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Dietary glycemic index/glycemic load and their relationship with inflammatory markers in women with polycystic ovary syndrome

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Summary *Objective:* the present study evaluates energy intake, macronutrient composition and dietary glycemic index (GI) and glycemic load (GL) in women with polycystic ovary syndrome (PCOS) and age and body mass index (BMI)- matched controls, and relationship between carbohydrate variables with inflammatory markers. *Methods:* This case_control study was conducted on 90 women aged 18-35 years including 45 women with PCOS and 45 age and BMI-matched healthy controls. Dietary intakes, GI and GL were assessed using three 24-hour recalls. Biochemical profile including high-sensivity C-reactive protein (hs-CRP), interleu- kin-6 (IL-6) and tumor necrosis factor α (TNF-α) as inflammatory markers and also metabolic variables related to insulin resistance were measured. Anthropometric measurements were performed in all subjects. *Results:* Dietary GI and starch intake were significantly higher in PCOS group compared with age and BMI-matched control group ($P < 0.05$). Fasting insulin, homeostatic model assessment of insulin resistance (HOMA-IR) and hs-CRP were higher in patients compared with controls ($P < 0.05$). No significant cor- relation was detected between biochemical variables and macronutrient intakes. *Conclusion:* Total energy and macronutrient intakes were similar between the groups except for dietary GI and starch intake. However, quality and quantity of carbohydrate consumed was not associated with insulin resistance and inflammatory markers in the PCOS group.

Keywords: Polycystic ovary syndrome, inflammation, glycemic index

Introduction

Polycystic ovary syndrome (PCOS) is the most common endocrinopathies among women of reproductive age and affects about 6-14% of them in developing countries (1, 2). PCOS is characterized by hyperandrogenism with some symptoms, such as hirsutism, acne and/or alopecia, chronic anovulation, menstural irregularities and reproductive abnormalities. Diagnosis is according to the presence of two out of the three features: oligoovulation and/or anovulation, hyperandrogenism (clinical and/or biochemical) and polycystic ovaries on ultrasound test, according to Rotterdam ESHRE/ASRM- sponsored PCOS consensus workshop (3, 4). Insulin resistance (IR) is an important metabolic complication of PCOS (5) which increases the risk of type 2 diabetes mellitus (T2DM), hypertension, dyslipidemia and cardiovascular disease (6). PCOS is also a proinflammatory state (7). Increased levels of high-sensivity C-reactive protein (hs-CRP), interleukin-6 (IL-6) and tumor necrosis factor α (TNF-α) show low-grade chronic inflammation associated with IR, which are risk factors of cardiovascular disease (8). Studies show that some of the inflammatory markers are elevated in PCOS (7-10) but the findings are inconsistent. A meta-analysis (7) has shown that women with PCOS show higher levels of circulating CRP that is independent of obesity. Etiology of PCOS is not known well, but

both genetic and environmental factors, such as nutrition and lifestyle play a role in pathogenesis (4, 11). It has been proposed that long term consumption of high-glycemic index (GI) foods create a high glycemic response and increase the insulin secretion. This chronic hyperinsulinemia can develop IR (12). IR plays an important role in the pathogenesis of PCOS and is an important marker of cardiovascular disease risk factors (13). The glycemic index is a method of ranking the carbohydrate-containing foods according to their effects on postprandial glycemic response (14). Not only the quality, but also the quantity of carbohydrate influences the glycemic response. So the concept of glycemic load (GL) is used also. This is the product of the amount of available carbohydrate and the GI of the food (15). Several studies have investigated the dietary GI/GL and intake of foods with high-GI in women with PCOS and controls. Some researchers found that women with PCOS consumed greater amount of specific foods with a high glycemic index, compared with matched control women (5, 16). In 2010, Barr et al. (17) showed that there were no significant differences in dietary GI and GL between women with PCOS or controls. Although Graff et al.(12) showed that women with PCOS had higher dietary GI/GL but statistical significance was lost after adjustment for age and BMI. Studies show that glucose can stimulate an inflammatory reaction in the mononuclear cells (MNCs) of women with PCOS (7). The possible correlation of GI/GL with indices of inflammation has been surveyed in diseases and healthy individuals. In PCOS, Mehrabani et al.(18) showed that women who assigned to a high-protein, low-GL hypocaloric diet, had lower levels of hs-CRP compared to women who assigned to a conventional hypocaloric diet after 12 weeks. Adverse effects of diets with high GI/GL on inflammatory factors that has been shown in several studies (19-22) can be an explanation for the association between such diets and cardiovascular disease. With this background, we hypothesized that quality and quantity of carbohydrate are associated with emerging cardiovascular disease risk factors in this syndrome. The possible correlation of GI/GL and inflammatory markers in PCOS has not been surveyed yet , so to answer the question of whether there is an association between dietary carbohydrate indexes and inflammatory markers, we designed the current case-control study.

