1 Abstract

2 Objective: To compare dietary intake and lifestyle behaviour in women with polycystic ovarian
3 syndrome (PCOS) and healthy women.

Methods: 160 healthy women (partner with male infertility) were recruited to a control group;
168 women with PCOS (diagnosed on ultrasound) were recruited to a case study group for this
cross-sectional comparative study. The case group was classified into three phenotypes based on
presence or absence of menstrual disorder (M), hyperandrogenism (HA), and polycystic ovary
according to sonography (PCO): HA+PCO (n=53), PCO+M (n=57) and M+HA+PCO (n=66).
Dietary intake and lifestyle behaviour were measured using a food frequency questionnaire
(FFQ) and a lifestyle questionnaire (LQ).

Results: The mean energy (P<0.001) and fat intake (P<0.001) were greater in PCOS groups 11 compared with the control group. The average energy and fat intake were greater in 12 HA+M+PCO group after age and BMI adjustment compared with other phenotypes (P<0.001). 13 In comparison with the control group, lifestyle scores were lower in the PCOS group in the fields 14 of physical activity, weight and nutrition control after age and BMI adjustment (P<0.001). The 15 average score of lifestyle in the fields of physical activity, weight and nutrition control, and 16 17 psychological health was lower in the phenotype HA+M+PCO compared with other phenotypes (P<0.001). 18

19 Conclusions: Limited energy and fat intake is strongly recommended in Iranian women with 20 PCOS especially in phenotype HA+M+PCO. Consultation on improvement of psychological 21 health and the importance of weight and nutrition control, and appropriate physical activity in 22 patients especially in HA+M+PCO is advocated.

23 Keywords: Lifestyle, polycystic ovarian syndrome, diet

25 Introduction

Polycystic ovarian syndrome (PCOS) is a common and complex endocrine disorder that affects women of reproductive age ⁽¹⁾. Three phenotypes are recognized based on the presence or absence of symptoms using the Rotterdam criteria: oligo-ovulation (irregular menses) with polycystic ovary in sonography (M+PCO), hyperandrogenism with polycystic ovary in sonography (HA+PCO) and hyperandrogenism with oligo-ovulation (irregular menses) and polycystic ovary (M+PCO+HA) ^(2, 3). Although, PCOS can exist in the absence of obesity, 70 percent of women with PCOS are obese ⁽⁴⁾.

33

Obesity can intensify metabolic and fertility outcomes related to this syndrome. For this reason, 34 the treatment of PCOS focuses mainly on weight loss if obesity is present because energy 35 limitation is associated with improved metabolic status and energy intakes ⁽⁵⁻⁷⁾. However, the 36 relationship between diet and PCOS is not yet well understood and there is limited information 37 available in this field. It is reported that total energy intake and intake of micronutrients is similar 38 in PCOS and control groups (Carmina et al.⁸) and that consumption of certain foods with high 39 glycaemic index is greater in women with PCOS (Douglas et al.⁹). Extensive long-term studies 40 have identified a significant relationship between diet and the risk of hypertension, type 2 41 diabetes mellitus (T2DM), and cardiovascular disease in a healthy population ⁽¹⁰⁻¹⁴⁾. Most studies 42 which assess the nutritional status of women with PCOS are flawed because they do not allow 43 44 for the different clinical phenotypes in PCOS which result in significantly different clinical and metabolic parameters. Pikee et al. ⁽¹⁵⁾ have reported that women with the hyperandrogenism 45 46 phenotype have a higher BMI and worse clinical and endocrine status (TG/HDL, LH/FSH, 47 testosterone, LH) compared with other phenotypes. The only study which has investigated the

potential differences between phenotypes in PCOS and their dietary intake has reported that
women with phenotype HA+M+PCO had more daily energy intake compared with healthy
women ⁽¹⁶⁾.

51

Dietary habits are rooted in the culture of communities, and since the relationship between dietary habits and the prevalence of some cardiovascular disease and T2DM is proven, it seems logical and imperative to compare dietary intake between women with different phenotypes of PCOS in different cultures. To deliver appropriate nutritional interventions in the case of obesity or increased risk factors related to diet, PCOS cardiovascular status is required. The purpose of the present study was to compare dietary intake and lifestyle factors in women with three different phenotypes of PCOS with a control group of healthy women.

59

60 Methods

61 *Design and data collection*

The present study is a cross sectional study with a control and case group divided into three subgroups. The study was conducted at an acute hospital in Hormozgan Province, Iran. Women were recruited through the infertility clinic at a provincial hospital. The case group included women with PCOS. The control group comprised healthy women who had been referred to this clinic because of male infertility.

