Clinical Nutrition 38 (2019) 2342–2348

Contents lists available at ScienceDirect

Clinical Nutrition

journal homepage: http://www.elsevier.com/locate/clnu

Original article

Dietary intake, body composition and metabolic parameters in women with polycystic ovary syndrome

Nayara Bernardes da Cunha [a], Camila Toffoli Ribeiro [b], Catarina Mendes Silva [a], Ana Carolina Japur de Sá Rosa-e-Silva [c], Daurea Abadia De-Souza [d, *]

[a] Multidisciplinary Residency Program in Clinical Nutrition, Multidisciplinary Residency in Health, Faculty of Medicine, Federal University of Uberlândia, Av. Pará, 1720, Bloco 2H, Uberlândia, MG, CEP-38405-320, Brazil
[b] Department of Gynecology and Obstetrics, Faculty of Medicine, Federal University of Uberlândia, Av. Pará, 1720, Bloco 2H, Uberlândia, MG, CEP-38405-320, Brazil
[c] Department of Gynecology and Obstetrics, School of Medicine of Ribeirão Preto, University of São Paulo, Avenida Bandeirantes, 3900, Ribeirão Preto, SP, CEP 14049-900, Brazil
[d] Department of Internal Medicine, Faculty of Medicine, Federal University of Uberlândia, Av. Pará, 1720, Bloco 2H, Sala 1, Uberlândia, MG, CEP-38405-320, Brazil

ARTICLE INFO

Article history:
Received 23 February 2018
Accepted 18 October 2018

Keywords:
Polycystic ovary syndrome
Dietary pattern
Body composition
Dual-energy X-ray absorptiometry
Metabolic parameters

SUMMARY

Background & aims: Overweight polycystic ovary syndrome (PCOS) patients present exacerbation of clinical symptoms and increased risk for chronic diseases. The effects of inadequate dietary intake have been investigated in body weight gain in PCOS women. The aim of this study was to evaluate the dietary pattern and to analyze possible associations with the metabolism and body composition in PCOS women.
Methods: A case–control study was performed with thirty-nine women diagnosed with PCOS and thirty-five control women, matched by age and body mass index. A body composition assessment was performed by Dual-energy X-ray absorptiometry (DXA) and food intake was assessed using the seven-day food record. The metabolic parameters evaluated were fasting glucose, insulin, Homeostasis Model Assessment-estimated Insulin Resistance (HOMA-IR) index and oral glucose tolerance test (OGTT).
Results: No significant differences were observed in dietary intake of women with or without PCOS. In the analysis of the associations between dietary intake, metabolic parameters and body composition, PCOS women showed an inverse correlation between dietetic fiber intake and HOMA-IR index ($r = -0.365$; $p = 0.024$). Also in PCOS group, dietary fiber intake presented an inverse correlation with total body fat ($r = -0.401$; $p = 0.011$), trunk fat ($r = -0.388$; $p = 0.015$), and android fat ($r = -0.431$; $p = 0.006$). PCOS women group had higher glucose 120', compared to those without PCOS ($p = 0.015$).
Conclusion: These results provide evidence that the adequate intake of dietary fiber contributes to more appropriate body composition and glucose metabolism in PCOS women and possibly toward the prevention of chronic non-communicable diseases.

1. Introduction

Polycystic ovary syndrome (PCOS) is a common endocrine disease. PCOS affects 4–8% of women during reproductive age through the criteria put forward by the National Institutes of Health (NIH) or 14%–19% in line with the European Society of Human Reproduction and Embryology and the American Society for Reproductive Medicine (ESHRE/ASRM) [1–4]. Although the responsible causal factors for the development of PCOS are still not totally clear, some researchers have demonstrated the involvement of genetic and environmental factors [5–8].

PCOS is essentially characterized by the presence of physiopathological alterations typical of metabolic syndrome, with emphasis placed on insulin resistance and hyperinsulinemia [3,9]. The presence of these dysfunctions increases the risk of developing type 2 diabetes and cardiovascular diseases, among other diseases associated with metabolic alterations [10].

Specifically with regard to body composition, women with PCOS present a high overweight ratio. Although there is no consensus

* Corresponding author. Fax: +55 34 3225 8602.
E-mail addresses: NAYARABC8@hotmail.com (N.B. Cunha), camtoffoli@yahoo.com.br (C.T. Ribeiro), catarinamsilva@yahoo.com.br (C.M. Silva), anasars@fmrp.usp.br (A.C.J.S. Rosa-e-Silva), daureas@ufu.br (D.A. De-Souza).

https://doi.org/10.1016/j.clnu.2018.10.012
0261-5614/

reached in the literature [11,12], some investigators have demonstrated that patients with PCOS present an increase in central abdominal fat when compared to controls matched by body weight [13,14]. According to Carmina et al. [13], this difference was not observed when comparing obese PCOS and the obese control group, i.e., the increase of central abdominal fat is due to the differences found between the control group and the eutrophic or overweight PCOS patients [13]. Overweight PCOS patients, mainly those with central obesity, present less sensibility and elevated serum insulin levels, exacerbation of clinical symptomatology and a higher risk for chronic non-communicable diseases [14–16].