Materials and Methods

Subject recruitment

This case_control study conducted on 90 females including 45 women with PCOS and 45 healthy volunteer women (control group). Patients were recruited through the gynecology and infertility clinic of the Moheb Yas comprehensive Women hospital in Tehran. All women who participated in this study were in reproductive age (range 18-35 years) and written informed consent was obtained from all of them before initiating the study. Diagnosis of PCOS was according to the Rotterdam criteria (23), by the presence of at least two of the three features of diagnosis: amenorrhea (no periods for > 6 mo) or oligomenorrhea (< 8 periods/y,cycle length > 45d or both), clinical (hirsutism, acne) or biochemical evidence for hyperandrogenemia (total testosterone >54 ng/dL or free testosterone >9.2 pg/mL), polycystic ovaries on ultrasound test (presence of >12 follicles in each ovary, with each follicle measuring 2–9 mm in diameter; increased ovarian volume > 10 mL; or a combination). Controls had regular menstural cycles, no signs of hirsutism and without endocrine disorders recruited through written advertisement. Groups were matched on age and BMI. Exclusion criteria included pregnancy, lactating, BMI > 40 kg/m², presence of thyroid disorders, hyperprolactinemia, cushing syndrome, congenital adrenal hyperplasia, cardiovascular diseases, type 1 or type 2 diabetes and also inflammatory diseases. Use oral contraceptives, insulin sensitizing, glucose lowering, glucocorticosteroids medications within 6 months from assessment. Other exclusion criteria consist of present adherence to a modified diet (eg. low energy or low fat or carbohydrate diet). The study protocol was approved by the Ethics of Tabriz University of Medical Sciences.

Antropometric assessments

Anthropometric measurements included body weight, height and waist circumference. For measurements, participants had minimal clothing, with their shoes removed. Weight was measured to the nearest 0.1 kg with a calibrated scale (SECA, Hamburg, Germany) and height was measured with non-stretchable

measurement tape with the precision of 0.1 cm. BMI was calculated as weight (kg) divided by the square of height (m). Waist circumference (WC) was measured at the midpoint between the inferior margin of the lower rib and the iliac crest.

Biochemical analysis

Venous blood samples were provided from all subjects after an overnight fasting . Blood samples were centrifuged at 3,000×g for 15 min and serum was extracted. All samples were stored at −70˚C until biochemical assays.

Analyses were performed in the Endocrinology and Metabolism Research Center-Cellular and Molecular Biology Lab of SBMU. Glucose levels were determined by colorimetric- enzymatic methods (Parsazmun Kit, Tehran, Iran). Insulin was assayed using Monobind, ELISA kit, USA. Assay sensivity was 0.75 μIU/ml. Serum IL-6 and TNF-α were measured by enzyme linked immunosorbent assay (ELISA kit, Diaclone, France) which had a sensitivity of 2 pg/mL for measurement of IL-6 and 8 pg/ml for measurement of TNF-α in human serum. Serum hs-CRP was measured by enzyme linked immunosorbent assay (ELISA kit, Diagnostics Biochem Canada Inc., Ontario, Canada) with a sensitivity of 10 ng/ml, respectively. IR was calculated by the homeostasis model assessment (HOMA) as follows: insulin (microunits/mL) multiplied by glucose (mg/dL) and dividing the product by 405(24).

Assessment of dietary intake

The dietary intake was assessed using three 24-hour recalls including (two week days and one weekend day) by a trained dietitian. Standard weights of food items, household measures and photos of proportions of usual weight were explained to the participants. After collecting the data of all the food and beverages consumed, data were then converted to grams. Energy, carbohydrate and fiber intake was obtained by Nutritionist IV software. To calculate GL and GI, we used the international tables of GI and GL values with glucose as the reference food (25). The average intake of foods containing carbohydrate was calculated. Dietary glycemic index was calculated by summing the products of the glycemic index for each food multiplied by the amount of available carbohydrate in the food portion and dividing by the total amount of available carbohydrate in the diet (26). Dietary glycemic load was calculated by multiplying the available grams of carbohydrates in the diet by the GI divided by 100 (27).