A simple sampling method was used. The sample size was calculated using the following
formula (Douglas et al.⁹) with CI 95% as at least 145 women in each group.

69
$$N = \frac{\left(Z_{1-\frac{\alpha}{2}} + Z_{1-\beta}\right)^2 \left(S_1^2 + S_2^2\right)}{\Delta^2}$$

70 μ1: 61.5; μ2: 69.2; S1: 21.1; S2: 25.0.

71 The research team approached 362 women to explain the purpose of study; written consent was obtained from each participant who volunteered to participate and questionnaires were 72 distributed and completed at the same clinic appointment. 34/362 women declined for unknown 73 74 reasons. Inclusion criteria for all groups were: age 15-40 years, married, Iranian nationality, and absence of linguistic or cognitive problems, lack of underlying disease (diabetes, hypertension, 75 76 diagnosed anaemia, or any other disease requiring a special diet). Additional inclusion criteria for the case group were women with PCOS based on two of the following three Rotterdam 77 criteria: ultrasound scan of polycystic ovary (>12 follicles in both ovaries and ovarian volume 78 79 >10 mm); clinical signs of hyperandrogenism: clinical score of hyperandrogenism >7 or obvious acne); menstrual cycles of greater than 35 days or amenorrhea (absence of menstruation for 199 80 days). And absence of congenital adrenal hyperplasia, thyroid dysfunction, hyperprolactinemia. 81 The selected participants in the case group were classified into three phenotypes: HA+PCO, 82

84

83

85 *Measures*

M+PCO, HA+M+PCO.

86 Menstrual history measured as the interval between two menses during the previous 12 months:

87 <21 days, 21-34 days, 35-60 days, >199 days, variable.

88 BMI: calculated as an individual's weight in kilograms divided by height in metres²

89 Body hair: based on the Ferriman-Gallway hirsutism scoring scale which measures nine

- 90 androgen sensitive areas in the body. Each area is rated from zero to 4 based on the degree of
- 91 terminal hair growth. A score of 7 or more indicated hirsutism ⁽¹⁷⁾.

Acne: a global acne grading system was used to measure acne. This scale includes six body areas of face, chest, and upper back based on the level of involvement, distribution, density, and pilosebaceous units. Each body area is rated from zero to four. The most severe lesion of each area determines the score of that area. The score of each area is multiplied by the factor score which is based on the involved area: forehead, left and right cheek, nose, chin, chest and upper back. The total acne score is obtained by multiplying the factor score by sum score of involved areas ⁽¹⁸⁾.

99 Socio economic status: formal education of women was considered as an indicator of social
100 status ⁽¹⁹⁾.

Food frequency questionnaire: dietary intake was measured using a modified food frequency 101 questionnaire (FFQ) based on Iranian dietary questionnaire which contains 168 items. The 102 reliability and validity of the questionnaire are approved in Iran⁽²⁰⁾. FFQ included a list of foods 103 with a standard size of a food. Subjects were asked to report the frequency of consumption of 104 each food during the past month on a daily, weekly or monthly basis. The amount of nutritional 105 items consumed was converted to grams using household scales. This dietary information was 106 analyzed using the software Nutrition4 which calculated the amount of energy, macronutrients 107 108 (carbohydrates, lipid, and protein) and micronutrients (at least 30 micronutrients) including fat soluble vitamins, water soluble vitamins and minerals ^(20, 21). 109

110

Lifestyle questionnaire (LSQ) which compromises 70 items in 10 subscales including physical health (8 items), sports and fitness (7 items), weight management and nutrition (7 items), disease prevention (7 items), mental health (7 items), spiritual health (6 items), social health (7 items), avoidance of drugs, alcohol and opiates (6 items), accident prevention (8 items) and

115 environmental health (7 items). All items are graded on a four-point Likert scale scoring in range from 0 (=never) to 3 (=always). The higher the score, the better the lifestyle. Lali et al. ⁽²²⁾ have 116 confirmed the validity and reliability of this questionnaire for Iranian society. 117 118 Ethical consideration 119 The ethics committee of Hormozgan University of Medical Science approved the present study. 120 After explaining the purpose of the study and securing the confidentiality of information, all 121 subjects gave written consent. 122 123 Statistical analysis 124 The Mean \pm SD and n (%) were used for quantitative and qualitative variables respectively. T 125 126 test and ANOVA test were used for intergroup comparison of PCOS patients for quantitative variables and comparison of PCOS phenotypes respectively. BMI and age were adjusted by co-127 variance analysis. Data were analyzed using Statistical Package for the Social Sciences 21.0 128

129 (SPSS Inc., Chicago, IL, USA). Significance level of P<0.05 was accepted.

