatment.

In the US adolescent and adult population, non-Hispanic blacks (NHBs) have a similar or even lower prevalence of metabolic syndrome compared to non-Hispanic whites (NHWs).[5,6] This may seem paradoxical as NHBs have a higher insulin resistance and higher prevalence of T2DM and CVD.[5,7] Few studies have evaluated racial and ethnic differences in metabolic syndrome in women with PCOS, and these have had conflicting findings.[8,9] Hillman and colleagues[8] showed an increased prevalence of metabolic syndrome in black compared with white women with PCOS, although others have not shown any significant differences between the 2 racial groups.[9] Previous studies have shown similar prevalence of metabolic syndrome in Hispanic Americans compared with NHBs and NHWs although these studies included relatively few Hispanics.[9,10] There is also conflicting evidence whether the prevalence of insulin resistance differs between the racial and ethnic groups[11-13] and it has been suggested that any differences may be driven by body mass index (BMI) independent of race.[10] Observed differences in the metabolic phenotypes across racial and ethnic groups in women with PCOS in various studies may be due to differences in study design and sample sizes of the relevant populations. A prospective study with a large sample size taking into

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consideration important comorbidities such as obesity, abdominal adiposity, and insulin resistance is essential to determine if racial/ethnic differences exist in the metabolic phenotype of PCOS.

We sought to determine whether there are any racial and ethnic differences in the prevalence of hyperandrogenemia, insulin resistance, and metabolic syndrome among women with PCOS using a secondary analysis of data obtained from a multicenter, randomized controlled clinical trial, the Pregnancy in PCOS (PPCOS) II study.[14,15]

Materials and Methods

Study design

These data were derived from the PPCOS II trial. The design of the trial as well as the baseline characteristics and outcomes of this study have been published.[14-16] Briefly, the PPCOS II trial was a multicenter, randomized, controlled, double-blind clinical trial sponsored by the Reproductive Medicine Network (RMN) and conducted at 11 clinical sites across the United States (ClinicalTrials.gov number: NCT00719186). The purpose of the trial was to determine live birth rates after clomiphene citrate or letrozole ovulation induction for up to 5 treatment cycles in 750 infertile women. In this secondary analysis, we evaluated the baseline androgenic and metabolic phenotype between different racial and ethnic groups recruited to the study. Institutional review board approval was obtained at each study site and participants underwent the written informed consent process.

Participants

Women with PCOS aged 18-40 years were included if they met modified Rotterdam criteria for PCOS as follows: ovulatory dysfunction in combination with either hyperandrogenemia based on hirsutism or an elevated testosterone level and/or polycystic ovaries defined by increased number of small antral follicles ($\geq$12 follicles <10 mm in diameter) or an increased individual ovarian volume (>10 cm^3) in $\geq$1 ovary or both. Other disorders that mimic the PCOS including thyroid disease and prolactin excess were excluded.[17]

All measurements used in this analysis were taken at the screening visits; thus, prior to any treatment. Each participant had her height and weight and waist-and-hip circumferences measured at the initial screening visit. Blood pressure (BP) was determined in the arm in a sitting position after a 5-minute rest. A transvaginal ultrasound was performed to evaluate the ovaries for evidence of polycystic ovarian morphology.

Hirsutism, acne score, and sebum measurements were assessed by trained study personnel as previously reported.[14-16] Facial sebum was measured using a sebumeter on the middle forehead. Hirsutism was assessed using the modified Ferriman-Gallwey score. Acne was assessed using both inflammatory and noninflammatory facial lesions including open and closed comedones, papules, pustules, and nodules. These were counted to obtain a total acne score.

Racial and ethnic groups

Participants self-reported race as black, white, Asian, American Indian or Native Alaskan Americans, or Native Hawaiian or Pacific Islander. Ethnicity was reported as Hispanic or non-Hispanic. For simplicity, we used the classification used by the Endocrine Society Scientific Statement on Health Disparities[18] and classified the groups as NHWs consisting of whites who did not also self-identify as Hispanic; NHBs consisting of blacks who did not also self-identify as Hispanic; and Hispanics, which consisted of any person selecting Hispanic as their ethnicity. Mixed race (N = 22), Asians (N = 23), and Native Americans or Native Alaskans (n = 3) were excluded from the analysis in view of their small numbers and for the sake of clarity.

