Dietary glycemic index is associated with less favorable anthropometric and metabolic profiles in polycystic ovary syndrome women with different phenotypes

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Objective: To compare glycemic index (GI) in the usual diet of polycystic ovary syndrome (PCOS) and control women and to investigate whether dietary GI is associated with body composition and anthropometric and metabolic variables across PCOS phenotypes.
Design: Cross-sectional study.
Setting: University hospital outpatient clinic.
Patient(s): Sixty-one women with PCOS and 44 nonhirsute women with ovulatory cycles.
Intervention(s): Metabolic work-up, biochemical and hormonal assays, assessment of body composition and rest metabolic rate, physical activity (pedometer), and food consumption (food frequency questionnaire).
Main outcome measure(s): GI, glycemic load, dietary intake, and hormone and metabolic profile in PCOS versus control and in PCOS women stratified by tertiles of GI and PCOS phenotype.
Result(s): Mean age was 23.7 ± 6.3 years. Participants with PCOS had higher body fat percentage, fasting insulin, insulin resistance, lipid accumulation product, and androgen levels compared with control women. PCOS and control women in the highest tertile of GI had higher body mass index and waist circumference than those in the lowest tertile. Dietary GI was higher in the classic PCOS group. Obesity and this more severe PCOS phenotype explained 28.3% of variance in dietary GI.
Conclusion(s): Dietary GI is increased in the classic PCOS phenotype and associated with a less favorable anthropometric and metabolic profile. Obesity and classic PCOS phenotype are age-independent predictors of higher dietary GI. (Fertil Steril® 2013;100:1081–8. ©2013 by American Society for Reproductive Medicine.)
Key Words: Diet, insulin resistance, hyperandrogenism, glycemic index, polycystic ovary syndrome

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Polycystic ovary syndrome (PCOS), a complex heterogeneous condition affecting women of reproductive age, is primarily characterized by ovulatory dysfunction and hyperandrogenism (1, 2). The prevalence of PCOS varies according to the diagnostic criteria used, with estimates ranging from 9% in women of reproductive age according to National Institutes of Health criteria up to 18% with Rotterdam criteria (1–3).

Obesity is a prevalent characteristic of PCOS (4, 5), ranging from 12.5% (6) to 100% (7), with a pooled estimated prevalence of 49%, as shown by a recent meta-analysis (8). The presence of obesity may exacerbate the metabolic and reproductive disorders

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associated with the syndrome (9), including insulin resistance (IR) and dyslipidemia (10–13). A meta-analysis (14) has shown that women with PCOS have higher levels of triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and total cholesterol (TC) and lower high-density lipoprotein cholesterol (HDL-C) levels compared with control women, regardless of body mass index (BMI).

It has been suggested that regular consumption of carbohydrate-rich foods, which promote a high glycemic response, increasing glucose and insulin levels (15–17), enhances the risk of obesity (18, 19). A meta-analysis of clinical trials has shown that diets based on foods with low glycemic response (or low glycemic index [GI]) and low carbohydrate content (low glycemic load [GL]) are more effective at reducing BMI and body fat mass than control diets (20). A recent study (21) showed that low-GI and/or -GL diets exert a protective effect against cardiovascular disease in adult women. One possible explanation for the association between high dietary GI and GL and cardiovascular disease is the adverse effects of these diets on blood lipid levels. Some studies have found that high-GI and -GL diets lead to an increase in TG (22) and LDL-C (22, 23) and to a reduction in HDL-C (24, 25), in addition to causing systemic inflammation (26).

Furthermore, it has been proposed that the prolonged consumption of high-GI foods increases the demand for insulin. This chronic hyperinsulinemia plays a critical role in the development of IR, eventually leading to type 2 diabetes (27). However, the few studies analyzing the association between reproductive or metabolic disturbances in PCOS and dietary GI and GL in women with PCOS have produced inconsistent results (28–32).

Therefore, the aims of the present study were to compare GI and GL in the usual diet of PCOS and control women and to investigate whether dietary GI and GL are associated with body composition as well as with anthropometric and metabolic variables in PCOS women with different phenotypes.