130

131 **Results**

132 *Study population*

Table 1 shows the clinical and demographic information of all participants. There was no significant difference between the control and case study groups in terms of demographic and clinical characteristics except for menstrual cycle interval, hirsutism and acne scores (P>0.05). There were no significant differences between three PCOS phenotype groups in terms of demographic and clinical characteristics.

138

139 *Dietary intake*

Table 2 shows the comparison of women with PCOS and the women in the control group based on their dietary intake. Energy and fat intake were greater in the PCOS group after adjustment for BMI and age compared with control group (P<0.01). Table 3 shows dietary intake in the different phenotypes of PCOS. Energy intake and fat intake were significantly higher in phenotype HA+M+PCO compared with phenotypes HA+PCO and M+PCO. This statistical difference remained after adjustment for age. Other micro/macro nutrient intake was similar across phenotype groups.

147

148 *Lifestyle*

Table 4 shows the comparison of PCOS and control groups based on the domains of lifestyle.
Scores of physical activity, nutrition and weight control in the PCOS group were significantly
lower than in the control group after BMI and age adjustment, indicating a poorer lifestyle in
women in the PCOS group in the mentioned domains above.

153

Table 5 shows the lifestyle scores across the different PCOS phenotype groups of women. Scores
of physical activity, nutrition and weight control and psychological health were significantly
lower in HA+M+PCO phenotype after BMI and age adjustment compared with other phenotypes
(P<0.001) indicating a poorer lifestyle of HA+M+PCO phenotype.

158

159 **Discussion**

160 The results of the present study showed that after BMI and age adjustment, energy, fat, saturated fatty acids and polyunsaturated fatty acids intake were significantly greater in the PCOS group 161 compared with the control group. Given that quality of the diet increases by fibre and 162 micronutrients intake, and decreases by saturated fat intake ⁽²³⁾, the quality of diet may be lower 163 in the PCOS group compared with the control group. Our findings suggest that the quality of diet 164 165 is positively affected by polyunsaturated fatty acids intake. Previous studies show that quality of diet is a dietary intake measure which is associated with unfavourable metabolic outcomes and 166 increased risk of chronic disease mortalities ^(24, 25). Wild et al. ⁽²⁶⁾ reported increased fat intake 167 and decreased fibre intake in women with PCOS compared with a control group. Moran et al. (27) 168 showed women with PCOS had a better diet quality as indicated by a higher diet quality score 169 and higher energy, fibre, folate, iron, calcium, magnesium, niacin, phosphorus, potassium, 170 171 sodium, vitamin E and zinc intake and lower percentage energy from saturated fat intake, glycaemic index and retinol intake than women without PCOS. It should be noted that in this 172 study, diagnosis of PCOS was self-reported and dietary intake was measured using a dietary 173 questionnaire for epidemiological studies. These questionnaires measure 80 types of nutrients 174 consumed during the past 12 months which differs from the present study as the FFQ is an 175 176 appropriate tool for measurement of diet components in a certain period. However, data accuracy 177 may be limited due to the ability of the responders to remember their intake. Moreover, women with PCOS reported higher carbohydrate (229 vs. 61 g), protein (78 vs. 66.3 g), fat (85 vs. 61.1 178 g), saturated fat (26 vs. 22.5 g) intake compared with reference population ⁽²⁸⁾. A high energy 179 diet has been reported in Iranian⁽²⁹⁾ and Brazilian⁽¹⁶⁾ women with PCOS. Using three 24-hour 180 dietary recall questionnaires, Ahmadi et al. (29) reported that daily energy intake (about 300 kcal) 181 182 was higher in women with PCOS. These women also had more total and saturated fat intake

compared with control group ⁽²⁹⁾. A 24 hours dietary diary has similar limitations to the FFQ as it 183 also depends on an individual respondent's memory. However, it is an appropriate method which 184 can present accurate information when it is used by a trained interviewer in a standard approach. 185 Furthermore, the results of the present study show for the first time that the energy and saturated 186 fat intake were greater in HA+M+PCO phenotype after age and BMI adjustment compared with 187 other phenotypes. To date, this is the only study to compare dietary intake PCOS phenotypes 188 with healthy women. The results of Graff et al. (16) using a 121 items FFQ showed that energy 189 intake was greater in classic phenotype of PCOS (HA+M+PCO) compared with the control 190 191 group; however, this statistical difference did not exist after age and BMI adjustment. It should be noted that Graff et al considered only two phenotypes of HA+PCO+M (n=39) and ovulatory 192 PCOS (n=22). In the present study, classification of PCOS phenotype was based on the 193 Rotterdam criteria. 194