Laboratory analysis

Fasting blood samples were obtained at screening, batched, and analyzed at the Ligand Assay and Analysis Core Laboratory at University of Virginia.[13,14] All assays had intraassay and interassay coefficients of variation <10%.[14] Total testosterone and proinsulin were measured using radioimmunoassay (RIA).[14] The testosterone RIA has been shown to be similar to commonly used liquid chromatography-tandem mass spectrometry assays.[19] Sex hormone binding globulin (SHBG) and insulin were measured by chemiluminescent 2-site assay. Free androgen index (FAI) was calculated from results of total testosterone and SHBG using the formula: FAI = total testosterone in nmol/L/SHBG in nmol/L $\times$ 100 as previously described.[20] Glucose was measured by the glucose oxidase method. Lipid profiles including total cholesterol, high-density lipoprotein (HDL)-cholesterol, and triglycerides (TG) were measured using the Abbott Architect c16000 automated analyzer (Abbott Diagnostics, Lake Forest, IL). Low-density lipoprotein cholesterol was calculated using the standard Friedewald equation.

Outcome variables

Clinical measures of hyperandrogenemia included hirsutism, acne score, and sebum. A Ferriman-Gallwey score of $\geq$8 was considered evidence of hirsutism.[21] Abnormal acne was defined as an acne score >5 and an abnormal sebum score was defined as $\geq$100. Biochemical evidence of hyperandrogenemia was assessed using total testosterone, SHBG, FAI, and androstenedione. Total testosterone >50 ng/dL and FAI >5 were considered evidence of biochemical hyperandrogenemia.

Insulin resistance was assessed using fasting insulin, proinsulin, and the homeostasis model assessment (HOMA): HOMA = (fasting glucose in nmol/L $\times$ fasting insulin in μIU/mL)/22.5. Fasting insulin >20 μIU/mL and HOMA $\geq$3.8 were considered abnormal.[22] The metabolic syndrome was defined as the presence of 3 of 5 risk factors consisting of: (1) central obesity (waist circumference >88 cm); (2) low HDL-cholesterol (<50 mg/dL); (3) hypertriglyceridemia (TG $\geq$150 mg/dL); (4) hypertension (systolic BP $\geq$130 mm Hg or diastolic BP $\geq$85 mm Hg); and (5) fasting hyperglycemia (fasting glucose $\geq$100 mg/dL).[23]

The Framingham modified 10-year risk of coronary heart disease was determined by calculating a risk score based on age, HDL- cholesterol, total cholesterol, hypertension, and smoking status.[24]

TABLE 1
Baseline characteristics of non-Hispanic white, non-Hispanic black, and Hispanic women with polycystic ovary syndrome

	Race/ethnicity			
Variables	Non-Hispanic white	Non-Hispanic black	Hispanic American	Overall *P* value
Age, y				
N	476	98	128	
Mean ± SD	28.8 ± 4.2	28.7 ± 4.9	29.2 ± 4.1	.74
Body mass index, kg/m^2				
N	476	98	128	
Mean ± SD	35.1 ± 9.8	35.7 ± 7.9	36.4 ± 7.9	.35
Abnormal ≥30 (%)	317/476 (66.6)	70/98 (71.4)	97/128 (75.8)	.12
Waist circumference, cm				
N	475	98	127	
Mean ± SD	106.5 ± 21.6	104.9 ± 16.4	108.7 ± 17.3	.36
Abnormal >88 (%)	361/475 (76.0)	81/98 (82.7)	108/127 (85.0)	.05
Polycystic ovarian morphology in either ovary				
N (%)	473/476 (99.4)	97/98 (99.0)	128/128 (100.0)	.57
Right ovarian volume, cm^3				
N	468	97	128	
Mean ± SD	12.8 ± 7.8	12.4 ± 6.0	12.6 ± 7.3	.92
Left ovarian volume, cm^3				
N	471	97	127	
Mean ± SD	11.8 ± 6.5	11.4 ± 5.7	11.5 ± 6.1	.84

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Statistical analysis

In the descriptive analysis, continuous data are presented as means (SD), with one-way analysis of variance or *t* test used to determine differences among the racial/ethnic groups. Categorical data are presented as number of subjects/total number (percentage), with χ^2 analysis or Fisher exact test used to compare differences between the racial/ethnic groups. Statistical significance was defined as a 2-sided *P* value of <.05. All analyses were performed in software (SAS, V9.3; SAS Institute, Cary, NC).

Results

A total of 750 women were recruited for the PPCOS II trial. There were 702 women with PCOS available for inclusion into this secondary analysis after exclusion of women of mixed race, Asians, Native Americans, or Native Alaskans. The final analytic sample comprised 476 NHWs, 98 NHBs, and 128 Hispanics.