Statistical analysis

All analyses were carried out using the Statistical Package for the Social Sciences, version 16 (SPSS). A kolmogorov-Smirnov test was used to test normality of data distribution. Descriptive statistics are presented as mean (SD) or median (25^{th}- 75^{th} percentiles). Independent sample T-test and Mann–Whitney U-test were used to compare means and median values between two groups. Pearson correlation or Spearman rank correlation tests were used to identify associations. Partial correlation analysis was performed to test the association between parameters with adjustment for age and BMI as confounding variables. Statistical significance was considered at P <0.05.

Results

Anthropometric and biochemical variables are shown in Table 1. No significant difference was observed in age, BMI or waist circumference between participants. PCOS group showed significantly higher levels of fasting insulin, hs-CRP and HOMA-IR (P<0.05) respectively, and their fasting glucose, IL-6 and TNF-α levels were higher compared to controls, but the difference was not significant. HOMA-IR increased with BMI (r = 0.34, P < 0.05 in control women; r = 0.58, P < 0.01 in PCOS women) and WC (r = 0.43, P < 0.01 in control women; r = 0.61, P <0.01 in PCOS women). HOMA-IR was also significantly and positively associated with IL-6 (r = 0.43, P < 0.01) and hs-CRP (r = 0.52, P < 0.01) in PCOS group but not in control group.

Table 2. presents dietary intakes in the PCOS and control group. Total energy, carbohydrate, GL, dietary fiber, sugar, protein and fat intake did not differ between the groups. Dietary GI and starch levels were

Table 1. Descriptive characteristics and metabolic and inflammatory variables of cases with PCOS and controls

Variable	PCOS (n=45)	Control (n=45)	*P* value
Age	26.64 (4.63)	27.56 (4.61)	0.353
Weight (kg)	69.28 (13.01)	68.15 (13.21)	0.68
BMI (kg/m²)	26.37 (5.22)	25.29 (5.15)	0.327
WC (cm)	83.93 (14.6)	81.53 (8.46)	0.343
Fasting glucose(mg/dL)	109.02 (15.92)	104.84 (9.54)	0.136
Fasting insulin(μIU/ml)	13.56 (12.39)	8.06 (4.61)	**0.007**
HOMA-IR	2.37 (1.28-4.34)	1.91 (1.20-2.81)	**0.037**
hs-CRP (ng/ml)	4452 (2367.5-4243	20151 (1072-4243)	**0.001**
IL-6 (pg/ml)	4.03 (3.42-4.71)	3.72 (3.36-4.91)	0.449
TNF-α(pg/ml)	20.30 (15.60-29.55)	17.30 (14.90-22.20)	0.135

BMI, body mass index; WC, waist circumference; HOMA-IR, homeostasis model assessment-insulin resistance; hs-CRP, high sensivity C-reactive protein; IL-6, interleukin-6; TNF-α, tumor necrosis factor α. P value based on Independent sample T-test or Mann–Whitney U test. Data are presented as mean (SD) or medians (25th percentile –75th percentile

Table 2. dietary factors of cases with PCOS and controls

Variable	PCOS (n=45)	Control (n=45)	*P* value
Total energy intake (kcal/d)	1919 (1655.5 to 2140)	1880 (1621.15 to 2076.50)	0.476
Carbohydrate (g/d)	301.36 (98.25)	269.62 (57.75)	0.065
GI	66.23 (8.34)	62.59 (7.38)	**0.031**
GL	161.13 (64.59)	145.85 (37.63)	0.175
Dietary fiber (g/d)	14.06 (4.69)	13.52 (4.98)	0.596
Starch (g/d)	194.27 (85.22)	160.62 (48.16)	**0.023**
Sugar (g/d)	62.65 (30.41)	62.93 (23.59)	0.962
Protein (g/d)	65.52 (55.16 to 78.73)	63.19 (54.64 to 77.15)	0.663
Fat (g/d)	58.18 (45.95 to 70.87)	57.33 (48.74 to 65.41)	0.979

GI, glycemic index; GL, glycemic load. P value based on Independent sample T-test or Mann–Whitney U test. Data are presented as mean (SD) or medians (25th percentile –75th percentile

significantly higher among PCOS cases compared with controls (P<0.05). Correlation tests showed significant positive association between carbohydrate and starch intake and dietary GL with HOMA-IR in PCOS group (P < 0.01 for starch and GL, P <0.05 for carbohydrate). Significant positive relation was also identified between starch intake with hs-CRP in case group (P < 0.05). An inverse relation was observed between starch intake and dietary GL with TNF-a within PCOS group (r = -0.373, p < 0.05). However, after adjustment for age and BMI, all the significant correlations were lost (table 3).