195

It has been shown that HA+M+PCO phenotype has three times higher levels of androgen which 196 in turns increases the prevalence of glucose tolerance disorder and insulin resistance more than 197 other phenotypes ⁽¹⁶⁾. Testosterone stimulates appetite and an increased level of androgen in 198 women is associated with appetite control disorder ⁽³⁰⁾. Moreover, hyperandrogenism is essential 199 in determining the risk of cardiovascular disease in PCOS phenotypes ⁽³¹⁾. Current clinical 200 evidence suggests that testosterone increases abdominal fat accumulation (32-34). Increased 201 202 abdominal adiposity is associated with increased Leptin hormone and Leptin resistance which may be associated with increased energy intake ⁽³⁵⁾. High fat intake (total and saturated fat) in the 203 204 present study is a concern for patients with PCOS specially HA+PCO+M phenotype; because 205 fatty acid intake affects the glucose metabolism by making changes in insulin signaling and cell

membrane function. In addition, high saturation fat diet is associated with decrease insulin
 resistance ⁽³⁶⁾.

208

However, a high level of physical activity improves the glucose metabolism and sensitivity to 209 insulin, and reduced abdominal obesity ⁽²²⁾; the risk of which is higher in phenotypes 210 HA+PCO+M. Our results showed for the first time that phenotype HA+M+PCO has less 211 physical activity compared with two other phenotypes. Wright et al. (37) have reported no 212 significant difference in self-reported physical activity between American PCOS and non PCOS 213 patients. This finding is similar to the study of Ahmadi et al. ⁽²⁹⁾, Álvarez-Blasco et al. ⁽³⁸⁾ in 214 which there was no significant difference in physical activity between PCOS and non PCOS 215 patients in Iran and Spain. However, it should be noted that similar tools are not used in these 216 studies to measure physical activity. Wright et al. (37) have used Paffenburg physical activity 217 questionnaire that is validated in male and female samples. Álvarez-Blasco et al. ⁽³⁸⁾ and Ahmadi 218 et al. ⁽²⁹⁾ have used interview questionnaires which their validity is uncertain. In the present 219 study, we have used the Iranian version of the lifestyle questionnaire where the validity and 220 reliability are approved ⁽³⁰⁾. 221

222

A strength of the present study is that the diagnosis of PCOS phenotypes for allocation to case study groups was done by an expert physician in the infertility clinic and patients were classified to the clinical phenotypes based on the Rotterdam criteria. Moreover, a validated FFQ was used for diet evaluation which is a standard tool for measurement of long term dietary habits in cross sectional and cohort studies. Lifestyle questionnaire was used to evaluate participants' lifestyle. Nevertheless, there are some limitations in the present study. Patients were selected from the 229 only referral infertility clinic of Hormozgan province which limits generalization of the study results. Furthermore, the association between metabolic and hormonal features of the study 230 population and dietary intake was not included in this study; future studies should consider 231 different phenotypes of PCOS. In addition, since diet analysis was limited to the nutrition 232 database, glycaemic load could not be measured in this study. In the current study, we assess 233 234 physical activity by a lifestyle questionnaire that was designed by Likert scale. We did not assess physical activity using metabolic equivalents (METS). This should be considered in the 235 interpretation of our findings. 236

237

238 Conclusions

Reduced energy and fat intake is strongly recommended to Iranian women with PCOS especially 239 with the HA+M+PCO phenotype. It is critical to closely examine the metabolic and endocrine 240 status of women with menstrual disorder and hyperandrogenism because the treatment strategies 241 for energy deficit-related menstrual disturbances differ from that of disturbances attributable to 242 hyperandrogenaemia. Further investigation is necessary to explore whether different endocrine 243 etiologies underlay menstrual disorder and hyperandrogenism in different phenotypes of PCOS. 244 245 Consultation on improvement of psychological health and the importance of nutrition and weight control, and appropriate physical activity in these patients especially HA+M+PCO phenotype is 246 necessary. Future studies on the evaluation of the risk of metabolic side effects, dietary intake 247 248 and lifestyle is PCOS phenotypes are recommended.