Baseline characteristics

There were no significant differences in the mean ages between NHWs, NHBs, and Hispanic Americans. BMI and waist circumference did not differ among the 3 groups. Educational level of high school or less was more commonly seen in Hispanic women. Income of <\$50,000 was more common in NHBs.

All Hispanic women had polycystic ovarian morphology on either one or both ovaries and 99.4% of NHWs and 98.9% of NHBs had at least 1 ovary with polycystic ovarian morphology. There were no significant differences in right or left ovarian volumes between the racial/ethnic groups (Table 1).

Clinical and biochemical hyperandrogenemia

Hispanics had a higher prevalence of hirsutism and acne compared to both NHBs and NHWs although there were no significant differences between NHBs and NHWs. NHBs had a significantly higher mean sebum score compared to Hispanics and NHWs but there were no significant differences between Hispanics and NHWs. There were no significant differences in total testosterone between the groups. Mean serum SHBG levels were significantly lower in Hispanics compared to both NHBs and NHWs and therefore Hispanics had a significantly higher FAI compared to NHWs. There were no significant

TABLE 2

Clinical and biochemical hyperandrogenemia in non-Hispanic white, non-Hispanic black, and Hispanic women with polycystic ovary syndrome

Variables	Race/ethnicity			
	Non-Hispanic white	Non-Hispanic black	Hispanic	Overall *P* value
Hirsutism				
N	476	98	128	
Mean ± SD	17.0 ± 8.7	15.8 ± 8.5	17.6 ± 7.5	.25
Abnormal ≥8 (%)	413/476 (86.8)[a]	81/98 (82.7)[b]	120/128 (93.8)[c,d]	.03
Acne				
N	475	98	128	
Mean ± SD	8.2 ± 16.3[a]	5.8 ± 16.7[b]	13.6 ± 14.9[c,d]	<.01
Abnormal >5 (%)	167/475 (35.2)[a]	25/98 (25.5)[b]	82/128 (64.1)[c,d]	<.01
Sebum, μg/cm^2				
N	465	93	125	
Mean ± SD	107.1 ± 56.2[a]	121.4 ± 61.8[b,c]	103.3 ± 45.8[d]	.04
Abnormal ≥100 (%)	221/465 (47.5)	53/93 (57.0)	58/125 (46.4)	.22
Total testosterone, ng/dL				
N	474	98	128	
Mean ± SD	54.0 ± 27.1	61.2 ± 37.7	53.6 ± 26.2	.07
Abnormal >50 (%)	225/474 (47.5)	57/98 (58.2)	60/128 (46.9)	.14
SHBG, nmol/L				
N	474	98	128	
Mean ± SD	36.2 ± 25.1[a]	33.4 ± 18.7[b]	26.5 ± 18.1[c,d]	<.01
Abnormal ≤25 (%)	194/474 (40.9)[a]	38/98 (38.8)[b]	79/128 (61.7)[c,d]	<.01
Free androgen index				
N	474	98	128	
Mean ± SD	7.3 ± 5.6[a]	8.5 ± 7.6	9.0 ± 5.6[c]	.01
Abnormal >5 (%)	268/474 (56.5)[a]	63/98 (64.3)	97/128 (75.8)[c]	<.01
Androstenedione, ng/mL				
N	474	98	128	
Mean ± SD	4.2 ± 1.7	4.6 ± 2.2	4.0 ± 1.4	.05

SHBG, sex hormone binding globulin.

[a] Vs; [b] Vs; [c] Significantly different at $P < .05$; [d] Significantly different at $P < .05$.

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differences in mean serum androstenedione levels between the groups (Table 2).

Insulin resistance

Hispanic women were more insulin resistant than NHWs as shown by significantly higher mean fasting insulin, proinsulin, and HOMA levels. Moreover, a significantly higher proportion of Hispanics had abnormal fasting insulin and HOMA compared to NHWs. These differences persisted particularly in women with BMI <35 kg/m^2, although not seen in women with BMI ≥35 kg/m^2 (Supplemental Tables 1 and 2). Moreover, the differences persisted when controlling for study site (Supplemental Table 3). There were no significant differences in insulin resistance parameters when NHBs were compared with NHWs and Hispanics (Table 3).