Inadequate food intake in qualitative and quantitative terms have been investigated as one of the casual factors responsible for the excess body weight in women with PCOS [17]. However, the evidences identified in the literature relating food consumption to PCOS are still contradictory [18]. In a study evaluating food intake by means of a semi-quantitative food intake questionnaire, it was identified that women with PCOS present a higher total energy and fat consumption [19]. In addition, other investigators has shown that PCOS women present higher intake of foods with a high glycemic index and lower fiber intake [20]. On the other hand, several researchers failed to demonstrate any difference between the dietary intake of women with or without PCOS [21–23].

The aims of the present study were to characterize the dietary pattern and analyze possible associations between the consumption of specific nutrients with metabolic parameters and body fat distribution in women with PCOS.

2. Participants and methods

2.1. Participants

This case–control study was performed at the Endocrine Gynecology Outpatient of the Clinical Hospital of Uberlandia, Federal University of Uberlandia, Brazil, between September 2015 to May 2017.

The study participants were recruited by means of pamphlets posted in strategic locations of the campus, email and verbal communication. Potential study participants were instructed to present their medical histories, as well as being submitted to a genealogical exam. In order to determine ovarian morphology, a two-dimensional pelvic or transvaginal ultrasound exam was performed.

Thirty-nine women were included in the study group, all ranging between 18 and 35 years of age, and all diagnosed with PCOS, as they presented two or more criteria from the Rotterdam consensus [24]: oligomenorrhea and/or anovulation; clinical signs and/or biochemical hyperandrogenism; and/or polycystic ovaries on an ultrasound exam. Ovaries were characterized as polycystic through an ultrasound exam, and present one of the following alterations: 12 or more follicles measuring between 2 and 9 mm in diameter or with increased ovary volume ($\geq$10 cm^3), on at least one of the ovaries [25]. The presence of oligomenorrhea and/or anovulation was identified by the occurrence of the menstrual cycle with a duration of >38 days [26]. Hyperandrogenism was identified through the presence of acne in the physical exam and/or hirsutism (score for Ferriman-Gallwey $\geq$ 8) [27].

Thirty-five women were included in the control group, with ages between 18 and 35, all presented regular and spontaneous menstrual cycles and who did not present any previous history of infertility. All the women included in the control group were matched by age and Body Mass Index (BMI) with women with PCOS. In the clinical practice, in order to perform the pairing, a difference of up to ±2.0 kg/m^2 on the BMI was used as the criterion, while observing that each control had the same BMI category as the recruited PCOS patient [28].

Immediately after the inclusion of the study participants, an anthropometric evaluation was performed and information was collected for the filling out of a semi-structured questionnaire. The collection of blood samples for performing biochemical exams (fasting glucose, basal insulin, and after glucose challenge test), and the body composition evaluation using the Dual-energy X-ray absorptiometry (DXA) were performed in specialized clinics at a later moment.

Those patients who had been taking any kind of hormonal medication over the last three months, or oral hypoglycemic agents, or medication for inducing weight loss; those diagnosed with other endocrine diseases (congenital adrenal hyperplasia, hypothyroidism or hyperthyroidism, hyperprolactinemia, Cushing's syndrome, androgen secreting tumors or other related disorders); pregnant women or breast feeding mothers, were excluded from the study.

All the participants of the study signed the term of free and informed consent. The study was conducted according to the guidelines established in the Declaration of Helsinki, after being approved by the Ethics Committee for Research on Human beings at the Federal University of Uberlandia.

2.2. Study protocol

A specific and semi-structured questionnaire was created with information pertinent to diet history, including preferences and food aversions, intolerances or food allergies, standards of chewing, habitual daily water intake and use of dietary supplements. In addition, were investigated the intestinal habits and the history of the evolution for their body weight. General and sociodemographic information, along with personal history and family history were also registered.

2.3. Anthropometric evaluation

Measurements for body weight, height, waist circumference (measured at the midpoint between the last costal arch and the iliac crest) and the circumference of the hip (measured along the largest diameter of the gluteal region), were taken. Following this, the BMI was calculated.

For body weight measurements, a digital scale was used (Welmy W300, precision of 0.1 kg). During the weighing process, the patient was barefoot, erect and stood in the center of the platform with her arms close to the body, eyes fixed at a point in front of her, and with the least amount carried objects as possible. The height measurement (precision of 0.5 cm) was taken on a vertical anthropometer coupled to the digital scale. The patient remained upright, barefoot, in the center of the platform with their back to the marker and heels together and their feet at an angle of 45°. The patient kept their arms down and their head back, looking at a fixed point at eye height. The BMI was calculated by the formula: body weight (kg)/height $(m)^2$ and was classified according to references from the World Health Organization [28]. The value of the waist circumference was obtained from the average of three measurements taken with an inextensible anthropometric tape (Sanny Medical, SN-4010, precision of 0.5 cm) at the midpoint point between the last costal arch and the iliac crest arch. The hip circumference (precision of 0.5 cm) was measured in the region localized between the waist and the thighs, at the point of largest diameter.

2.4. Evaluation of body composition

The evaluation body composition was performed using DXA of the whole body (Lunar Prodigy DXA System, software version 11.20). After the removal of all metallic objects, a radiologist

technician performed the exam. Measurements were made regarding the amount of fat body mass and lean body mass, along with the obtainment of information concerning the distribution of body fat in the android and trunk compartments. More specifically, the compartments relevant to the android fat (lower limit – horizontal line connecting the upper anterior superior iliac spines; upper limit – defined as 20% above the lower border; lateral boundaries – waist margins parallel to the chin line) were identified. In addition, the compartments relevant to the trunk fat (horizontal line below the chin and vertical lines passing through colli femori) were identified. The ratio between the fat mass and lean mass [fat mass (%)/lean mass (%)] was calculated.