Discussion

This case-control study demonstrates that energy and macronutrient intake of women with PCOS is similar to the age and BMI-matched healthy women except for starch intake and glycemic index of the diet.

Previous studies also posed no difference in energy and macronutrient intakes between women with and without PCOS (5, 16, 28, 29). Larsson et al. (2) reported that PCOS women with higher BMI than controls, consumed higher carbohydrate, but energy intake was the same. In our study, PCOS women consumed more carbohydrate than control women and their dietary GL was higher than control group, although the difference was not significant, higher dietary GI of PCOS patients shows that they may prefer carbohydrates of lower quality. Few studies have evaluated dietary GI and GL in women with PCOS. Graff et al. (12) showed that PCOS women had higher dietary GI/GL compared with controls, although after adjustment for age and BMI, statistical significance was lost. Barr et al. (17) showed that there were no significant differences in dietary GI and GL between women with PCOS or controls. No previous study had investigated starch intake in PCOS, however Altierie et al. (16) reported that PCOS subjects consumed more starchy sweets and had a tendency to consume higher amount of starchy foods with high GI. It seems that these data should be interpreted with caution, because of different dietary patterns among different countries and diverse methods of dietary assessments. PCOS patients are at high risk for diabetes and cardiovascular disease. Similar to previous studies (1, 30-32), our survey showed that patients with PCOS had higher levels of fasting insulin, insulin resistance and hs-CRP than age and BMI matched control women. IR was positively associated with BMI for both groups. It was also positively associated with IL-6 and hs-CRP only in PCOS group, which may show a pathophysiologic link between IR and inflammation in PCOS.

The main objective of this study was to define whether the quality and quantity of carbohydrate intake was associated with insulin resistance and inflammatory markers that has been recognized as car-

Table 3. Correlation analyses between carbohydrate variables and IR and inflammatory markers before and after adjusting for age and BMI in PCOS patients

	Before adjustment*				after adjustment*			
	IR	**HS-CRP**	**IL-6**	**TNF-a**	**IR**	**HS-CRP**	**IL-6**	**TNF-a**
GI								
	r = 0.186	*r* = 0.018	*r* = -0.014	*r* = -0.284	*r* = 0.230	*r* = 0.022	*r* = -0.001	*r* = -0.217
	P = 0.221	*P* = 0.908	*P* = 0.929	*P* = 0.058	*P* = 0.139	*P* = 0.888	*P* = 0.992	*P* = 0.162
GL								
	r = 0.388	*r* = 0.242	*r* = 0.201	*r* = -0.321	*r* = 0.097	*r* = 0.158	*r* = -0.038	*r* = -0.246
	P = **0.009**	*P* = 0.109	*P* = 0.185	*P* = **0.031**	*P* = 0.535	*P* = 0.313	*P* = 0.810	*P* = 0.112
Total carbohydrates(g/day)								
	r = 0.364	*r* = 0.183	*r* = 0.140	*r* = -0.200	*r* = 0.050	*r* = 0.173	*r* = -0.070	*r* = -0.191
	P = **0.014**	*P* = 0.228	*P* = 0.361	*P* = 0.189	*P* = 0.748	*P* = 0.266	*P* = 0.656	*P* = 0.220
Starch (g/day)								
	r = 0.406	*r* = 0.304	*r* = 0.187	*r* = -0.373	*r* = 0.047	*r* = 0.184	*r* = -0.073	*r* = -0.256
	P = **0.006**	*P* = **0.042**	*P* = 0.218	*P* = **0.012**	*P* = 0.763	*P* = 0.238	*P* = 0.642	*P* = 0.098
Sugar (g/day)								
	r = 0.000	*r* = -0.038	*r* = -0.114	*r* = 0.159	*r* = -0.022	*r* = -0.067	*r* = -0.127	*r* = 0.019
	P = 0.995	*P* = 0.806	*P* = 0.455	*P* = 0.297	*P* = 0.887	*P* = 0.671	*P* = 0.418	*P* = 0.901
Dietary fiber (g/day)								
	r = 0.061	*r* = -0.043	*r* = 0.061	*r* = -0.114	*r* = -0.020	*r* = -0.047	*r* = 0.001	*r* = 0.010
	P = 0.692	*P* = 0.780	*P* = 0.691	*P* = 0.457	*P* = 0.997	*P* = 0.766	*P* = 0.994	*P* = 0.949