MATERIALS AND METHODS

Participants

This cross-sectional study was carried out with women aged 14 to 35 years, enrolled not earlier than two years after menarche, recruited by advertisement in the media between 2009 and 2012. We enrolled volunteers with: 1) hirsutism and irregular menses; 2) hirsutism and regular menses; and 3) regular menses and no hirsutism (control).

One hundred five participants met the inclusion criteria. PCOS was diagnosed in 61 patients according to the Rotterdam criteria: 39 were classified as classic PCOS (biochemical and/or clinical hyperandrogenism and oligo/amenorrheic [fewer than nine cycles/year] or anovulatory cycles, with or without polycystic ovary [PCO] appearance at ultrasound) and 22 as ovulatory PCOS (hirsute women with normal androgen levels, regular ovulatory cycles confirmed by luteal-phase progesterone >3.8 ng/mL, and PCO). PCO was defined as ovarian volume >10 mm^3 in at least one ovary. The control group included 44 nonhirsute women with regular and proven ovulatory cycles (luteal-phase progesterone >3.8 ng/mL). Neither the PCOS nor the control participants had received any drugs known to interfere with hormonal levels for ≥3 months before the study. Women diagnosed with other hyperandrogenic disorders (nonclassic congenital adrenal hyperplasia, Cushing syndrome, androgen-secreting neoplasms), thyroid disorders, or hyperprolactinemia were excluded, as previously reported (33–35). Other exclusion criteria were pregnancy, BMI >40 kg/m^2, and diabetes mellitus. The study protocol was approved by the local Institutional Review Board. Written informed consent was obtained from each of the subjects.

Study Protocol

Anthropometric measurements were performed in duplicate and included body weight, height, and waist circumference (waist measured at the midpoint between the lower rib margin and the iliac crest in a plane perpendicular to the long axis of the body, with the subject standing balanced on both feet ~20 cm apart with both arms hanging freely) (36–39). BMI was calculated according to World Health Organization (WHO) guidelines (40).

Hirsutism was defined as a modified Ferriman-Gallwey score ≥8 (41). Blood pressure was measured after a 10-minute rest, in the sitting position, with feet on the floor and the arm supported at heart level. All PCOS and control participants underwent transvaginal ultrasound or, if they were sexually inactive, abdominal ultrasound.

Hormonal and metabolic assessments were made between the 2nd and 10th days of the menstrual cycle or on any day if the patient was amenorrheic. All samples were obtained between 8 and 10 a.m. Blood samples were drawn after an overnight 12-hour fast for determination of plasma cholesterol, HDL-C, and TG. Glucose was measured before and 2 hours after the ingestion of a 75-g oral glucose load.

Blood samples were also drawn for measurements of insulin, SHBG and total testosterone (TT). IR was estimated by homeostasis model assessment (HOMA). HOMA index was calculated by multiplying insulin (μIU/mL) by glucose (mmol/L) and dividing the product by 22.5 (42). The lipid accumulation product (LAP) was calculated with the formula [waist (cm) − 58] × TG concentration (mmol/L), as previously reported (37, 43, 44). Free androgen index (FAI) was estimated by dividing TT (nmol/L) by SHBG nmol/L) and multiplying by 100.

PCOS and control groups were stratified by tertiles of GI.

Biochemical and Hormonal Assays

TC, HDL-C, TG, and glucose levels were determined by colorimetric-enzymatic methods (Bayer 1650 Advia System). LDL-C was determined indirectly by using the formula LDL-C = TC − HDL-C − TG/5 (45).

TT levels were measured by chemiluminescence (Siemens Advia Centaur XP), with a sensitivity of 0.10 ng/mL and intra- and interassay coefficients of variation (CVs) of 3.3% and 7.5%, respectively. SHBG was measured by chemiluminescence (Immulite 2000 Siemens), with a sensitivity of

0.02 nmol/L and intra- and interassay CVs of 5.3% and 6.6%, respectively. Plasma insulin levels were measured by electrochemiluminescence (Siemens Advia Centaur XP), with a sensitivity of 0.50 U/mL and intra- and interassay CVs of 2.8% and 2.1%, respectively.

Assessment of Body Composition and Rest Metabolic Rate

Body composition was assessed with the use of bioimpedance (Inbody 230; Biospace). This device directly measures the impedance of each body segment to 20 kHz and 100 kHz. Assessments were performed in the morning after a fast of ≥4 hours, with an empty bladder, with the use of a standard lab coat and stripped of all metal objects. Patients were instructed not to practice vigorous exercise the day before and on the day of the test (46).