Metabolic syndrome

Hispanic women had a markedly higher prevalence of metabolic syndrome compared to NHBs (42.2% vs 24.5%, respectively) but not to NHWs (33.8%). This difference was not seen in women

TABLE 3
Insulin resistance in non-Hispanic white, non-Hispanic black, and Hispanic women with polycystic ovary syndrome

Variables	Race/ethnicity Non-Hispanic white	Non-Hispanic black	Hispanic	Overall *P* value
Fasting insulin, μIU/mL				
N	474	98	128	
Mean ± SD	17.8 ± 19.6	22.3 ± 23.2	23.9 ± 48.4	.05
Abnormal >20 (%)	151/474 (31.9)[a]	34/98 (34.7)	57/128 (44.5)[b]	.03
Fasting proinsulin, pmol/L				
N	474	98	128	
Mean ± SD	16.9 ± 12.3[a]	19.4 ± 18.9	21.4 ± 18.1[b]	.01
HOMA				
N	474	98	128	
Mean ± SD	3.9 ± 4.9[a]	5.0 ± 5.7	6.2 ± 19.2[b]	.04
Abnormal ≥3.8 (%)	182/474 (38.4)[a]	45/98 (45.9)	67/128 (52.3)[b]	.01

HOMA, homeostasis model assessment.

[a] Vs; [b] Significantly different at $P < .05$.

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with BMI <35 kg/m^2 but persisted in women with BMI ≥35 kg/m^2 (Supplemental Tables 4 and 5). When controlling for study site, the difference in metabolic syndrome was no longer significant (Supplemental Table 6). The 10-year CVD risk score did not significantly differ between the groups. NHBs and Hispanics had a significantly higher prevalence of systolic hypertension compared with NHW, although there were no significant differences in diastolic BP between the groups (Table 4).

Hispanic women had a significantly higher fasting glucose level and higher proportion of patients with abnormal glucose levels compared with NHWs. There were no significant differences in total or low-density lipoprotein cholesterol between groups. Hispanics had a significantly higher mean HDL level compared with NHWs and NHBs. NHBs had a significantly lower mean serum TG levels (85.7 ± 37.3 vs 120.1 ± 60.5 vs 130.2 ± 57.0 mg/dL, $P < .01$) and strikingly lower proportion of women with hypertriglyceridemia (5.1% vs 28.3% vs 30.5%, $P < .01$) compared with both NHWs and Hispanics regardless of BMI and study site (Table 4; Supplemental Tables 4-6).

Comment

This secondary analysis of a large, prospective, randomized, multicenter trial indicates that there are significant racial/ethnic differences in insulin resistance, metabolic syndrome, and hyperandrogenemia in women with PCOS. This is especially striking as we report no differences in obesity or abdominal adiposity among the groups. Hispanic women have the most severe phenotype overall, with a significantly higher prevalence of hyperandrogenemia, insulin resistance, systolic hypertension, and hyperglycemia than NHWs. In contrast, NHBs had an overall PCOS phenotype that was comparable to or even milder than that of NHWs. NHB women with PCOS had a far lower prevalence of metabolic syndrome than Hispanic women and less hypertriglyceridemia than both Hispanics and NHWs.

In the general US population, NHBs and Mexican Americans have greater hyperinsulinemia and insulin resistance[25] compared with NHWs, which is independent of obesity. In accordance with these findings, we found a higher prevalence of insulin resistance in Hispanics compared with NHWs particularly in women who were not obese. In contrast to these findings, we did not observe any differences in insulin resistance between NHBs and NHWs with PCOS, which is consistent with another large prospective multicenter trial[13] of women with PCOS. Although several studies have indicated that hyperinsulinemia and insulin resistance is worse in black,[11] Mexican American,[22] and Caribbean Hispanic[26] compared to white women with PCOS, data from other studies imply that these differences are almost entirely driven by BMI and are independent of race.[10]

The higher prevalence of hyperandrogenemia seen in Hispanics but not in other racial/ethnic groups may be related to the higher prevalence of hyperinsulinemia, insulin resistance, and consequently low serum SHBG observed in this ethnic group. Insulin stimulates androgen production and reduces hepatic SHBG synthesis,[27] thereby increasing bioavailable androgens. NHBs and NHWs did not differ with respect to SHBG and insulin resistance and hence demonstrated no differences in the prevalence of hirsutism or serum FAI levels, which is consistent with findings in the general population[28] and in other studies of women with PCOS.[10,13,29,30]

The overall prevalence of metabolic syndrome in women with PCOS has been

TABLE 4
Metabolic phenotype of non-Hispanic white, non-Hispanic black, and Hispanic women with polycystic ovary syndrome