*GI, glycemic index; GL, glycemic load. . Bold values indicate significant results.*Variables were adjusted for age and BMI*

diovascular risk factors in PCOS. We found that total carbohydrate, GL and starch significantly correlated with IR but the significance was lost after adjustment for age and BMI. Our results contradicts the recent study (12) which showed a correlation of GI with IR in PCOS. No association were found between quality and quantity of carbohydrate consumed and inflammation status after adjustment for age and BMI. However these results are relatively new and should be interpreted with caution, but it seems that quality of carbohydrates may not influence inflammatory parameters in PCOS. The strength of our study is lack of earlier investigation of association between dietary GI/GL and inflammation in PCOS. Weaknesses are the relatively small sample size and also lack of analyzing the other inflammatory factors which may have relationships with dietary factors in PCOS.

In conclusion, our results represent that quality of dietary carbohydrate is different between PCOS and control group. Women with PCOS had higher dietary glycemic index and starch intake, as well as higher levels of fasting insulin, HOMA-IR and hs-CRP concentrations, than the control group. However no significant association were found between GI/GL and IR and inflammation in PCOS. Further interventional studies are needed to define the effect of GI/GL of the diet on cardiovascular risk factors in PCOS.

References

1. Pedroso DCC, Miranda-Furtado CL, Kogure GS, et al. Inflammatory biomarkers and telomere length in women with polycystic ovary syndrome. Fertil Steril 2015;103:542-7.
2. Larsson I, Hulthén L, Landén M, Pålsson E, Janson P, Stener-Victorin E. Dietary intake, resting energy expenditure, and eating behavior in women with and without polycystic ovary syndrome. Clin Nutr 2016;35:213-8
3. Rondanelli M, Perna S, Faliva M, Monteferrario F, Repaci E, Allieri F. Focus on metabolic and nutritional correlates of polycystic ovary syndrome and update on nutritional management of these critical phenomena. Arch gynecol obstet 2014;290:1079-92.
4. Balen A. The pathophysiology of polycystic ovary syndrome: trying to understand PCOS and its endocrinology. Best Pract Res Clin Obstet Gynaecol 2004;18:685-706.
5. Douglas CC, Norris LE, Oster RA, Darnell BE, Azziz R, Gower BA. Difference in dietary intake between women with polycystic ovary syndrome and healthy controls. Fertil steril 2006;86:411-7.
6. Reaven GM. Pathophysiology of insulin resistance in human disease. Physiol rev 1995;75:473-86.
7. Escobar-Morreale HF, Luque-Ramírez M, González F. Circulating inflammatory markers in polycystic ovary syndrome: a systematic review and metaanalysis. Fertil steril 2011;95:1048-58.
8. Samy N, Hashim M, Sayed M, Said M. Clinical significance of inflammatory markers in polycystic ovary syndrome: their relationship to insulin resistance and body mass index. Dis markers 2009;26:163-70.
9. Vgontzas AN, Trakada G, Bixler EO, et al. Plasma interleukin 6 levels are elevated in polycystic ovary syndrome independently of obesity or sleep apnea. Metabolism 2006;55:1076-82.
10. Gonzalez F, Thusu K, Abdel-Rahman E, Prabhala A, Tomani M, Dandona P. Elevated serum levels of tumor necrosis factor alpha in normal-weight women with polycystic ovary syndrome. Metabolism 1999;48:437-41.
11. Dasgupta S, Reddy BM. Present status of understanding on the genetic etiology of polycystic ovary syndrome. J postgrad med 2008;54:115-25
12. Graff SK, Mário FM, Alves BC, Spritzer PM. Dietary glycemic index is associated with less favorable anthropometric and metabolic profiles in polycystic ovary syndrome women with different phenotypes. Fertil steril 2013;100:1081-8.
13. Souza dos Santos AC, Soares NP, Costa EC, de Sá JCF, Azevedo GD, Lemos TMAM. The impact of body mass on inflammatory markers and insulin resistance in polycystic ovary syndrome. Gynecol Endocrinol 2015;31:225-8.
14. Silvera SA, Jain M, Howe GR, Miller AB, Rohan TE. Glycaemic index, glycaemic load and ovarian cancer risk: a prospective cohort study. Public health nutr 2007;10:1076-81.
15. Monro JA, Shaw M. Glycemic impact, glycemic glucose equivalents, glycemic index, and glycemic load: definitions, distinctions, and implications. Am J clin nutr 2008;87:237-43.
16. Altieri P, Cavazza C, Pasqui F, Morselli AM, Gambineri A, Pasquali R. Dietary habits and their relationship with hormones and metabolism in overweight and obese women with polycystic ovary syndrome. Clin endocrinol 2013;78:52-9.
17. Barr S, Hart K, Reeves S, Sharp K, Jeanes Y. Dietary glycaemic index, glycaemic load and insulin resistance in lean and overweight women with polycystic ovary syndrome and controls. Proc Nutr Soc 2010;69:126.
18. Mehrabani HH, Salehpour S, Amiri Z, Farahani SJ, Meyer BJ, Tahbaz F. Beneficial effects of a high-protein, low-glycemic-load hypocaloric diet in overweight and obese women with polycystic ovary syndrome: a randomized controlled intervention study. J Am Coll Nutr 2012;31:117-25.
19. Bulló M, Casas R, Portillo M, et al. Dietary glycemic index/load and peripheral adipokines and inflammatory markers in elderly subjects at high cardiovascular risk. Nutr Metab Cardiovasc Dis 2013;23:443-50.
20. Buyken AE, Flood V, Empson M, et al. Carbohydrate nu-