Rest metabolic rate was assessed with the use of the Fitmate equipment (Cosmed). Patients were evaluated in the morning after a fast of ≥5 hours in a quiet, low-light, and temperature-controlled environment. Patients were instructed not to exercise the day before and on the day of the test, and not to consume caffeine and alcohol the day before and on the day of the test (47, 48).

Assessment of Habitual Physical Activity

Habitual physical activity was estimated with the use of a digital pedometer (BP 148 Techline). The device was configured individually according to the weight (kg) and step length of the individual.

Subjects were instructed to record the total number of steps taken each day in a recording sheet each night before sleep (49). Participants repeated this procedure over 6 consecutive days, generating an average weekly number of steps. They were encouraged not to alter their usual physical activity habits during the study.

Assessment of Food Consumption

The assessment of dietary intake was performed with the use of a food frequency questionnaire (FFQ) previously validated in the adult population of the city of Porto Alegre, Rio Grande do Sul (50). This questionnaire assesses 121 items of food consumption during the preceding month. The following were evaluated: calorie and carbohydrate intake, GI, GL, and intake of protein, saturated, monounsaturated, and polyunsaturated fatty acids, fiber, and micronutrientes (51–54). Nutritional composition was calculated with the use of the Brazilian Table of Food Composition.

GI was calculated according to the U.N. Food and Agriculture Organization/WHO (55). First, the percentage of each food type in relation to the total carbohydrate content was determined; this value was multiplied by the specific GI of each food type and divided by 100. The GI values for all foods listed in the FFQ were then added to predict the GI of the diet. The GL of each food type was calculated by multiplying the corresponding GI by the amount of carbohydrate contained in the daily serving of that food (g) and dividing by 100. The daily or meal GL was obtained by adding the GL of all foods consumed during the day or the meal, respectively. The GI values published in the International Table of Glycemic Index Values and Glycemic Load of Atkinson, Foster-Powell, and Brand-Miller (2008) were used, with white bread as the standard reference.

Statistical Analyses

The sample size was estimated based on a previous study with infertile patients (56), considering a power of 80% and alpha of 5%. To detect a difference of 2.0 points in GI between PCOS and control, 88 women would be required (44 in each group).

Results are expressed as mean ± SD or median and interquartile range. Comparisons between the two group means were analyzed by Student *t* test; comparisons between median values were analyzed with the Mann-Whitney *U* test; comparisons between tertiles of GI and groups were analyzed by two-way analysis of variance (ANOVA); comparisons between PCOS phenotypes and control were analyzed by one-way ANOVA; adjustments for BMI and age were done by analysis of covariance. Bonferroni adjustment was used for multiple comparisons. Variables with nongaussian distribution were log-transformed for statistical analysis and back-transformed for data presentation. Pearson correlation coefficient or Spearman test was calculated between variables, according to gaussian or nongaussian distribution, respectively. Comparisons between ratios were carried out with the use of the χ^2 test. Multiple linear regression analysis was used to determine the independent effect of age, BMI, and presence of classic PCOS phenotype with dietary GI as dependent variable. All analyses were performed with the Statistical Package for the Social Sciences, version 16 (SPSS). Data from FFQ were entered in duplicate in Epidata software, version 3.1 (Epidata Association) and subsequently transported to SPSS for analysis. Data were considered to be significant at $P<.05$.

RESULTS

One hundred five women of childbearing age (23.7 ± 6.3 years) were studied. Most were white (87.6%). The remaining subjects were of mixed African and European ancestry. The prevalence of obesity was 44.3% in PCOS women and 31.8% in control women ($P=.288$).

Overall PCOS and Control Groups

The clinical profile and metabolic and hormone variables of the PCOS and control groups are summarized in Table 1. The groups did not differ in age, BMI, waist circumference, and glucose. Pedometer-measured physical activity and rest metabolic rate also were similar between the groups. PCOS women had higher body fat percentage, fasting insulin, HOMA-IR, and LAP compared with control women. As expected, PCOS patients also presented higher androgen and lower SHBG levels.

