

## QUANTITATIVE ASSESSMENT OF NUTRITION IN PATIENTS WITH THE POLYCYSTIC OVARY SYNDROME (PCOS)

Małgorzata Szczuko<sup>1\*</sup>, Magdalena Skowronek<sup>1</sup>, Marta Zapalowska-Chwyć<sup>2</sup>, Andrzej Starczewski<sup>2</sup>

<sup>1</sup>Department of Biochemistry and Human Nutrition, Pomeranian Medical University in Szczecin, Poland

<sup>2</sup>Clinic of Gynecology and Urogynecology, Pomeranian Medical University in Szczecin, Poland

### ABSTRACT

**Background.** PCOS (polycystic ovary syndrome) is called a pathology of the XX century and affects at least 10-15% women of childbearing age. The therapy involves pharmacotherapy of hormonal imbalance, as well as the change of lifestyle, including the diet.

**Objective.** Performing the quantitative assessment of components of diets of women with PCOS, comparing the results with current dietary standards for Polish people and defining dietary requirements for the patients.

**Material and Methods.** The study was performed on 54 women of childbearing age (average age  $26.03 \pm 5.52$ ) with PCOS syndrome diagnosed according to on the Rotterdam criteria. Anthropometric measurements of the patients were made and BMI and WHR calculated. Quantitative assessment of women's diets was performed based on the analysis of 3-day food diaries and food records taken from the previous 24h with the interview method. The data were introduced to a dietary software DIETA 5.0, calculating the average intake of the energy, nutrients, vitamins, minerals, cholesterol and dietary fibre. The obtained results were compared to Polish dietary guidelines.

**Results.** Examined group was characterized by increased waist circumference ( $98.71 \pm 13.6$  cm) and an average WHR was  $0.92 \pm 0.08$ . An increased average value of BMI was also shown ( $28.91 \pm 5.54$  kg/m<sup>2</sup>). The patients consumed, on average,  $1952.5 \pm 472.7$  kcal daily, and the risk of insufficient intake of protein was determined in 36.7% of examined women. The highest risk of deficiency in minerals in women with PCOS was related to calcium (634 mg), potassium (3493 mg) and magnesium (250.1 mg), whereas with reference to vitamins deficiency as much as 70% of tested women were at risk of insufficient intake of folic acid, 36.7% of them - vitamin C, and 26.7% - vitamin B12. The average consumption of vitamin D was at the level of 3.4 µg. Test group was characterized by excessive average consumption of total fat (50%), SFA (70.4%) and saccharose (50%). The percentage of people with excessive average intake of cholesterol was at the level of 40.74%. As much as 83.3% patients consumed too low amounts of dietary fibre in their diets (<25g).

**Conclusions.** In diet therapy of women with PCOS there should be higher intake of folic acid, vitamins D and C, cobalamin, dietary fibre and calcium. The consumption of total fats, saturated fatty acids and cholesterol should be reduced, as through facilitating the development of diabetes and cardio-vascular diseases, they affect the dysfunction of ovaries. The diet of some of the patients should be also supplemented by potassium, magnesium and zinc. The introduction of a properly balanced diet should be the key in the treatment of women with PCOS diagnosed according to Rotterdam criteria.

**Key words:** *polycystic ovary syndrome, PCOS, nutrition, diet, nutrients, nutritional deficiencies*

### STRESZCZENIE

**Wstęp.** Syndrom policystycznych jajników (PCOS) nazywany jest patologią XX wieku i dotyka co najmniej 10–15% kobiet w wieku reprodukcyjnym. Terapia dotyczy zarówno leczenia farmakologicznego zaburzeń hormonalnych, metabolicznych jak również zmiany stylu życia, w tym sposobu żywienia.

**Cel.** Ocena składu ilościowego jadłospisów kobiet z PCOS, porównanie go z obowiązującymi normami żywienia dla ludności polskiej oraz sprecyzowanie zaleceń żywieniowych dla pacjentek.

**Material i metody.** Badaniami objęto 54 kobiety w wieku rozrodczym, z rozpoznaniem, według kryteriów Rotterdamskich, zespołem PCO (średni wiek to  $26,03 \pm 5,52$  lat). Wykonano pomiary antropometryczne oraz obliczono wskaźniki BMI i WHR. Oceny ilościowej sposobu żywienia kobiet dokonano w oparciu o analizę trzydniowych dzienniczek oraz jadłospisu zebranego metodą wywiadu o spożyciu z ostatnich 24 godzin. Dane wprowadzono do programu dietetycznego DIETA 5.0, obliczając średnią podaż energii, składników odżywczych, witamin, składników mineralnych, cholesterolu oraz błonnika. Uzyskane wyniki porównano z obowiązującymi w Polsce normami żywienia.