2.5. Evaluation of food intake

The dietary pattern was determined by the seven-day food report [29]. The participants were guided to fill the form in the time and place of meals taken, reporting the quantity and the type of food or preparation and drinks ingested. At the moment of devolution of the food register, each described item was conferred with the patient. Among other dietary information, the food preparation and the size of the portions, as well as the foods effectively consumed in the meals were duly verified.

In accordance with the seven-day food report, the total energy ingested and the quantities consumed for carbohydrates, proteins, fat, saturated fatty acids, cholesterol and fiber were all identified. The amount of each nutrient was calculated in Excel worksheet using the Brazilian table of food composition (BTFC) [30]. When some component remained unidentified, the Food composition databases of the United States Department of Agriculture (USDA) was used [31].

2.6. Biochemical analyses

The blood samples used for performing the biochemical analyses were obtained after a 12-hour fasting. The serum levels of total cholesterol, HDL cholesterol, LDL cholesterol and triglycerides (enzymatic colorimetric method, Roche Diagnóstica©, Brazil) were evaluated. In addition, the simplified oral glucose tolerance test (OGTT) was performed, in two screenings, with the first in fasting and the second two hours after the ingestion of 75 g of glucose. The serum blood glucose levels (hexokinase method Roche Diagnóstica©, Brazil) and of basal insulin (chemiluminescence, Roche Diagnóstica©, Brazil) were evaluated. The Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) index was calculated using the formula: HOMA-IR = fasting glucose (mg/dL) x fasting insulin (μUI/mL)/405 [32].

2.7. Statistical analysis

The statistical analysis was performed using the software SPSS version 21.0 (Armonk, NY: IBM Corp.). The results were represented as mean ± standard deviation (SD) in the case of parametric data and as the median and interquartile range for non-parametric data. The Student t test was used for analyzing the anthropometric measurements (weight, height, BMI, waist and hip circumference) and the metabolic parameters (fasting glucose, basal insulin, HOMA-IR index and after glucose challenge test), and the body composition measurements (total fat, trunk fat, and android fat). The comparisons among the non-parametric results were carried out using the Mann–Whitney U test. Comparisons were made in order to identify if after the separation of the participants into specific subgroups, in accordance with the BMI classification proposed by WHO [28], were maintained the interpretations referring to the dietary parameters (energy intake, carbohydrate, protein, lipid, saturated fatty acids, cholesterol and fiber) and the distribution of body fat (total fat, trunk fat, and android fat), using the test of Kruskal–Wallis (1-*way* ANOVA). The Spearman coefficient was used to analyze the correlations between energy intake and nutrients with the metabolic parameters and the body composition of women with and without PCOS. By the identification of discrepant values in the checking of outliers, one control group participant was excluded. Statistical significance was considered as $p \leq 0.05$, through the adoption of the two-tailed test.

3. Results

Anthropometric and some metabolic parameters of the PCOS and their BMI and age-matched control group were presented on Table 1. No significant differences were observed in weight, BMI, waist, hip, fasting glucose, fasting insulin, HOMA-IR index between the PCOS and control groups. The PCOS group had higher glucose 120', when compared to those without PCOS ($p = 0.015$) (Table 1).

In relation to body composition, it was identified that total body fat, trunk fat, and android fat were similar in PCOS and control groups (Table 1). In the comparisons performed between the participants included in the PCOS subgroups eutrophic (n = 20) and eutrophic control (n = 19) (BMI = 18.5–24.9 kg/m^2); and overweight PCOS (n = 19) and overweight control (n = 15) (BMI ≥ 25.0 kg/m^2), it was demonstrated that total fat ($p < 0.001$), trunk fat ($p < 0.001$), and android fat ($p < 0.001$) were higher in overweight women than eutrophic women, with ($p < 0.001$) or without ($p < 0.001$) PCOS.

The dietary pattern of eutrophic and overweight women with or without PCOS was presented on Table 2. No significant differences

Table 1
Anthropometric, metabolic and body composition features of the study participants.

Variable	PCOS (n = 39)	Controls (n = 34)*	*P value*
Age (y)	25.17 ± 3.86	25.67 ± 4.42	0.610
Weight (kg)	65.40 (53.50–86.00)	62.75 (54.11–75.33)	0.821
BMI (kg/m^2)	24.43 (20.90–33.84)	23.95 (21.62–31.01)	0.730
Waist (cm)	83.50 (72.00–103.25)	81.00 (72.00–93.87)	0.436
Fasting glucose (mg/dL)	86.66 ± 8.46	85.27 ± 7.50	0.471
Fasting insulin (μUI/mL)	9.70 (6.90–18.50)	9.00 (5.80–13.35)	0.109
HOMA-IR	2.05 (1.38–4.04)	1.93 (1.14–2.77)	0.131
Glucose 120′ (mg/dL)	103.86 ± 23.61	91.24 ± 16.47	0.015
Total body fat (%)	40.61 ± 9.68	41.60 ± 9.94	0.670
Trunk fat (%)	42.10 ± 11.09	41.65 ± 10.33	0.857
Android fat (%)	45.10 ± 12.44	44.29 ± 11.45	0.776

Data were represented as means ± standard deviation (SD) or median and interquartile range. Abbreviations: BMI: Body Mass Index; HOMA-IR: Homeostasis Model Assessment-estimated Insulin Resistance. Statistical comparisons were performed by Student *t* test for means and by Mann–Whitney *U* test for medians (*P* value ≤ 0.05). *By the identification of discrepant values in the checking of outliers, one control group participant was excluded.