Variables	Race/ethnicity Non-Hispanic white	Non-Hispanic black	Hispanic	Overall *P* value
Metabolic syndrome (%)	161/476 (33.8)	24/98 (24.5)[a]	54/128 (42.2)[b]	.02
Cardiovascular disease risk score				
N	475	98	128	
Mean ± SD	1.4 ± 0.9	1.6 ± 1.2	1.4 ± 0.8	.14
Abnormal >1.1 (%)	244/475 (51.4)	50/98 (51.0)	72/128 (56.3)	.60
Systolic BP, mm Hg				
N	475	98	128	
Mean ± SD	119.1 ± 12.5	122.4 ± 14.9	120.5 ± 12.5	.05
Abnormal ≥130 (%)	90/475 (19.0)[a,c]	28/98 (28.6)[b]	35/128 (27.3)[d]	.03
Diastolic BP, mm Hg				
N	475	98	128	
Mean ± SD	76.8 ± 9.2	78.9 ± 9.8	77.0 ± 10.0	.12
Abnormal ≥85 (%)	97/475 (20.4)	25/98 (25.5)	32/128 (25.0)	.36
Fasting glucose, mg/dL				
N	474	98	128	
Mean ± SD	84.7 ± 11.7[a]	87.3 ± 15.2	89.2 ± 13.6[b]	<.01
Abnormal ≥100 (%)	31/474 (6.5)[a]	7/98 (7.1)	19/128 (14.8)[b]	.01
HDL cholesterol, mg/dL				
N	474	98	128	
Mean ± SD	37.1 ± 10.8[a]	37.2 ± 10.1[c]	40.5 ± 9.5[b,d]	.01
Abnormal <50 (%)	413/474 (87.1)	87/98 (88.8)	103/128 (80.5)	.11
LDL cholesterol, mg/dL				
N	474	98	128	
Mean ± SD	121.2 ± 34.1	124.6 ± 32.2	116.7 ± 30.5	.19
Abnormal >130 (%)	168/474 (35.4)	39/98 (39.8)	44/128 (34.4)	.66
Triglycerides, mg/dL				
N	474	98	128	
Mean ± SD	120.1 ± 60.5[a]	85.7 ± 37.3[b,c]	130.2 ± 57.0[d]	<.01
Abnormal ≥150 (%)	134/474 (28.3)[a]	5/98 (5.1)[b,c]	39/128 (30.5)[d]	<.01

BP, blood pressure; *HDL*, high-density lipoprotein; *LDL*, low-density lipoprotein.

[a] Vs; [b] Significantly different at $P < .05$; [c] Vs; [d] Significantly different at $P < .05$.

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reported to range from 33-46%,[9,31,32] which is comparable to what we observed in Hispanics and NHWs in this study, but much higher than that of NHBs. Hispanic Americans had the highest prevalence of metabolic syndrome in this study, which is consistent with findings from the National Health and Nutrition Examination Survey involving US women,[5] but contrary to findings in women with PCOS where no differences have been noted between the groups.[9,10] In the general US adult and adolescent population, the high prevalence of the metabolic syndrome in Hispanics is attributed mostly to a higher prevalence of obesity, insulin resistance, and diabetes.[33] In our population, the high prevalence of metabolic syndrome in Hispanics may be due to the high proportion of abnormal systolic BP and fasting glucose.

We did not find any differences in prevalence of metabolic syndrome between NHBs and NHWs. This is consistent with the general US adolescent and adult population where the prevalence of metabolic syndrome is either similar or lower in blacks compared to whites.[5,6] However, inconsistent racial/ethnic differences in the prevalence of metabolic syndrome have been reported in women with PCOS.[8-10] Hillman and colleagues[8] reported a significantly higher prevalence of metabolic syndrome in adolescent and adult black compared to white women with PCOS. On the contrary, others have reported similar prevalence of metabolic syndrome in NHBs and NHWs, which is consistent with our findings.[9,10] The relative absence of the dyslipidemia associated with metabolic syndrome is the most likely reason for the lower prevalence of metabolic syndrome in NHBs compared with NHWs in the general adult population.[34]