\*Corresponding author: Małgorzata Szczuko, Department of Biochemistry and Human Nutrition, Pomeranian Medical University in Szczecin, Broniewskiego street 24; 71-460 Szczecin; tel. +48 91 4414810; fax. +48 91 4414807, e-mail: malgorzata.szczuko@pum.edu.pl;

**Wyniki.** Badana grupa charakteryzowała się zwiększonym obwodem pasa  $98,71 \pm 13,6$  cm, a średnia WHR była równa  $0,92 \pm 0,08$ . Wykazano zwiększoną średnią wartość wskaźnika BMI ( $28,91 \pm 5,54$  kg/m<sup>2</sup>). Pacjentki spożywały średnio  $1952,5 \pm 472,7$  kcal dziennie, a zagrożenie niedostatecznym spożyciem białka stwierdzono u 36,7% badanych kobiet. Największe ryzyko wystąpienia deficytu składników mineralnych u kobiet z PCOS dotyczyło wapnia (634 mg), potasu (3493mg), magnezu (250,1 mg) natomiast wśród witamin aż 70% kobiet badanych było zagrożonych niewystarczającym spożyciem folianów, 36,7% niedoborem witaminy C a 26,7% witaminą B12. Średnie spożycie witaminy D kształtowało się na poziomie 3,4 µg. Badana grupa charakteryzowała się nadmiernym średnim spożyciem tłuszczu ogółem (50%) NKT (70.4%) i sacharozy (50%). Odsetek osób z nadmiernym średnim poborem cholesterolu był na poziomie 40.74%. Aż 83.3% pacjentek miało zbyt niską podaż błonnika w diecie (<25g).

**Wnioski.** W dietoterapii kobiet z PCOS należy zwiększyć podaż folianów, witaminy D i C, kobalaminy, błonnika oraz wapnia. Powinno ograniczyć się spożycie tłuszczu ogółem, nasyconych kwasów tłuszczowych i cholesterolu które pogłębiając rozwój cukrzycy i chorób sercowo-naczyniowych wpływają na dysfunkcję jajnika. Dietę części pacjentek z PCOS należałoby również uzupełnić w potas, magnez, cynk. Wprowadzenie prawidłowo zbilansowanej diety powinno być kluczem w leczeniu kobiet z PCOS rozpoznawanych kryteriami Rotterdamskimi.

**Słowa kluczowe:** zespół policystycznych jajników, PCOS, żywienie, dieta, składniki pokarmowe, niedobory żywieniowe

## INTRODUCTION

It is estimated that PCOS occurs in ca. 10-15% women of childbearing age [12]. It is called a pathology of the XX century, since it can cause numerous health issues. The symptoms occurring in this syndrome are mainly related to lower fertility, menstruation disorders and hyperandrogenism, which is manifested by acne and hirsutism.

Several concepts of the development of PCO syndrome are considered, including ovarian, insulin-dependent and gonadotropic concepts. Improper dietary habits are often noticed among the women with PCO syndrome, and those are related to the intake of food of low nutritional value and high energy content, which seems to be in accordance with the concept of insulin-dependent cause of PCOS. As reported in the literature, insulin increases the production of androgens, what affects ovarian theca cells, and leads to higher concentration of those hormones in women with PCOS, as compared to healthy ones [26]. It has been proposed that insulin activates the expression of CYP-17 gene, which takes part in androgens production [40]. Insulin sensitizes ovarian granulosa cells to LH too quickly, thus stops the maturation of the follicle. LH acting synergistically with insulin leads to the activation of androstenedione synthesis and hormonal imbalance [40].

It also seems that insulin indirectly affects steroid genesis via insulin-like growth factor (IGF-1). Its elevated concentration in follicle fluid contributes, together with insulin, to increased androgenesis, as well as to premature ovarian follicle atresia. The reciprocal effect of insulin-like growth factor (IGF-1) strengthens the above mentioned symptoms [14]. Moreover, the studies showed that the increased concentration of testosterone in blood of women with PCOS is also affected by decreased production of SHBG (sex hormone binding globulin) by the liver, caused by inhibiting activity of insulin [19].