249

250	Disclosure statement
251	The authors report no conflicts of interest.
252	
253	Transparency declaration
254	The lead author affirms that this manuscript is an honest, accurate, and transparent account of the
255	study being reported. The reporting of this work is compliant with the STROBE checklist. The
256	lead author affirms that no important aspects of the study have been omitted and that any
257	discrepancies from the study.
258	
259	

REFERENCES

1. Bozdag G, Mumusoglu S, Zengin D, *et al.* (2016) The prevalence and phenotypic features of polycystic ovary syndrome: a systematic review and meta-analysis. *Hum Reprod* **31**, 2841-2855

2. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group (2004) Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. *Fertility and sterility* **81**, 19-25

3. Azziz R, Carmina E, Dewailly D, *et al.* (2006) Positions statement: criteria for defining polycystic ovary syndrome as a predominantly hyperandrogenic syndrome: an Androgen Excess Society guideline. *J Clin Endocrinol Metab* **91**, 4237-4245

4. Azziz R, Sanchez LA, Knochenhauer ES, *et al.* (2004) Androgen excess in women: experience with over 1000 consecutive patients. *J Clin Endocrinol Metab* **89**, 453-462

5. Pasquali R, Gambineri A, Biscotti D, *et al.* (2000) Effect of long-term treatment with metformin added to hypocaloric diet on body composition, fat distribution, and androgen and insulin levels in abdominally obese women with and without the polycystic ovary syndrome. *J Clin Endocrinol Metab* **85**, 2767-2774

6. Pasquali R, Fabbri R, Venturoli S, *et al.* (1986) Effect of weight loss and antiandrogenic therapy on sex hormone blood levels and insulin resistance in obese patients with polycystic ovaries. American journal of obstetrics and gynecology **154**, 139-144

7. Kiddy DS, Hamilton-Fairley D, Seppala M, *et al.* (1989) Diet-induced changes in sex hormone binding globulin and free testosterone in women with normal or polycystic ovaries: correlation with serum insulin and insulin-like growth factor-I. *Clin Endocrinol* **31**, 757-763

8. Carmina E, Legro RS, Stamets K, *et al.* (2003) Difference in body weight between American and Italian women with polycystic ovary syndrome: influence of the diet. *Hum Reprod* **18**, 2289-2293

9. Douglas CC, Norris LE, Oster RA, *et al.* (2006) Difference in dietary intake between women with polycystic ovary syndrome and healthy controls. *Fertil Steril* **86**, 411-417

10. Liu S, Manson JE, Stampfer MJ, *et al.* (2000) A prospective study of whole-grain intake and risk of type 2 diabetes mellitus in US women. *Am J Public Health* **90**, 1409-1415

11. Meyer KA, Kushi LH, Jacobs DR, *et al.* (2000) Carbohydrates, dietary fiber, and incident type 2 diabetes in older women. *Am J Clin Nutr* **71**, 921-930

12. Colditz GA, Manson JE, Stampfer MJ, *et al.* (1992) Diet and risk of clinical diabetes in women. *Am J Clin Nutr* **55**, 1018-1023

13. Salmeron J, Manson JE, Stampfer MJ, *et al.* (1997) Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. *JAMA* **277**, 472-477

14. Hu FB, Stampfer MJ, Manson JE, *et al.* (1997) Dietary fat intake and the risk of coronary heart disease in women. *N Engl J Med* **337**, 1491-1499

15. Pikee S, Shivani S, Jayshree B (2016) Endocrine and metabolic profile of different phenotypes of polycystic ovarian syndrome. *J Obstet Gynaecol India* **66**, 560-566

16. Graff SK, Mario FM, Alves BC, *et al.* (2013) Dietary glycemic index is associated with less favorable anthropometric and metabolic profiles in polycystic ovary syndrome women with different phenotypes. *Fertil Steril* **100**, 1081-1088

17. Ferriman D, Gallwey JD (1961) Clinical assessment of body hair growth in women. *J Clin Endocrinol Metab* **21**, 1440-1447

18. Shyangdan D, Clar C, Ghouri N, *et al.* (2011) Insulin sensitisers in the treatment of nonalcoholic fatty liver disease: a systematic review. *Health Technol Assess* **15**, 1-110

19. Donyavi T, Naieni KH, Nedjat S, *et al.* (2011) Socioeconomic status and mortality after acute myocardial infarction: a study from Iran. *Int J Equity Health* **10**, 1475-9276

20. Hosseini Esfahani F, Asghari G, Mirmiran P, *et al.* (2010) Reproducibility and relative validity of food group intake in a food frequency questionnaire developed for the Tehran lipid and glucose study. *J Epidemiol* **20**, 150-158

21. Piyathilake CJ, Azrad M, Jhala D, *et al.* (2006) Mandatory fortification with folic acid in the United States is not associated with changes in the degree or the pattern of global DNA methylation in cells involved in cervical carcinogenesis. *Cancer Biomark* **2**, 259-266