Table 2
Dietary pattern of women with and without PCOS.

Variable	PCOS (n = 39)				Controls (n = 34)*				*P value*
	Total	Eutrophy (n = 20)	Overweight (n = 19)	*P value*	Total	Eutrophy (n = 19)	Overweight (n = 15)*	*P value*	
Energy (kcal)	1651.42 (1184.19–1949.22)	1683.64 (1415.49–2156.04)	1479.12 (1030.53–1922.42)	0.576	1487.88 (1240.79–1903.91)	1704.98 (1120.49–2120.01)	1372.38 (1258.75–1665.95)	0.452	0.748
Energy (kcal/kg)	23.56 (17.34–31.96)	29.00 (24.17–43.34)	19.01 (13.27–22.61)	0.001	22.56 (17.88–33.82)	31.42 (22.36–42.48)	19.10 (17.29–22.17)	0.009	0.825
Carbohydrate (g)	203.47 (122.24–241.27)	203.47 (155.30–298.17)	190.35 (106.16–231.40)	0.644	182.90 (134.62–235.86)	190.76 (133.90–223.34)	162.73 (134.87–254.01)	0.584	0.479
Carbohydrate (g/kg)	2.85 (2.05–4.40)	3.59 (2.83–5.18)	2.44 (1.28–2.90)	0.012	2.87 (1.81–3.83)	3.32 (2.13–4.00)	2.46 (1.30–2.93)	0.098	0.642
Carbohydrate (%)	49.22 (41.53–55.50)	49.93 (43.70–57.43)	48.14 (37.47–51.04)	0.247	46.83 (38.75–50.79)	46.37 (37.61–49.87)	49.01 (38.75–52.91)	0.288	0.320
Protein (g)	75.35 (60.46–99.74)	78.09 (57.61–104.44)	67.47 (60.46–90.37)	0.608	69.61 (54.08–87.83)	74.80 (61.86–112.85)	57.75 (50.73–83.05)	0.498	0.558
Protein (g/kg)	1.08 (0.75–1.78)	1.53 (1.02–2.00)	0.79 (0.69–1.22)	0.031	0.97 (0.77–1.39)	1.27 (0.95–2.29)	0.77 (0.72–0.95)	0.003	0.715
Protein (%)	18.43 (14.74–24.47)	17.54 (14.86–24.02)	20.52 (14.65–27.37)	0.539	17.33 (14.90–22.88)	18.98 (14.95–23.15)	16.75 (14.21–19.53)	0.669	0.682
Fat (g)	57.08 (36.18–70.32)	57.11 (41.16–72.48)	50.54 (33.54–70.32)	0.563	55.54 (44.28–84.45)	67.49 (43.38–87.78)	52.05 (44.53–82.50)	0.579	0.301
Fat (g/kg)	0.85 (0.51–1.12)	0.93 (0.81–1.46)	0.70 (0.42–0.89)	0.047	0.86 (0.61–1.38)	1.25 (0.75–1.67)	0.70 (0.41–0.89)	0.053	0.603
Fat (%)	31.61 (24.83–36.15)	31.51 (24.70–35.55)	31.56 (24.83–36.81)	0.402	34.29 (28.80–35.98)	34.15 (31.83–35.74)	34.44 (28.55–36.68)	0.357	0.083
SFA (g)	16.13 (11.90–22.44)	15.80 (12.44–23.66)	16.20 (11.01–22.44)	0.937	17.28 (11.62–25.49)	18.94 (9.78–25.98)	16.21 (11.68–25.33)	0.940	0.562
SFA (g/kg)	0.25 (0.18–0.32)	0.26 (0.23–0.43)	0.23 (0.12–0.25)	0.195	0.26 (0.16–0.43)	0.28 (0.19–0.48)	0.19 (0.14–0.30)	0.061	0.740
SFA (%)	9.76 (7.47–12.11)	8.75 (7.33–12.43)	10.02 (7.76–12.08)	0.910	9.91 (8.22–11.97)	9.68 (8.33–11.95)	10.16 (7.07–14.14)	0.840	0.715
Cholesterol (mg)	199.54 (132.00–317.36)	211.07 (127.33–350.23)	187.73 (132.00–315.16)	0.197	209.50 (126.90–334.26)	254.25 (155.17–364.70)	144.65 (111.17–219.74)	0.236	0.974
Cholesterol (mg/kg)	3.55 (1.77–4.80)	4.15 (1.95–7.12)	1.96 (1.66–3.82)	0.309	3.14 (1.82–5.24)	4.79 (3.10–6.70)	1.89 (1.65–2.91)	0.006	0.921
Fiber (g)	10.37 (8.57–15.56)	14.27 (8.75–22.90)	9.26 (7.64–11.61)	0.126	11.83 (8.24–17.88)	11.70 (9.15–16.59)	11.99 (8.00–18.61)	0.135	0.580
Fiber (g/kg)	0.15 (0.10–0.29)	0.24 (0.15–0.44)	0.11 (0.09–0.15)	0.006	0.19 (0.10–0.26)	0.23 (0.13–0.30)	0.13 (0.09–0.26)	0.056	0.666