Elevated TGs are one of the components of the metabolic syndrome because of their strong association with insulin resistance.[35] The significantly low TGs seen in NHBs in this study is consistent with what is reported in the general population[34] as well as in women with PCOS.[8,9,36] Although Hispanics in this study were more insulin resistant than NHWs, they did not differ with respect to TG levels, similar to data from others comparing Mexican Americans to NHWs.[37] The low TG levels in NHBs have been attributed to decreased production secondary to low visceral adipose tissue mass and low intrahepatic TG content as well as increased clearance due to high lipoprotein lipase activity and low apolipoprotein CIII levels.[38] However, in view of the high overall cardiovascular morbidity for NHBs, which exceeds that of NHWs, it has been suggested that a lower threshold for TG may be applicable to NHBs as a true harbinger of CVD risk.[6] Adverse effects of relative dyslipidemia may therefore become evident at lower TG levels for NHBs, who may have a greater sensitivity to increases in TG.[39]

A major strength of this analysis is the standardized and prospective collection of data at the initial screening visit. All measurements and scoring were done by trained personnel using systematic techniques. Serum measurements were done in a single, central laboratory. Moreover, because the patients were recruited from 11 sites across the United States with geographic diversity, the findings are widely generalizable. The main limitation of this study is that our population was young and included healthy women seeking conception and therefore all potential women with PCOS available at each site, including those with uncontrolled chronic medical conditions, were not assessed. The findings therefore may not be extrapolated to an older PCOS population or to those with prevalent disease. However, the findings are likely applicable to the general PCOS population presenting to an infertility clinic. Although self-reporting of race is fairly well validated, it does have its limitations. Race as a variable is not always simply about the genetic variable, rather it reflects the combination of genetics and culture as well as environmental influence in a way that is hard to quantify and therefore the lack of granular information is a limitation in this study as well as most studies on race. Finally we did not perform intensive tests of insulin action, which provide a more accurate assessment of insulin resistance.

In summary, we found that Hispanic women have an overall severe PCOS phenotype in terms of both hyperandrogenism and dysmetabolism. In contrast, we observed that NHBs appear to have a milder metabolic phenotype in view of the lower prevalence of metabolic syndrome and hypertriglyceridemia. Further studies are needed to understand the dichotomy between the low TG levels and high prevalence of chronic CVD in NHBs in the general population. It is possible that different thresholds for cardiometabolic markers should be used to detect preclinical disease in women of different races and ethnicity. ■

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SUPPLEMENTAL TABLE 1
Insulin resistance in non-Hispanic white, non-Hispanic black, and Hispanic women with polycystic ovary syndrome (body mass index <35)

Variables	Race/ethnicity			
	Non-Hispanic white	Non-Hispanic black	Hispanic	Overall *P* value
Fasting insulin, μIU/mL				
N	236	46	55	
Mean $\pm$ SD	9.4 $\pm$ 12.0[a]	13.8 $\pm$ 11.5	17.0 $\pm$ 21.4[b]	$<$.01
Abnormal $>$20 (%)	26/236 (11.0)[a]	7/46 (15.2)	16/55 (29.1)[b]	$<$.01
Fasting proinsulin, pmol/L				
N	236	46	55	
Mean $\pm$ SD	11.7 $\pm$ 8.8[a,c]	16.1 $\pm$ 17.5[b]	17.0 $\pm$ 16.0[d]	$<$.01
HOMA				
N	236	46	55	
Mean $\pm$ SD	2.0 $\pm$ 2.6[a,c]	3.2 $\pm$ 3.4[b]	3.7 $\pm$ 4.9[d]	$<$.01
Abnormal $\geq$3.8 (%)	33/236 (14.0)[a,c]	12/46 (26.1)[b]	17/55 (30.9)[d]	$<$.01

HOMA, homeostasis model assessment.

[a] Vs; [b] Significantly different at $P < .05$; [c] Vs; [d] Significantly different at $P < .05$.

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SUPPLEMENTAL TABLE 2
Insulin resistance in non-Hispanic white, non-Hispanic black, and Hispanic women with polycystic ovary syndrome (body mass index ≥35)

Variables	Race/ethnicity			
	Non-Hispanic white	Non-Hispanic black	Hispanic	Overall *P* value
Fasting insulin, μIU/mL				
N	238	52	73	
Mean $\pm$ SD	26.1 $\pm$ 22.0	29.8 $\pm$ 28.0	29.1 $\pm$ 61.0	.68
Abnormal $>$20 (%)	125/238 (52.5)	27/52 (51.9)	41/73 (56.2)	.84
Fasting proinsulin, pmol/L				
N	238	52	73	
Mean $\pm$ SD	22.2 $\pm$ 13.0	22.4 $\pm$ 19.8	24.8 $\pm$ 18.8	.43
HOMA				
N	238	52	73	
Mean $\pm$ SD	5.8 $\pm$ 5.7	6.5 $\pm$ 6.9	8.0 $\pm$ 25.0	.39
Abnormal $\geq$3.8 (%)	149/238 (62.6)	33/52 (63.5)	50/73 (68.5)	.65

HOMA, homeostasis model assessment.