22. Lali M, Abedi A, Kajbaf MB (2012) Construction and validation of the lifestyle questionnaire (LSQ). *Psychol Res* **1**, 64-80

23. McNaughton SA, Ball K, Crawford D, *et al.* (2008) An index of diet and eating patterns is a valid measure of diet quality in an Australian population. *J Nutr* **138**, 86-93

24. McNaughton SA, Dunstan DW, Ball K, *et al.* (2009) Dietary quality is associated with diabetes and cardio-metabolic risk factors. *J Nutr* **139**, 734-742

25. Wirt A, Collins CE (2009) Diet quality--what is it and does it matter? *Public Health Nutr*12, 2473-2492

26. Wild RA, Painter PC, Coulson PB, *et al.* (1985) Lipoprotein lipid concentrations and cardiovascular risk in women with polycystic ovary syndrome. *J Clin Endocrinol Metab* **61**, 946-951

27. Moran LJ, Ranasinha S, Zoungas S, *et al.* (2013) The contribution of diet, physical activity and sedentary behaviour to body mass index in women with and without polycystic ovary syndrome. *Hum Reprod* **28**, 2276-2283

28. Barr S, Hart K, Reeves S, *et al.* (2011) Habitual dietary intake, eating pattern and physical activity of women with polycystic ovary syndrome. *Eur J Clin Nutr* **65**, 1126-1132

29. Ahmadi A, Akbarzadeh M, Mohammadi F, *et al.* (2013) Anthropometric characteristics and dietary pattern of women with polycystic ovary syndrome. *Indian J Endocrinol Metab* **17**, 672-676

30. Asarian L, Geary N (2006) Modulation of appetite by gonadal steroid hormones. *Philos Trans R Soc Lond B Biol Sci* **361**, 1251-1263

31. Azziz R, Carmina E, Dewailly D, *et al.* (2009) The Androgen Excess and PCOS Society criteria for the polycystic ovary syndrome: the complete task force report. *Fertil Steril* **91**, 456-488

32. Kirchengast S, Huber J (2001) Body composition characteristics and body fat distribution in lean women with polycystic ovary syndrome. *Hum Reprod* **16**, 1255-1260

33. Eisner JR, Dumesic DA, Kemnitz JW, *et al.* (2003) Increased adiposity in female rhesus monkeys exposed to androgen excess during early gestation. *Obes Res* **11**, 279-286

34. Nohara K, Laque A, Allard C, *et al.* (2014) Central mechanisms of adiposity in adult female mice with androgen excess. *Obesity* **22**, 1477-1484

35. Galic S, Oakhill JS, Steinberg GR (2010) Adipose tissue as an endocrine organ. *Mol Cell Endocrinol* **316**, 129-139

36. Galgani JE, Uauy RD, Aguirre CA, *et al.* (2008) Effect of the dietary fat quality on insulin sensitivity. *Br J Nutr* **100**, 471-479

37. Wright CE, Zborowski JV, Talbott EO, *et al.* (2004) Dietary intake, physical activity, and obesity in women with polycystic ovary syndrome. *Int J Obes Relat Metab Disord* **28**, 1026-1032

38. Alvarez-Blasco F, Luque-Ramirez M, Escobar-Morreale HF (2011) Diet composition and physical activity in overweight and obese premenopausal women with or without polycystic ovary syndrome. *Gynecol Endocrinol* **27**, 978-981

Variable**	Pl	nenotype of PCC	Control	P value§	
	H+PCO	H+PCO+M	M+PCO	-	
	(n=53)	(n= 66)	(n= 57)		
Variable					
Age *	29.30±6.25	28.41±3.49	29.03±9.31	29.85±6.40	0.43
Education *	12.69±3.05	11.63±4.11	11.04 ± 4.92	10.82±4.31	0.62
Weight *	79.43 ± 13.1	76.31±15.41	78.35±10	75.3 ± 20.8	0.52
BMI*	39.80±6.53	38.43±7.11	35.42±2.34	33.32±2.96	0.87
Acne score*	6.93±1.38	6.58 ± 2.30	6.83±1.10	3.62±0.90	< 0.001
Hirsutism score*	11.30±0.84	11.03±0.31	11.17±0.01	9.40±0.18	< 0.001
Average menstrual cycle **					
<21	2 (3.77)	5 (7.57)	4 (7.01)	13 (8.1)	
21-35	22 (41.50)	28 (42.42)	30 (52.63)	76 (47.5)	
35-60	15 (28.30)	18 (27.27)	9 (15.78)	8 (5)	< 0.001
>199 days	6 (11.32)	8 (12.12)	9 (15.78)	18 (11.3)	
Variable	8 (15.09)	7 (10.60)	5 (8.77)	44 (27.5)	