Women eating habits are determined, among others, by the habits taken from family home, economic status, culture (religion, tradition), education, origin and age. The relation between level of nutritional education and the risk of breast cancer was shown by [17]. According to *Chavarro et al.* [10], fertility can be improved by lowering the consumption of trans fats with simultaneous increase in consumption of monounsaturated fatty acids, limiting the intake of animal proteins in exchange for plant proteins, diet rich in dietary fibre and products with low glycemic index (GI), consumption of full fat dairy products and proper amount of minerals and vitamins in a diet. In studies of *Chavarro et al.* [11] it was shown that both the reduction in animal proteins in favour of plant protein and the increase in the consumption of plant protein resulted in lowering the risk of infertility caused by ovulation disorders. This fact justifies reduced secretion of insulin and higher sensitivity to it after the consumption of products being the source of plant proteins. Some sources report the consumption of plant proteins influences the decrease of the concentration of insulin-like growth factor 1 (IGF-1), which is involved in the development of PCOS [33]. However, no negative effect on fertility was shown after the consumption of fish and eggs proteins. Taking into consideration other risk factors, the increase in the consumption of trans fatty acids and carbohydrates by 2% of the energy intake, instead of polyunsaturated fatty acids (omega 6), causes increased risk of infertility among women by 76% and 73%, respectively [11] as well as obesity increases risk of hypercholesterolemia in women in reproductive age [31]. Metabolic and hormonal parameters in the group of women with PCOS are improved by cis fatty acids, in comparison to trans fatty acids, which was proved in the study of *Kasim-Karakas et al.* [20]. Trans isomers of fatty acids lower the activity of peroxisome proliferator-activated receptors  $\gamma$  (PPAR), leading to increased ovulation disorders [7]. The consumption

of dietary fibre is equally important. As shown in the studies of [13], the increased consumption of dietary fibre by 10g, in group of women aged 32 and above, lowered the risk of fertility disorders due to ovulation disorders by 44%. It is also known that vitamin B6 influences the maintenance of the proper concentration of progesterone in blood, therefore proper consumption of the products rich in this vitamin (lean meat, milk, eggs, grain products, dry legumes) is justified. The deficiency in vitamin B12 can contribute to the disorders in ovulation and embryo implantation. This vitamin is essential to produce red and white blood cells. In diet, the sources of vitamin B12 are products of animal origin, mainly eggs, meat and fish. Thus women with PCOS being on a vegetarian diet should take supplements being the source of group B vitamins, due to risk in their deficiencies [25]. Inositol also belongs to the group of B vitamins. There are nine stereoisomers of inositol (chemical compounds, which atoms are bounded in the same sequences differing by spatial arrangement), including myo-inositol (MYO) and D-chiro-inositol (DCI). Everyday diet supplies ca. 1g of inositol, mainly in form of myo-inositol. Data from the literature confirm, that in women with PCO syndrome the insulin resistance is affected by the deficiency in inositol. The intake of myo-inositol sensitizes the tissues to insulin, thus leading to decrease in insulin resistance. Inositol influences the process of maturation of an egg cell, because it lowers the concentration of LH, prolactin and testosterone [22]. Inositol phosphates are the molecules built from myo-inositol. They are classified as auxiliary antioxidants, the so called synergistic antioxidants. They do not directly stop the oxidation chain reaction, but they can enhance the activity of main antioxidants. The sources of inositol phosphate are dry legumes, seeds of oil plants and grains.

Proper intake of antioxidants in a diet, including vitamins C and E,  $\beta$ -carotene, zinc, copper, selenium and coenzyme Q10, protects against free radicals (lipid peroxides). Their adverse effect on the organism is based on triggering oxidative stress, and, as a consequence, the damage of, among others, cellular membranes [28]. Additionally, vitamin E takes part in reproductive processes and regulates the activity of internal secretion. Low concentration of vitamin E (its sources are plant oils, leafy vegetables, legumes) correlates with ovulation disorders [1]. Proper intake of vitamin C (wild rose fruits, cabbages, berries, yellow and green vegetables and fruits) and bioflavonoids can also prevent miscarriages. Among the antioxidants used in PCOS treatment the commonly mentioned is also coenzyme Q10 (ubiquinone), which takes part in ATP metabolism in mitochondria as a catalyst of metabolic processes [23]. Ubiquinone synthesis requires vitamin B12 and folic acids, which are often deficient in diets