Table 2 presents dietary GI and GL and calorie, macronutrient, and micronutrient intake in the PCOS and control groups. PCOS women had higher daily calorie intake and higher GI and GL. However, after adjustment for age and BMI, statistical significance was lost for GI and GL. The calorie

TABLE 1

Clinical characteristics and anthropometric and metabolic variables in PCOS patients and control subjects.

Variable	PCOS (n = 61)	Control (n = 44)	*P* value
Age	22.7 ± 6.2	25.0 ± 6.3	.070
BMI (kg/m^2)	28.9 ± 5.6	27.1 ± 5.7	.099
Waist circumference (cm)	85.4 ± 12.8	83.5 ± 12.7	.464
Total body fat (%)	38.9 ± 7.4	34.7 ± 9.5	.020
Steps/d	5,519 (3,658–7,002)	5,811 (4,339–7,267)	.702
Resting metabolic rate	1,469 ± 227	1,453 ± 249	.763
Glucose 0′ (mg/dL)	86.8 ± 9.1	87.0 ± 7.5	.902
Insulin (μU/mL)	16.7 (9.8–21.2)	9.9 (6.8–12.5)	.002
HOMA-IR	3.5 (2.1–4.7)	2.1 (1.4–2.8)	.006
LAP	25.3 (16.0–54.6)	20.1 (10.3–31.7)	.039
TT (ng/mL)	0.72 ± 0.25	0.54 ± 0.17	.001
Free androgen index	15.1 (10.7–27.4)	7.2 (4.4–11.9)	.001
SHBG (nmol/L)	27.1 (16.6–36.4)	40.2 (29.5–62.9)	.001

Note: Values are expressed as mean ± SD (Student *t* test) or median (interquartile range) (Mann-Whitney test). BMI = body mass index; HOMA-IR = homeostasis model assessment of insulin resistance; LAP = lipid accumulation product; PCOS = polycystic ovary syndrome; TT = total testosterone.

Graff. Dietary glycemic index in PCOS phenotypes. Fertil Steril 2013.

consumption of PCOS women was greater than that of the control group regardless of age and BMI. Fiber, macronutrient, and micronutrient intake did not differ between the groups.

GI was correlated with BMI in the control group (R = 0.310, 95% confidence interval [CI] 0.062–0.558; P=.016) but not in the PCOS group (R = 0.178, 95% CI −0.078–0.434; P=.171). GI was also correlated with LAP in the PCOS group (R = 0.404, 95% CI 0.119–0.689; P=.007), but not in the control group (R = 0.088, 95% CI −0.222–0.398; P=.589).

Stratification of PCOS and Control Groups by Tertiles of GI

Analysis of the PCOS and control groups by tertiles of dietary GI revealed that those with higher GI (third tertile) had higher BMI and waist circumference compared with the group with lower GI (first tertile; Table 3). Considering only the PCOS group, patients in the third tertile showed higher values of LAP and lower fiber intake compared with patients in the first tertile, who consumed low-GI diets.

Percentage of body fat, HOMA-IR, energy and carbohydrate intake, and resting metabolic rate were similar between tertiles of GI in both PCOS and control groups. In the PCOS group, fiber intake was lower in the third tertile compared with the first tertile.

Classic PCOS Patients, Ovulatory PCOS Patients, and Control Subjects

Table 4A presents the comparison between patients with classic PCOS, patients with ovulatory PCOS, and control

TABLE 2

Dietary glycemic index and load and energy, macro- and micronutrient intake in PCOS patients and control subjects.