Data were represented as median and interquartile range. Abbreviations: SFA: Saturated Fatty Acid. Statistical comparisons were performed by Kruskal–Wallis 1-way ANOVA test (*P* value ≤ 0.05). *By the identification of discrepant values in the checking of outliers, one control group participant was excluded.

were observed in dietary intake of women with or without PCOS. Comparable results were observed in specific subgroups, i.e., eutrophic women subgroup and overweight women subgroup, with or without PCOS, had similar dietary patterns. Specifically in the PCOS group, for results expressed by kilogram of weight, it was identified that overweight women consumed less energy ($p = 0.001$), carbohydrate ($p = 0.012$), protein ($p = 0.031$), fat ($p = 0.047$) and fibers ($p = 0.006$) than normal weight PCOS women. In the group without PCOS, for results expressed by kilogram of weight it was observed that overweight women consumed lower amounts of energy ($p = 0.009$), protein ($p = 0.003$) and cholesterol ($p = 0.006$) than eutrophic women without PCOS.

There was a positive correlation between dietary protein ($r = 0.344$; $p = 0.050$), fat ($r = 0.447$; $p = 0.009$) and saturated fatty acids ($r = 0.403$; $p = 0.020$) with the after glucose challenge test of women without PCOS (Table 3). In the analysis of the associations between dietary intake, metabolic parameters and body composition, PCOS women showed an inverse correlation between dietary fiber intake and the HOMA-IR index ($r = -0.365$; $p = 0.024$), i.e., the higher the fiber intake, the lower the HOMA-IR index value was observed in PCOS women. In addition, for a similar fiber intake in women with and without PCOS, the range of HOMA-IR index values is higher in PCOS than in control groups (Fig. 1A and B).

In PCOS group, dietary fiber intake presented an inverse correlation with total body fat ($r = -0.401$; $p = 0.011$), trunk fat ($r = -0.388$; $p = 0.015$), and android fat ($r = 0.431$; $p = 0.006$) (Table 3). The relationship between fiber intake and visceral adiposity (android fat) in both groups is shown in Fig. 1C and D.

4. Discussion

The present study demonstrated that women with PCOS present a negative correlation between the consumption of dietary fiber and resistance to insulin, i.e., the higher the consumption of fiber, greater will be the insulin sensitivity presented by the patient. The least resistance to the action of insulin associated with consumption of dietary fiber identified in basal conditions in women with PCOS is lost in time 120 minutes of the OGTT. These results permit the interpretation that to obtain a better metabolic control in PCOS women, they should be guided toward the regular daily consumption of the recommended quantities of dietary fiber (20–35 g/day) [33], as well as the restriction in the consumption of sugar and sweet foods. It is important to highlight that these guidelines are included in the dietary recommendations for healthy individuals, elaborated by responsible organs from a number of countries [34], including the Alimentary Guide for the Brazilian Population, proposed by the Brazilian Ministry of Health [35].

Few researchers have investigated the relationship between food intake, metabolic parameters and body composition in women with PCOS. In the present study, it was demonstrated, exclusively for PCOS women, that dietary fiber intake is negatively associated with body composition, in particular in terms of central fat, i.e., a body composition parameter that has been specifically investigated in women with PCOS [12,13,15]. In agreement with these results, Nybacka et al. [36] identified that the increase in the intake of dietary fiber in women with PCOS presents a negative correlation with the increase in BMI. According to these researchers, the increase in dietary fiber intake is a primary predictor to the reduction in the BMI.

In the present study, it was demonstrated that women with PCOS and investigated controls, presented a similar ingestion of energy, macronutrients, saturated fatty acids, cholesterol and fiber. These results confirm those of previous studies that also did not identify differences in food consumption, between women with and those without PCOS [21–23].

Table 3
Relations between the food intake, metabolic parameters and body composition of women with and without PCOS.

Nutrient	Metabolic parameters				Body composition		
	HOMA-IR	Fasting insulin	Fasting glucose	Glucose 120′	Body fat	Trunk fat	Android fat
Carbohydrate (g)							
PCOS	r = −0.199	r = −0.163	r = −0.173	r = −0.020	r = −0.151	r = −0.152	r = −0.171
	p = 0.231	p = 0.335	p = 0.300	p = 0.905	p = 0.357	p = 0.355	p = 0.298
Controls*	r = −0.970	r = −0.076	r = −0.159	r = 0.230	r = 0.038	r = −0.070	r = −0.085
	p = 0.591	p = 0.673	p = 0.376	p = 0.197	p = 0.832	p = 0.696	p = 0.632
Protein (g)							
PCOS	r = −0.103	r = −0.189	r = −0.075	r = −0.026	r = −0.120	r = −0.064	r = −0.102
	p = 0.539	p = 0.263	p = 0.656	p = 0.875	p = 0.469	p = 0.697	p = 0.535
Controls*	r = −0.195	r = −0.203	r = −0.164	r = 0.344	r = −0.219	r = −0.231	r = −0.266
	p = 0.276	p = 0.258	p = 0.361	p = 0.050	p = 0.222	p = 0.189	p = 0.128
Fat (g)							
PCOS	r = −0.163	r = −0.267	r = 0.006	r = 0.108	r = −0.084	r = −0.087	r = −0.072
	p = 0.328	p = 0.111	p = 0.972	p = 0.520	p = 0.611	p = 0.597	p = 0.664
Controls*	r = −0.161	r = −0.139	r = −0.196	r = 0.447	r = −0.045	r = −0.048	r = −0.078
	p = 0.370	p = 0.441	p = 0.274	p = 0.009	p = 0.802	p = 0.788	p = 0.661
SFA (g)							
PCOS	r = −0.078	r = −0.185	r = 0.078	r = 0.141	r = 0.050	r = 0.056	r = 0.047
	p = 0.640	p = 0.273	p = 0.641	p = 0.400	p = 0.763	p = 0.734	p = 0.775
Controls*	r = −0.065	r = −0.068	r = −0.058	r = 0.403	r = 0.008	r = 0.005	r = −0.025
	p = 0.720	p = 0.707	p = 0.749	p = 0.020	p = 0.963	p = 0.977	p = 0.889
Fiber (g)							
PCOS	r = −0.365	r = −0.313	r = −0.306	r = −0.176	r = −0.401	r = −0.388	r = −0.431
	p = 0.024	p = 0.059	p = 0.062	p = 0.289	p = 0.011	p = 0.015	p = 0.006
Controls*	r = −0.326	r = −0.364	r = −0.009	r = 0.316	r = −0.122	r = −0.081	r = −0.097
	p = 0.064	p = 0.037	p = 0.959	p = 0.073	p = 0.499	p = 0.650	p = 0.587