Table 1. Demographic and clinical characterizes of participants

§ between PCOS and control group

*ANOVA test

**Kruskal wallis test

 \pounds P<0.05 between H+PCO and H+PCO+M phenotype

€ P<0.05 between H+PCO and M+PCO phenotype

¥ P<0.05 between H+PCO+M and M+PCO phenotype

Table 2. Comparison of PCOS patients and control group based on the dietary intake

Variable **	PCOS	Control	P value *	P value adjusted
	(n=168)	(n=160)		for BMI and age
Energy (Kcal/day)	2500.2±78.7	2202.8±49.6	< 0.001	< 0.001
Protein (g/day)	76.09 ± 10.79	74.25±9.36	0.41	0.82
Fat (g/day)	89.06±12.42	65.38±11.75	< 0.001	< 0.001
Saturated fatty acids (g/day)	35.97±9.24	21.16±5.56	< 0.001	< 0.001
Polyunsaturated fatty acids (g/day)	27.21±2.48	15.81 ± 2.48	< 0.001	< 0.001
Linoleic acid (g/day)	33.10±8.91	24.22 ± 9.40	0.21	0.32
EPA (g/day)	0.03 ± 0.002	0.01 ± 0.001	0.45	0.31
Sodium (mg/day)	2223.05 ± 31.08	1353.93±10.21	0.06	0.09
Iron (mg/day)	24.13±2.27	26.23±6.37	0.43	0.25
Magnesium (mg/day)	395.98±43.09	332.83 ± 26.28	0.51	0.40
Zinc (mg/day)	9.68±3.75	7.75 ± 2.55	0.59	0.41
Manganese (mg/day)	8.64±3.11	9.26±4.91	0.98	0.43
Fluoride (µg/day)	4129.04±60.51	3163.40±23.50	0.61	0.52
Iodine (µg/day)	0.28±0.31	0.02 ± 0.01	0.45	0.31
Vitamin A(µg/day)	2701.59±79.35	1347.72±13.10	0.39	0.15
Vitamin E (mg/day)	3.98 ± 0.67	3.11±0.85	0.92	0.81
Thiamin B1 (mg/day)	2.34 ± 0.57	1.72 ± 0.22	0.35	0.41
Niacin B3 (mg/day)	23.89 ± 5.78	17.84±3.97	0.63	0.51
Folate (µg/day)	410.81±14.33	302.89 ± 51.24	0.51	0.31
Carbohydrate (g/day)	380.26 ± 54.02	622.02±10.13	0.60	0.31
Potassium (mg/day)	5292.19±80.37	4034.62±36.03	0.43	0.22
Calcium (mg/day)	1234.81±12.91	861.700±16.69	0.81	0.12
Phosphorus (mg/day)	1800.16 ± 10.80	1075.22±79.80	0.53	0.72

*T test, **Mean±SD

Table 3. Dietary intake in different phenotypes of PCOS

Variable** Phenotype of PCOS			S	P value*	P value adjusted
	H+PCO	H+PCO+M	M+PCO		for BMI and age
	(n= 53)	(n= 66)	(n= 57)		
Energy (Kcal/ day)	2454.2±82.4	2600.8±51.09	$2346.80{\pm}4.08$	<0.001£€¥	< 0.001
Protein (g/day)	85.56 ± 81.12	91.69±82.38	65.96 ± 68.30	0.41	0.63
Fat (g/day)	119.27±55.46	89.81±45.38	77.56±49.59	<0.001£€¥	< 0.001
Saturated fatty acids (g/day)	29.80±31.67	45.06±10.31	21.75±15.79	<0.001£€¥	< 0.001
Polyunsaturated fatty acids (g/day)	41.54±12.97	25.34±13.13	25.11±15.25	<0.001£€¥	< 0.001
Linoleic acid (g/day)	39.40±12.46	45.90±16.17	23.59±14.59	0.52	0.35
EPA (g/day)	0.01 ± 0.01	0.05 ± 0.01	0.007 ± 0.01	0.09	0.06
Sodium (mg/day)	1993.41±15.71	2523±38.12	2907.27±34.58	0.31	0.58
Iron (mg/day)	21.94±17.26	28.46 ± 34.87	21.27±24.82	0.38	0.41
Magnesium (mg/day)	431.99±51.47	$450.84{\pm}64.62$	362.05 ± 37.70	0.83	0.96
Zinc (mg/day)	9.74±7.66	12.79 ± 2.96	$9.080 \pm .76$	0.61	0.82
Manganese (mg/day)	7.74 ± 4.03	10.69 ± 0.12	$7.17 \pm .20$	0.21	0.35
Fluoride (µg/day)	6454.85±47.21	5030.95±39.90	7064.76±84.99	0.63	0.41
Iodine (µg/day)	$0.2 \pm .01$	0.38 ± 0.12	$.08 \pm .01$	0.52	0.51
Vitamin A (µg/day)	$1962.76 \pm$	3512.09±12.02	2473.02±52.84	0.35	0.41
	16.38				
Vitamin E (mg/day)	4.30±0.36	4.19±0.66	3.44 ± 0.42	0.31	0.51
Thiamin B1 (mg/day)	2.35 ± 0.40	2.78 ± 0.70	1.82 ± 0.34	0.83	0.62
Niacin B3 (mg/day)	21.09 ± 0.18	30.58±0.91	18.95±0.16	0.34	0.91
Folate (µg/day)	557±74.20	591.36±64.42	377.16±25.36	0.48	0.72
Carbohydrate (g/day)	433.41±57.31	435.06±63.39	345.17±39.45	0.64	0.51
Potassium (mg/day)	5353.94±53.53	6128.09±10.01	4292.63±34.42	0.76	0.43
Calcium (mg/day)	1386.78±18.24	1238.75±11.09	1089.47±79.86	0.62	0.95
Phosphorus (mg/day)	1824.75 ± 16.02	1238.75±11.09	1089.46±79.86	0.52	0.81