Variable	PCOS (n = 61)	Control (n = 44)	*P* value[a]	*P* value[b] adjusted for age and BMI
Glycemic index	57.7 ± 5.3	55.7 ± 4.7	.047	.127
Glycemic load	176.3 (111.4–269.8)	143.8 (111.1–186.3)	.049	.106
Energy intake (kcal/d)	2,250 (1,710–3,786)	1,984 (1,620–2,335)	.034	.024
Carbohydrate (%)	52.5 ± 8.2	53.4 ± 7.4	.591	.648
Fat (%)	24.8 ± 6.1	25.5 ± 5.5	.649	.948
Protein (%)	15.5 ± 4.1	15.9 ± 3.7	.511	.568
Fiber (g/d)	24.3 (17.4–35.5)	21.3 (17.4–29.5)	.336	.435
Cholesterol (mg/d)	222 (176–348)	221 (176–286)	.585	.509
Saturated fatty acids (%)	32.5 ± 5.9	31.5 ± 6.6	.455	.616
Monounsaturated fatty acids (%)	30.2 ± 4.7	31.0 ± 5.0	.437	.308
Polyunsaturated fatty acids (%)	12.9 ± 3.4	13.7 ± 3.3	.251	.172
Calcium (mg/d)	731 (524–1,063)	648 (520–864)	.363	.544
Magnesium (mg/d)	256 (182–354)	227 (181–292)	.308	.347
Iron (mg/d)	9.6 (6.9–14.0)	8.7 (7.0–11.0)	.104	.124
Zinc (mg/d)	10.7 (7.4–14.3)	8.8 (7.1–11.9)	.088	.145
Folate (μg/d)	501 (322–654)	429 (355–552)	.471	.539
Sodium (mg/d)	2,329 (1,680–3,556)	1,903 (1,570–2,406)	.040	.101

Note: Values are expressed as mean ± SD or median (interquartile range). Abbreviations as in Table 1.

[a] Student *t* test or Mann-Whitney test.

[b] Analysis of covariance.

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TABLE 3

Anthropometric, dietary, and metabolic variables in PCOS patients and control subjects stratified by tertiles of diet glycemic index.

	PCOS				Control			
Variable	1st tertile (n = 20)	2nd tertile (n = 20)	3rd tertile (n = 21)	*P* value	1st tertile (n = 15)	2nd tertile (n = 14)	3rd tertile (n = 15)	*P* value
BMI (kg/m^2)	26.2 ± 5.0[a]	28.1 ± 5.2[a,b]	31.5 ± 6.4[b]	.038	24.0 ± 4.5[a]	26.8 ± 5.9[a,b]	29.5 ± 4.6[b]	.019
WC (cm)	78.7 ± 10.4[a]	84.0 ± 12.5[a,b]	89.6 ± 14.7[b]	.041	76.4 ± 8.9[a]	82.8 ± 13.4[a,b]	86.5 ± 10.7[b]	.034
Total body fat (%)	37.2 ± 5.9	37.9 ± 8.9	40.3 ± 7.6	.563	32.4 ± 10.3	34.0 ± 8.3	39.6 ± 7.2	.150
Energy intake (kcal/d)	2,332 (1,483–3,974)	2,097 (1,728–3,823)	2,365 (1,745–3,452)	.987	1,953 (986–2,186)	2,013 (1,666–2,319)	2,036 (1,702–2,369)	.231
Carbohydrate (%)	50 ± 8	54 ± 10	54. ± 6	.151	51 ± 8	55 ± 7	55 ± 7	.310
Fiber (g/d)	37 (25–60)[a]	26 (20–35)[a,b]	18 (14–26)[b]	.001	22 (15–34)	22 (19– 31)	19 (14–25)	.315
RMR (kcal/d)	1,512 ± 240	1,439 ± 184	1,457 ± 259	.579	1,408 ± 219	1,394 ± 249	1,518 ± 249	.317
LAP	22.3 (9–36.8)[a]	23.6 (16–36.3)[a]	50.7 (20–77)[b]	.045	22.5 (10.4–32)	14.8 (8.3–26.4)	22.4 (14–42.3)	.243
HOMA-IR	3.5 (1.3–5.0)	3.2 (2.3–4.6)	3.7 (1.5–4.7)	.969	2.1 (1.3–3.5)	1.8 (1.5–2.5)	2.3 (1.4–3.4)	.909

Note: Values are expressed as mean ± SD or median (interquartile range). In PCOS patients: 1st tertile IG ≤55.27; 2nd tertile IG >55.27 and <59.90; 3rd tertile IG ≥59.90. In control subjects: 1st tertile IG ≤53.46; 2nd tertile IG >53.46 and <59.01; 3rd tertile IG ≥59.01. Values are expressed as mean ± SD or median (interquartile range) (two-way analysis of variance and Bonferroni test for multiple comparisons). No interaction was found between groups (PCOS vs. control) and tertiles of dietary GI. LAP = lipid accumulation product; RMR = resting metabolic rate; WC = waist circumference; other abbreviations as in Table 1.