Abbreviations: SFA: Saturated Fatty Acid; HOMA-IR: Homeostasis Model Assessment-estimated Insulin Resistance. Analysis by Spearman's correlation coefficient (*P* value ≤ 0.05). *By the identification of discrepant values in the checking of outliers, one control group participant was excluded.

In the specific analysis concerning food intake for the subgroups made up of overweight women and eutrophic women, with or without PCOS, it was identified that regardless of the diagnosis of PCOS, the participants from the overweight subgroups present lower energy and macronutrient food intake (expressed in kilocalories/kg of body weight), than the participants of the eutrophic subgroups. Several factors should be analyzed in an attempt to explain these results. Initially, it is necessary to consider that due to

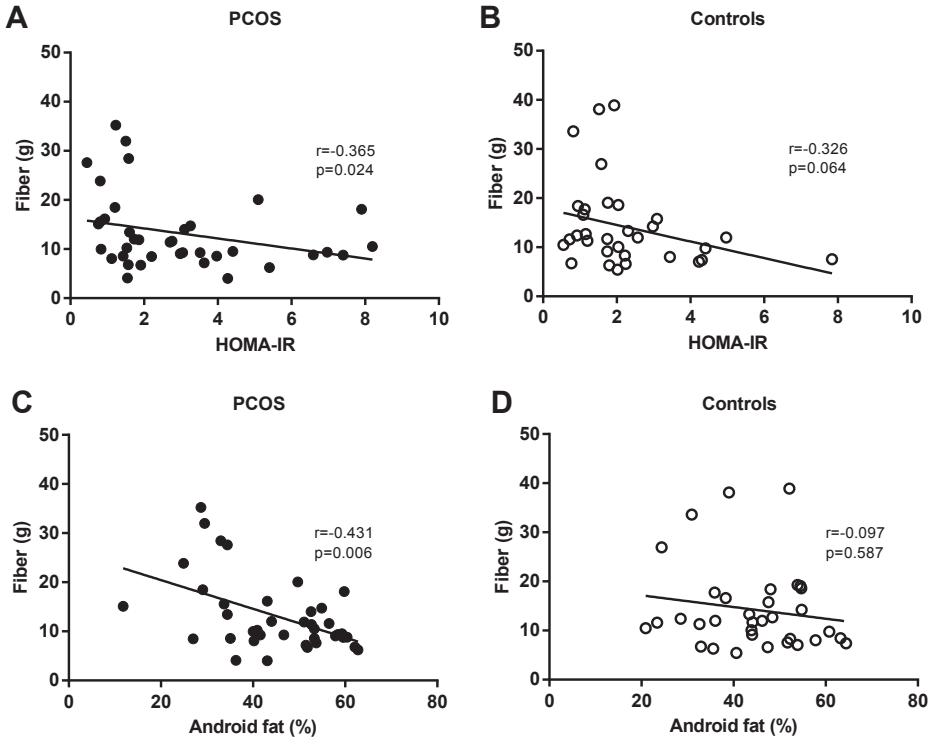

Fig. 1. Association between the fiber intake and HOMA-IR of women with PCOS (A) and controls (B); and association between the fiber intake and android fat of women with PCOS (C) and controls (D). Analysis by Spearman's correlation coefficient (*P* value ≤ 0.05). By the identification of discrepant values in the checking of outliers, one control group participant was excluded.

higher body mass, overweight patients may present a reduction in habitual physical activity, with a consequent decrease in daily energy expenditure [37]. Another possibility is that due to the alteration in body composition, with an increase in fat mass/lean mass ratio, there is a decrease in the metabolic rate and the daily energy needs, i.e., overweight patients may actually present a decrease in daily energy intake expressed in kilocalories/kg of current body weight [38]. In addition, it is necessary to consider that although there exist many advantages to using the seven-day food report [27], some women with or without PCOS, may have intentionally given an underreporting of food intake [39].

Underreporting of energy intake in food records has been mainly described among obese patients [39]. The identification of energy underreporting in food records can be made, e.g., by the measurement of the resting metabolic rate through indirect calorimetry [39] or the total energy expenditure by using the doubly labeled water technique [40]. In the present study, no method was used to confirm the energy intake of women with or without PCOS.