*ANOVA, **Mean±SD

 \pounds P<0.05 between H+PCO and H+PCO+M phenotype € P<0.05 between H+PCO and M+PCO phenotype

¥ P<0.05 between H+PCO+M and M+PCO phenotype

Variable **	PCOS	Control	P value *	P value adjusted
	(n=168)	(n=160)		for BMI and age
Physical health	16.32±3.62	16.66±3.01	0.38	0.62
Exercise and fitness	12.05 ± 3.52	15.42 ± 4.82	< 0.001	0.04
Weight control and nutrition	12.49 ± 4.02	16.54 ± 3.98	< 0.001	0.03
Illness prevention	15.82 ± 3.38	16.23±4.49	0.45	0.59
Psychological health	12.90 ± 4.22	16.34 ± 3.36	< 0.001	0.01
Spiritual health	17.62 ± 3.45	18.02 ± 4.28	0.69	0.71
Social health	17.56 ± 4.62	17.38 ± 5.01	0.53	0.73
Drug and alcohol avoidance	16.31±3.42	15.95 ± 3.41	0.42	0.59
Accident prevention	17.43 ± 3.42	17.56 ± 3.51	0.36	0.48
Environmental health	17.36±2.49	17.42±4.32	0.74	0.80

Table 4. Comparison of PCOS and control groups based on the domains of lifestyle

*T test, **Mean±SD

Table 5. The scores of lifestyles in different phenotypes of PCOS

Variable	Phenotype of PCOS			P value*	P value adjusted
	H+PCO	H+PCO+M	M+PCO		for BMI and age
	(n= 53)	(n= 66)	(n= 57)		
Physical health	16.02 ± 3.44	16.56 ± 3.53	16.45 ± 3.08	0.65	0.55
Exercise and fitness	12.34 ± 3.34	11.02 ± 2.34	13.40 ± 3.29	<0.001£€¥	< 0.001
Weight control and	11.93 ± 2.43	10.42 ± 2.35	13.39 ± 3.86	<0.001£€¥	< 0.001
nutrition					
Illness prevention	15.04 ± 3.22	14.93 ± 4.24	15.42 ± 4.42	0.45	0.31
Psychological health	12.42 ± 1.32	10.56 ± 2.94	13.20 ± 3.82	<0.001£€¥	< 0.001
Spiritual health	17.53 ± 7.34	16.92 ± 6.42	17.83 ± 5.01	0.33	0.25
Social health	17.32 ± 6.45	17.24 ± 3.56	17.45 ± 5.92	0.45	0.35
Drug and alcohol avoidance	16.42 ± 5.62	16.02 ± 4.41	16.93 ± 4.32	0.56	0.42
Accident prevention	17.05 ± 3.56	16.92 ± 2.39	17.21±4.35	0.71	0.65
Environmental health	17.32 ± 4.23	17.25 ± 1.95	17.28 ± 4.62	0.93	0.83

*ANOVA, **Mean±SD

 \pounds P<0.05 between H+PCO and H+PCO+M phenotype

€ P<0.05 between H+PCO and M+PCO phenotype

¥ P<0.05 between H+PCO+M and M+PCO phenotype