[a,b] Different letters represent significant statistical difference among tertiles in the same group ($P<.05$).

Graff. Dietary glycemic index in PCOS phenotypes. Fertil Steril 2013.

subjects. Classic PCOS patients showed higher BMI compared with both ovulatory PCOS and control participants. When adjusted for age and BMI, waist circumference was similar among the groups. Dietary GI was higher in the classic PCOS group compared with other groups even after adjustment for BMI and age. The difference in dietary GL between groups was not significant. Energy consumption was higher in the classic PCOS group compared with the control group, but statistical significance was lost after adjustment for BMI and age. TT was higher in classic PCOS compared with the other groups and remained significant after adjustment for age and BMI. Ovulatory PCOS participants presented an intermediate metabolic and hormonal profile in relation to classic PCOS and control participants.

Macronutrient and micronutrient intake was similar in classic and ovulatory PCOS phenotypes and control subjects (data not shown). Table 4B presents multiple linear regression analysis assessing the effects of age, BMI, and presence of classic PCOS phenotype on GI. BMI and classic PCOS were independent predictors of dietary GI, explaining 28.3% of variance in dietary GI.

DISCUSSION

This study shows that the dietary habits of PCOS women are similar to those of non-PCOS women of the same age and BMI, except for the consumption of foods with higher GI. Although these results confirm the general notion that metabolic disturbances are not influenced by dietary preferences in PCOS (57–59), they indicate that consumption of carbohydrates with higher GI is associated with obesity and central adiposity, and that PCOS women with the more severe phenotype tend to eat more high-GI carbohydrate foods.

PCOS women also had higher daily calorie intake than control women. The fact that percentage of macronutrient intake did not differ between groups, and that GI takes into consideration the type of carbohydrate rather than the amount of carbohydrate ingested, indicates that PCOS patients in general may favor carbohydrates of lower quality. This result can be at least partially explained by a difference in dietary fiber intake according to tertiles of dietary GI in the PCOS group. Other investigators (28, 60) have suggested that hunger and satiety might be impaired in PCOS. According to Farshchi et al. (61), many women with PCOS describe carbohydrate cravings and mention this as a cause of their difficulty in losing weight. This carbohydrate craving could explain a preference for a higher-GI diet, as observed in the classic, more severe, PCOS phenotype compared with ovulatory PCOS and control women, even in the presence of similar energy intake. Classic PCOS patients also had higher BMI. However, because of the study's cross-sectional design, the direction of the association between obesity and high dietary GI could not be determined. In contrast, multivariate analysis allowed us to confirm that both obesity and the classic PCOS phenotype are age-independent predictors of higher dietary GI.

Only a few studies have assessed dietary GI in women with PCOS. Barr et al. (29) found no differences in dietary

TABLE 4

A. Body mass index, dietary variables, and testosterone levels in women with classic or ovulatory PCOS and control women.

Variable	Classic PCOS (n = 39)	Ovulatory PCOS (n = 22)	Control (n = 44)	*P* value*	*P* value† adjusted for age and BMI
BMI (kg/m^2)	30.6 ± 5.6[a]	26.0 ± 4.3[b]	27.0 ± 5.6[b]	.002	—
Glycemic index	59.1 ± 5.6[a]	56.9 ± 5.0[b]	55.8 ± 4.7[b]	.044	.008
Glycemic load	176.3 (101.9–281.5)	173.5 (118.7–228.3)	143.8 (110.4–187.8)	.127	.245
Energy intake (kcal/d)	2,449 (1,681–3,872)[a]	2,141 (1,650–3,485)[a,b]	1,984 (1,620–2,335)[b]	.023	.078
TT (ng/mL)	0.77 ± 0.27[a]	0.62 ± 0.18[b]	0.54 ± 0.17[b]	.001	.001

B. Multiple linear regression analysis for independent effect of age, BMI, and presence of classic PCOS phenotype on glycemic index.

Glycemic index vs.	Coefficient (B) ± SE	*P* value	r^2
Age (y)	0.174 ± 0.107	.110	0.283
BMI (kg/m^2)	0.500 ± 0.130	.001	
Classic PCOS diagnosis (yes/no)	−4.657 ± 1.473	.003	

Note: Values are expressed as mean ± SD or median (interquartile range). Abbreviations as in Table 1.
* One-way analysis of variance.
† Analysis of covariance and Vonferroni multiple comparisons.
[a,b] Different letters represent significant statistical difference ($P<.05$).