In the comparison with matched controls by BMI and age, the PCOS patients included in the present study showed similar values for the measurements taken using DXA for total fat, trunk fat, and android fat. All overweight patients, independent to being part of the PCOS group or not, presented a higher quantity of total body fat, trunk fat, and android fat. In addition, there were no differences identified in the body fat of eutrophic women, with or without PCOS. These results confirm those of previous studies that did not identify differences in the quantities and in the distribution of body fat between women with PCOS and control groups [11]. However, it is necessary to highlight that there still does not exist a consensus in the literature concerning this question. In disagreement with the results presented in this study, Carmina et al. [13] identified that central abdominal fat was increased by 30% in eutrophic PCOS women and 71% in overweight PCOS women, in comparison to 50% of the control women without PCOS. Higher central fat mass in PCOS women compared with control groups was also demonstrated by Glintborg et al. [15].

As particular strengths in this study, one can highlight the use of the seven-day food report, an instrument that presents higher accuracy in estimating habitual food intake of individuals. Another feature is the use of a variety of parameters for evaluating possible metabolic alterations, including the HOMA-IR index, as well as the use of the DXA for evaluating body composition. The limitations of the study are related to the observational study design, as it is not possible to attribute causality in the results found, although there were associations identified between the consumption of fiber, metabolic parameters and body composition in PCOS women. It is hoped that in future clinical studies, it will be the effect of consuming adequate dietary fiber on the metabolism and body composition of PCOS patients.

In conclusion, the systematic guidance toward the adequate consumption of dietary fiber, considering as source foods the fruits, leafy vegetables and grains, contributes to adequate body composition, the control of the glucose metabolism and possibly to the prevention of chronic non-communicable diseases in PCOS women.

Conflict of interest statement and funding sources

None of the authors had any personal or financial conflict of interest.

CRediT authorship contribution statement

Nayara Bernardes da Cunha: Conceptualization, Formal analysis, Investigation, Methodology, Writing - original draft. **Camila Toffoli Ribeiro:** Conceptualization, Formal analysis, Investigation, Methodology, Writing - original draft. **Catarina Mendes Silva:** Conceptualization, Formal analysis, Investigation, Methodology, Writing - original draft. **Ana Carolina Japur de Sá Rosa-e-Silva:** Conceptualization, Methodology, Project administration, Writing - review & editing. **Daurea Abadia De-Souza:** Conceptualization, Methodology, Supervision, Writing - review & editing.

Acknowledgements

We express our appreciation to the participants of study for their collaboration. Researchers N.B.C. and C.M.S. received a grant from the Residência Multiprofissional em Saúde, section of the Ministério da Educação, Brazil.

References

[1] Sirmans SM, Pate KA. Epidemiology, diagnosis, and management of polycystic ovary syndrome. Clin Epidemiol 2013;6:1–13.
[2] Ding T, Hardiman PJ, Petersen I, Wang FF, Qu F, Baio G. The prevalence of polycystic ovary syndrome in reproductive-aged women of different ethnicity: a systematic review and meta-analysis. Oncotarget 2017;8(56):96351–8.
[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] Jeanes YM, Reeves S. Metabolic consequences of obesity and insulin resistance in polycystic ovary syndrome: diagnostic and methodological challenges. Nutr Res Rev 2017;30:97–105.
[5] Dumesic DA, Oberfield SE, Stener-Victorin E, Marshall JC, Laven JS, Legro RS. Scientific statement on the diagnostic criteria, epidemiology, pathophysiology, and molecular genetics of polycystic ovary syndrome. Endocr Rev 2015;36(5):487–525.
[6] Conway G, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Franks S, Gambineri A, et al. The polycystic ovary syndrome: a position statement from the European Society of Endocrinology. Eur J Endocrinol 2014;171:P1–29.
[7] Melo AS, Dias SV, Cavalli RC, Cardoso VC, Bettiol H, Barbieri MA, et al. Pathogenesis of polycystic ovary syndrome: multifactorial assessment from the fetal stage to menopause. Reproduction 2015;150:R11–24.
[8] Merkin SS, Phy JL, Sites CK, Yang D. Environmental determinants of polycystic ovary syndrome. Fertil Steril 2016;106(1):16–24.
[9] Scott D, Harrison CL, Hutchison S, Courten B, Stepto NK. Exploring factors related to changes in body composition, insulin sensitivity and aerobic capacity in response to a 12-week exercise intervention in overweight and obese women with and without polycystic ovary syndrome. PLoS One 2017;12(8):1–13.
[10] Jeanes YM, Reeves S. Metabolic consequences of obesity and insulin resistance in polycystic ovary syndrome: diagnostic and methodological challenges. Nutr Res Rev 2017;30:97–105.
[11] Barber TM, Golding SJ, Alvey C, Wass JAH, Karpe F, Franks S, et al. Global adiposity rather than abnormal regional fat distribution characterizes women with polycystic ovary syndrome. J Clin Endocrinol Metabol 2008;93(3):999–1004.
[12] Dolfing JG, Stassen CM, Haard PMM, Wolffenbuttel BHR, Schweitzer DH. Comparison of MRI-assessed body fat content between lean women with polycystic ovary syndrome (PCOS) and matched controls: less visceral fat with PCOS. Hum Reprod 2011;26(6):1495–500.
[13] Carmina E, Bucchieri S, Esposito A, Puente AD, Mansueto P, Orio F, et al. Abdominal fat quantity and distribution in women with polycystic ovary syndrome and extent of its relation to insulin resistance. J Clin Endocrinol Metabol 2007;92(7):2500–5.
[14] Battaglia C, Battaglia B, Mancini F, Paradisi R, Fabbri R, Venturoli S. Ultrasonographic extended-view technique for evaluation of abdominal fat distribution in lean women with polycystic ovary syndrome. Acta Obstet Gynecol Scand 2011;90:600–8.
[15] Glintborg D, Petersen MH, Ravn P, Hermann AP, Andersen M. Comparison of regional fat mass measurement by whole body DXA scans and anthropometric measures to predict insulin resistance in women with polycystic ovary syndrome and controls. Acta Obstet Gynecol Scand 2016;95:1235–43.
[16] Macruz CF, Lima SMRR, Salles JEN, Silva GMD, Scalissi NM. Assessment of the body composition of patients with polycystic ovary syndrome using dual-energy X-ray absorptiometry. Int J Gynecol Obstet 2017;136:285–9.
[17] Larsson I, Hulthén L, Landén M, Palsson 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(1):213–8.
[18] Rodrigues AMS, Martins LB, Franklin AMT, Candido AL, Santos LC, Ferreira AVM. Poor quality diet is associated with overweight status and obesity in patients with polycystic ovary syndrome. J Hum Nutr Diet 2014;28:94–101.

