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;■:■–■. ©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 $\sim$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] $\times$ 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