trition and inflammatory disease mortality in older adults. Am J clin nutr 2010;92:634-43.
21. Gögebakan Ö, Kohl A, Osterhoff MA, et al. Effects of weight loss and long-term weight maintenance with diets varying in protein and glycemic index on cardiovascular risk factors the diet, obesity, and genes (DiOGenes) study: a randomized, controlled trial. Circulation 2011;124:2829-38.
22. Heggen E, Klemsdal TO, Haugen F, Holme I, Tonstad S. Effect of a low-fat versus a low-gycemic-load diet on inflammatory biomarker and adipokine concentrations. Metab syndr relat disord 2012;10:437-42.
23. ESHRE TR, Group A-SPCW. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertil steril. 2004;81:19-25.
24. Bonora E, Targher G, Alberiche M, et al. Homeostasis model assessment closely mirrors the glucose clamp technique in the assessment of insulin sensitivity: studies in subjects with various degrees of glucose tolerance and insulin sensitivity. Diabetes care 2000;23:57-63.
25. Foster-Powell K, Holt SH, Brand-Miller JC. International table of glycemic index and glycemic load values: 2002. The Am J clin nutr 2002;76:5-56.
26. Keim N. Carbohydrate. In: Nancy LK, Roy JL, Peter JH. Modern Nutrition in Health and Disease,11th ed. Lippincott Williams and Wilkins, Philadelphia,2012; PP: 52.
27. Limkunakul C, Sundell MB, Pouliot B, Graves AJ, Shintani A, Ikizler TA. Glycemic load is associated with oxidative stress among prevalent maintenance hemodialysis patients. Nephrol Dial Transplant 2013:29:1047-53.
28. Wright C, Zborowski J, Talbott E, McHugh-Pemu K, Youk A. Dietary intake, physical activity, and obesity in women with polycystic ovary syndrome. Int J obes 2004;28:1026-32.
29. Toscani MK, Mario FM, Radavelli-Bagatini S, Spritzer PM. Insulin resistance is not strictly associated with energy intake or dietary macronutrient composition in women with polycystic ovary syndrome. Nutr Res 2011;31:97-103.
30. Mohlig M, Spranger J, Osterhoff M, et al. The polycystic ovary syndrome per se is not associated with increased chronic inflammation. Eur J Endocrinol 2004;150:525-32.
31. Boulman N, Levy Y, Leiba R, et al. Increased C-reactive protein levels in the polycystic ovary syndrome: a marker of cardiovascular disease. J Clin Endocrinol Metab 2004;89:2160-5.
32. Tarkun I, Arslan BnÇ, Canturk Z, Turemen E, ahi˙n T, Duman C. Endothelial dysfunction in young women with polycystic ovary syndrome: relationship with insulin resistance and low-grade chronic inflammation. J Clin Endocrinol Metab 2004;89:5592-6.

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