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GI and GL between 38 PCOS and 28 control subjects, whereas Douglas et al. (62) found that the dietary pattern of PCOS patients was characterized by consumption of a greater amount of specific foods with high GI, such as white bread. Recently, Altieri et al. (30) showed that PCOS and control subjects did not differ regarding energy, macronutrient, and advanced glycosylated end-product intake, but PCOS individuals had higher consumption of high-GI foods.

The finding that the percentage of macronutrient intake did not differ between PCOS and control subjects is consistent with previous studies by our group (57) and others (30, 62–64). Barr et al. (29) found that the percentage energy from carbohydrate intake was significantly lower and the percentage energy from fat significantly higher in lean PCOS patients compared with lean control subjects, but similar between overweight/obese PCOS patients and control subjects. Furthermore, Wright et al. (63) found that although women with PCOS had higher BMI than control women, there was no difference in dietary macronutrient intake; however, lean women with PCOS reported significantly lower energy intake than lean women without PCOS. Tsai et al. (65) reported that Taiwanese women with PCOS consume lower energy and carbohydrate compared with those with non–PCOS-related infertility. These data should be interpreted with caution, because of different dietary habits as well as ethnic origins among different countries, which could account for the differences among the studies.

Both PCOS and control women consuming a diet in the highest tertile of GI had higher BMI and waist circumference, independently from total calorie intake and number of steps per day. Several authors suggest that regular consumption of carbohydrate-rich foods, which promote a high glycemic response, can increase the risk of obesity (18, 19, 66). A meta-analysis of clinical trials showed that low-GI and -GL diets reduce BMI and body fat mass compared with control diets (20).

In fact, evidence indicates that a diet with higher GI seems to produce a worse lipid profile and increased cardiovascular risk that are independent from total calorie intake and level of physical activity. Recently, Dong et al. (21) showed that diets with low GI and/or low GL exert a protective effect against cardiovascular disease in adult women. This association between high dietary GI and GL and cardiovascular disease is probably due to the adverse effects of these diets on serum lipids (20, 22, 24, 25), causing systemic inflammation (26). Moreover, it is argued that a lower-GI diet seems to reduce expression of genes related to insulin resistance (67).

The efficacy of low-GI diets in improving insulin resistance and glucose control has been demonstrated (31, 68). In addition, a meta-analysis (69) concluded that there is evidence to support positive associations between high-GI and -GL diets and risk of type 2 diabetes. Mehrabani et al. (32) found that the combination of high-protein and low-GL foods in a modified diet for PCOS women caused a significant increase in insulin sensitivity compared with a conventional diet.

Testosterone stimulates appetite (70), and high androgen levels in women could be associated with appetite dysregulation, together with a craving for carbohydrates. The higher testosterone levels of patients with classic PCOS may be one reason why these women have a preference for higher-GI carbohydrates.

Hyperandrogenism seems to be essential for determining the cardiometabolic risk in the various PCOS phenotypes (35). A previous study by our group also indicated that androgen levels were 3 times higher in women with the classic anovulatory phenotype, who also had increased prevalence of impaired glucose tolerance and IR, compared with ovulatory women with PCO morphology (11). Furthermore, the clustering of more than one cardiovascular risk factor appeared to be higher in the classic PCOS phenotype, coexisting with higher androgen levels (71, 72). In this sense, the present finding of a correlation of GI with IR in PCOS but not in control subjects supports the notion that hyperandrogenism could play a role on this association.

Strengths of the present study include the absence of earlier analyses of dietary GI and GL in women with different

phenotypes of PCOS and the use of a robust validated FFQ that assesses 121 items of food consumption during the preceding month. A limitation is the relatively small sample size, which did not allow a comparative analysis of obese versus normal-weight subgroups of PCOS and control women.

In conclusion, the present results indicate that women with the classic PCOS phenotype present higher dietary GI than ovulatory PCOS and control women, as well as a worse anthropometric and metabolic profile. Further studies are needed to clarify the mechanisms underlying the association between these abnormalities, high dietary GI, and hyperandrogenism in PCOS.

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