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SUPPLEMENTAL TABLE 3

Insulin resistance in non-Hispanic white, non-Hispanic black, and Hispanic women with polycystic ovary syndrome adjusted by study site

Variables	Race/ethnicity			
	Non-Hispanic white	Non-Hispanic black	Hispanic	Overall *P* value
Fasting insulin, μIU/mL				
N	474	98	128	
Mean ± SD	17.8 ± 19.6	22.3 ± 23.2	23.9 ± 48.4	.03
Abnormal >20 (%)	151/474 (31.9)	34/98 (34.7)	57/128 (44.5)	.02
Fasting proinsulin, pmol/L				
N	474	98	128	
Mean ± SD	16.9 ± 12.3	19.4 ± 18.9	21.4 ± 18.1	<.01
HOMA				
N	474	98	128	
Mean ± SD	3.9 ± 4.9	5.0 ± 5.7	6.2 ± 19.2	.12
Abnormal ≥3.8 (%)	182/474 (38.4)	45/98 (45.9)	67/128 (52.3)	<.01

HOMA, homeostasis model assessment.

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SUPPLEMENTAL TABLE 4

Metabolic phenotype of non-Hispanic white, non-Hispanic black, and Hispanic women with polycystic ovary syndrome (body mass index <35)

Variables	Race/ethnicity Non-Hispanic white	Non-Hispanic black	Hispanic	Overall *P* value
Metabolic syndrome (%)	44/236 (18.6)	10/46 (21.7)	17/55 (30.9)	.13
Systolic BP, mm Hg				
N	236	46	55	
Mean ± SD	115.3 ± 11.9	117.8 ± 13.8	117.2 ± 11.5	.31
Abnormal ≥130 (%)	26/236 (11.0)	8/46 (17.4)	11/55 (20.0)	.14
Diastolic BP, mm Hg				
N	236	46	55	
Mean ± SD	74.2 ± 8.9	76.1 ± 9.3	73.3 ± 9.8	.30
Abnormal ≥85 (%)	30/236 (12.7)	8/46 (17.4)	8/55 (14.6)	.68
Fasting glucose, mg/dL				
N	236	46	55	
Mean ± SD	82.0 ± 11.3[a]	88.4 ± 18.0[b]	85.6 ± 10.2	<.01
Abnormal ≥100 (%)	9/236 (3.8)	5/46 (10.9)	3/55 (5.5)	.13
HDL cholesterol, mg/dL				
N	236	46	55	
Mean ± SD	40.3 ± 11.1	40.2 ± 10.1	41.1 ± 10.6	.77
Abnormal <50 (%)	190/236 (80.5)	38/46 (82.6)	41/55 (74.6)	.54
LDL cholesterol, mg/dL				
N	236	46	55	
Mean ± SD	120.8 ± 32.3	129.2 ± 32.7	121.1 ± 33.5	.27
Abnormal >130 (%)	88/236 (37.3)	22/46 (47.8)	23/55 (41.8)	.38
Triglycerides, mg/dL				
N	236	46	55	
Mean ± SD	102.3 ± 55.9[a]	85.9 ± 38.8[c]	136.0 ± 60.3[b,d]	<.01
Abnormal ≥150 (%)	49/236 (20.8)[a,c]	3/46 (6.5)[b]	19/55 (34.6)[d]	<.01

BP, blood pressure; *HDL*, high-density lipoprotein; *LDL*, low-density lipoprotein.

[a] Vs; [b] Significantly different at $P < .05$; [c] Vs; [d] Significantly different at $P < .05$.