of women with PCOS. Moreover, folic acid takes part in homocysteine metabolism [30]. It has been proved that too high concentration of homocystein in blood lowers reproductive capabilities and leads to pregnancy complications. The deficiency in folic acid correlates with higher risk of high blood pressure and cardio-vascular diseases, which coexist in PCOS [36]. Its sources are green vegetables, dry legumes, wholegrain products, liver and eggs. Considering mineral elements, an appropriate consumption of zinc is important, which is essential for proper metabolism of estrogens, progesterone and androgens affecting ovulation and the development of an embryo [37]. Similarly, the deficiency in magnesium can influence the reduction of progesterone level and thus cause menstruation disorders. Numerous dietary mistakes of women with PCOS often cause the extensive development of fatty tissue, especially the visceral one, and difficulties with body mass reduction. Polycystic ovary syndrome increases the risk of type 2 diabetes, blood hypertension, cardio-vascular diseases and cancer of reproductive organs. According to the current knowledge, 35%-50% of women with PCOS also have insulin resistance and/or type 2 diabetes. Moreover, the studies report that in every fourth obese women with PCOS the type 2 diabetes and insulin resistance occur before the age of 30 [27]. It is known, that the development of the disease is influenced by genetic, hormonal and environmental factors. There are studies confirming that PCOS is family-related and occurs more often in women having at least one member of the family suffering from type 2 diabetes [15]. The study determining the molecular causes of PCOS development are still being performed. In current literature there are no reports related to the qualitative and quantitative assessment of diets of women with PCOS enabling to apply complex diet therapy.

## MATERIALS AND METHODS

### *Tested group*

Screening tests were performed in the Clinic of Gynecology and Urogynecology, Pomeranian Medical University (PMU) in the Independent Public Clinic Hospital No. 1 in Police.

The selection of women for the study was deliberate. PCOS was diagnosed according to Rotterdam's criteria from 2003, which require the diagnosis of 2 out of 3 following criteria: rare ovulations or lack of thereof, and/or biochemical symptoms of hyperandrogenism, and/or image of polycystic ovaries in USG (polycystic ovaries morphology in transvaginal USG - the presence of 12 follicles or more in one or both ovaries, and/or increased volume of ovary >10ml). The image was obtained using Ultrasound Voluson 730 (GE,

Switzerland). Criteria for exclusion from the test were: pregnancy, age above 40, change in dietary habits just before the study (3 months) and co-existence of other diseases linked to hyperandrogenism (Cushing's syndrome, tumours releasing androgens, congenital or acquired adrenal hyperplasia). The research has been approved by the Bioethical Commission of the Pomeranian Medical University in Szczecin, No. KB-0012/134/12, with the annex to the permission No. KB-0012/36/14.

#### Assessment of nutritional status

To assess nutritional status of the patients the following anthropometric tests were used: body weight ( $\pm 0.1$  kg), body height, waist circumference and hip circumference - using anthropometric measuring tape ( $\pm 0.5$  cm). On the basis of those data the Body Mass Index (BMI - Body Mass Index) was calculated and the type of body built (WHR - Waist Hip Ratio) was determined [38].

#### Quantitative dietary assessment

The information on the consumption of products and meals were collected using two methods: food records method (food diaries) from 3 days and one-day food record from the last 24-hour dietary interview. Records from the food diary and the interview included: ingredients, quantity, mode of preparation and time of consumption of every meal. Menus were taken from two weekdays (Thursday and Friday) and two weekend days (Saturday and Sunday). Altogether 216 menus from women with PCOS were analysed. The sizes of consumed portions were determined according to the "Album of photographs of food products and dishes" of the Food and Nutrition Institute [32]. Next, the energy and nutrients were calculated using computer software Dieta 5 (National Food and Nutrition Institute, Warsaw, Poland), whose database was based on the "Tables of the contents and nutritional value of food" [21].

#### Statistical analysis

The results were statistically analyzed using the software package Statistica 10.0 (Statsoft, Tulsa, Oklahoma, USA). The arithmetical mean, standard deviation and the significance of differences were calculated using ANOVA.

## RESULTS

Nutritional status: In the group of 33 women with PCOS the average age of patients was  $26.31 \pm 5.52$ , and the average body weight was  $80.98 \pm 16.06$  kg. The average BMI value was  $29.16 \pm 5.8$  kg/m<sup>2</sup>. The characteristics of anthropometric parameters of tested women are presented in Table 1.