[19] Zhang J, Liu Y, Liu X, Xu L, Zhou L, Tang L, et al. High intake of energy and fat in Southwest Chinese women with PCOS: a population-based case-control study. PloS One 2015;10(5):1–13.
[20] Eslamian G, Baghestani AR, Eghtesad S, Hekmatdoost A. Dietary carbohydrate composition is associated with polycystic ovary syndrome: a case-control study. J Hum Nutr Diet 2017;30:90–7.
[21] 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(2):411–7.
[22] 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.
[23] 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.
[24] Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertil Steril 2004;81(1):19–25.
[25] Balen A. Ovulation induction for polycystic ovary syndrome. Hum Fertil 2003;3:106–11.
[26] Munro MG. Practical aspects of the two FIGO systems for management of abnormal uterine bleeding in the reproductive years. Best Pract Res Clin Obstet Gynaecol 2017;40:3–22.
[27] Ferriman D, Gallwey JD. Clinical assessment of body hair growth in women. J Clin Endocrinol Metabol 1961;21:1140–7.
[28] World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO Consultation on Obesity. Geneva: World Health Organization; 2000.
[29] Thompson FE, Byers T. Dietary assessment resource manual. J Nutr 1994;124(11 Suppl):2245S–317S.
[30] NEPA-UNICAMP – Núcleo de estudos e pesquisas em alimentação – NEPA/Universidade Estadual de Campinas – UNICAMP. Tabela brasileira de composição de alimentos – TACO. 4 ed. Campinas: NEPA-UNICAMP; 2011 [Portuguese].
[31] US Department of Agriculture, Agricultural Research Service, Nutrient Data Laboratory. USDA National nutrient database for standard reference, release 28 (slightly revised). Version Current. May 2016. Internet:, http://www.ars.usda.gov/ba/bhnrc/ndl.
[32] Jensterle M, Weber M, Pfeifer M, Prezelj J, Pfutzner A, Janez A. Assessment of insulin resistance in young women with polycystic ovary syndrome. Int J Gynaecol Obstet 2008;102(2):137–40.
[33] Marlett JA, McBurney MI, Slavin JL, American Dietetic Association. Position of the American Dietetic Association: health implications of dietary fiber. J Am Diet Assoc 2002;102(7):993–1000.
[34] Escott-Stump S, Earl R. Guidelines for dietary planning. In: Mahan LK, Escott-Stump S, editors. Krause's food & nutrition therapy, vol. 12. Rio de Janeiro: Elsevier; 2008. p. 337–62.
[35] BRASIL. Ministério da Saúde. Secretaria de Atenção à Saúde. Coordenação-Geral da Política de Alimentação e Nutrição. Guia alimentar para a população brasileira: promovendo a alimentação saudável. Brasília: Ministério da Saúde; 2006.
[36] Nybacka A, Hellström PM, Hirschberg AL. Increased fibre and reduced trans fatty acid intake are primary predictors of metabolic improvement in overweight polycystic ovary syndrome – substudy of randomized trial between diet, exercise and diet plus exercise for weight control. Clin Endocrinol 2017;87:680–8.
[37] Oppert JM, Laville M, Basdevant A. Obesity phenotype. In: Clement K, Sorensen TIA, editors. Obesity: genomics and postgenomics, vol. 1. Boca Raton: CRC Press; 2007. p. 1–18.
[38] Astrup A, Buemann B, Toubro S, Ranneries C, Raben A. Low resting metabolic rate in subjects predisposed to obesity: a role for thyroid status. Am J Clin Nutr 1996;63(6):879–83.
[39] Fricker J, Baelde D, Igoin-Apfelbaum L, Huet JM, Apfelbaum M. Underreporting of food intake in obese "small eaters". Appetite 1992;19(3):273–83.
[40] Moshfegh AJ, Rhodes DG, Baer DJ, Murayi T, Clemens JC, Rumpler WV, et al. The US department of agriculture automated multiple-pass method reduces bias in the collection of energy intakes. Am J Clin Nutr 2008;88:324–32.