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SUPPLEMENTAL TABLE 5

Metabolic phenotype of non-Hispanic white, non-Hispanic black, and Hispanic women with polycystic ovary syndrome (body mass index ≥35)

Variables	Race/ethnicity			
	Non-Hispanic white	Non-Hispanic black	Hispanic	Overall *P* value
Metabolic syndrome (%)	117/240 (48.8)[a]	14/52 (26.9)[b,c]	37/73 (50.7)[d]	.01
Systolic BP, mm Hg				
N	239	52	73	
Mean $\pm$ SD	122.8 $\pm$ 11.9	126.4 $\pm$ 14.8	123.0 $\pm$ 12.7	.16
Abnormal $\geq$130 (%)	64/239 (26.8)	20/52 (38.5)	24/73 (32.9)	.19
Diastolic BP, mm Hg				
N	239	52	73	
Mean $\pm$ SD	79.3 $\pm$ 8.7	81.4 $\pm$ 9.6	79.7 $\pm$ 9.4	.32
Abnormal $\geq$85 (%)	67/239 (28.0)	17/52 (32.7)	24/73 (32.9)	.64
Fasting glucose, mg/dL				
N	238	52	73	
Mean $\pm$ SD	87.3 $\pm$ 11.4[a]	86.3 $\pm$ 12.3[c]	92.0 $\pm$ 15.1[b,d]	.01
Abnormal $\geq$100 (%)	22/238 (9.2)[a]	2/52 (3.9)[c]	16/73 (21.9)[b,d]	$<$.01
HDL cholesterol, mg/dL				
N	238	52	73	
Mean $\pm$ SD	34.1 $\pm$ 9.4[a]	34.7 $\pm$ 9.5[c]	39.9 $\pm$ 8.6[b,d]	$<$.01
Abnormal $<$50 (%)	223/238 (93.7)[a]	49/52 (94.2)	62/73 (84.9)[b]	.04
LDL cholesterol, mg/dL				
N	238	52	73	
Mean $\pm$ SD	121.6 $\pm$ 35.9	120.4 $\pm$ 31.4	113.4 $\pm$ 27.8	.19
Abnormal $>$130 (%)	80/238 (33.6)	17/52 (32.7)	21/73 (28.8)	.74
Triglycerides, mg/dL				
N	238	52	73	
Mean $\pm$ SD	137.8 $\pm$ 59.9[a]	85.6 $\pm$ 36.3[b,c]	125.8 $\pm$ 54.3[d]	$<$.01
Abnormal $\geq$150 (%)	85/238 (35.7)[a]	2/52 (3.9)[b,c]	20/73 (27.4)[d]	$<$.01

BP, blood pressure; *HDL*, high-density lipoprotein; *LDL*, low-density lipoprotein.

[a] Vs; [b] Significantly different at $P < .05$; [c] Vs; [d] Significantly different at $P < .05$.

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SUPPLEMENTAL TABLE 6

Metabolic phenotype of non-Hispanic white, non-Hispanic black, and Hispanic women with polycystic ovary syndrome adjusted by study site

Variables	Race/ethnicity			
	Non-Hispanic white	Non-Hispanic black	Hispanic	Overall *P* value
Metabolic syndrome (%)	161/476 (33.8)	24/98 (24.5)	54/128 (42.2)	.22
Systolic BP, mm Hg				
N	475	98	128	
Mean ± SD	119.1 ± 12.5	122.4 ± 14.9	120.5 ± 12.5	.01
Abnormal ≥130 (%)	90/475 (19.0)	28/98 (28.6)	35/128 (27.3)	.15
Diastolic BP, mm Hg				
N	475	98	128	
Mean ± SD	76.8 ± 9.2	78.9 ± 9.8	77.0 ± 10.0	.01
Abnormal ≥85 (%)	97/475 (20.4)	25/98 (25.5)	32/128 (25.0)	.38
Fasting glucose, mg/dL				
N	474	98	128	
Mean ± SD	84.7 ± 11.7	87.3 ± 15.2	89.2 ± 13.6	.15
Abnormal ≥100 (%)	31/474 (6.5)	7/98 (7.1)	19/128 (14.8)	.77
HDL cholesterol, mg/dL				
N	474	98	128	
Mean ± SD	37.1 ± 10.8	37.2 ± 10.1	40.5 ± 9.5	.80
Abnormal <50 (%)	413/474 (87.1)	87/98 (88.8)	103/128 (80.5)	.77
LDL cholesterol, mg/dL				
N	474	98	128	
Mean ± SD	121.2 ± 34.1	124.6 ± 32.2	116.7 ± 30.5	.63
Abnormal >130 (%)	168/474 (35.4)	39/98 (39.8)	44/128 (34.4)	.44
Triglycerides, mg/dL				
N	474	98	128	
Mean ± SD	120.1 ± 60.5	85.7 ± 37.3	130.2 ± 57.0	<.01
Abnormal ≥150 (%)	134/474 (28.3)	5/98 (5.1)	39/128 (30.5)	<.01

BP, blood pressure; *HDL*, high-density lipoprotein; *LDL*, low-density lipoprotein.

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