Table 1. Anthropometric characteristics of the study group

Parameter	$\bar{x}$	SD
Age (years)	26.31	5.52
Waist circumference (cm)	99.18	14.82
Hip circumference (cm)	108.45	9.39
Body weight (kg)	80.98	16.06
Height (m)	1.67	0.06
BMI - Body Mass Index (kg/m <sup>2</sup> )	29.16	5.8
WHR- Waist-Hip Ratio (cm/cm)	0.91	0.08

SD- Standard Deviation

Table 2. The average content of nutrients in diets

Discriminant	Average content	$\pm$ SD	Range
Energy (kcal)	1952.5	472.7	1353.8-2956.8
Protein (g)	76.1	18.2	46.9-106.8
Fat(g)	72.9	21.9	38.4-122.2
SFA* (g)	26.1	8.3	13.4-47.6
MUFA** (g)	27.3	10.9	13.3-61.9
PUFA*** (g)	12.6	4.9	5.4-28.267
Cholesterol (mg)	301.5	172.2	90.4-1044.7
Carbohydrates (g)	265.0	82.2	170.9-428.6
Saccharose (g)	43.0	24.6	10.6-103.2
Dietary fibre (g)	19.7	7.5	9.7-44.8
Vitamin A ( $\mu$ g)	954.4	356.9	358.7-1682.7
Vitamin D ( $\mu$ g)	3.4	3.8	0.5-21.2
Vitamin E (mg)	11.6	4.6	3.8-26.4
Vitamin B1 (mg)	1.4	1.1	0.6-7.2
Vitamin B2 (mg)	1.6	0.6	0.7-4.3
Niacin (mg)	4.3	15.4	9.1-26.1
Vitamin B6 (mg)	2.1	0.8	0.9-4.1
Folate ( $\mu$ g)	271.8	79.7	123.4-458.0
Vitamin B12 ( $\mu$ g)	3.3	2.2	0.9-13.0
Vitamin C (mg)	98.3	77.1	25.1-421.7
Sodium (mg)	2903.1	805.8	1329.7-5073.8
Potassium (mg)	3493.4	1724.5	1559.0-8430.1
Calcium (mg)	634.3	228.9	301.9-1278.2
Phosphorus (mg)	1392.9	502.5	665.1-2852.5
Magnesium (mg)	250.9	85.9	176.8-583.9
Iron (mg)	11.7	4.4	6.5-23.3
Zinc (mg)	9.0	2.1	5.3- 14.8
Copper (mg)	1.1	0.4	2.4-0.6

\* SFA-saturated fatty acids;

\*\*MUFA-monounsaturated fatty acids;

\*\*\*PUFA-polyunsaturated fatty acids.

The largest percentage of the patients (76%) were women with BMI above the standard (BMI  $\geq 25$  kg/m<sup>2</sup>). The group of obese women (BMI  $\geq 30$  kg/m<sup>2</sup>)

comprised as much as 39% of studied population. Among the participants of the study there were none with BMI showing underweight (BMI < 18,5 kg/m<sup>2</sup>). Significant percentage of tested women (73%) had waist circumference  $\geq$  88 cm, which shows on high risk of metabolic syndrome occurrence among the respondents. In the test group of overweight (BMI  $\geq$  25 kg/m<sup>2</sup>) and obese (BMI  $\geq$  30 kg/m<sup>2</sup>) women, a large majority, i.e. 95.65%, represented android body type.

The results of the study provide the information about the average consumption of energy at the level of 1952.5 $\pm$  472.7 kcal, and the average content of nutrients in 216 diets are shown in Table 2.

After performing the quantitative assessment of the intake of nutrients using AI level it was observed that there was high probability of insufficient intake of calcium (634 mg), potassium (3493 mg) and vitamin D (3.4  $\mu$ g) (Table. 3).

Table 3. The assessment of the consumption using AI level

Discriminant	AI level	Average intake $\pm$ SD
Sodium (mg)	1500	2903.1 $\pm$ 805.8
Potassium (mg)	4700	3493 $\pm$ 1724.5
Calcium (mg)	1000	634 $\pm$ 228.9
Iron (mg)	8	11.6 $\pm$ 4.4
Vitamin D ( $\mu$ g)	5	3.4 $\pm$ 3.8
Vitamin E (mg)	8	11.6 $\pm$ 4.6

The evaluation of the consumption in the test group using Estimated Average Requirements (EAR) threshold showed, that 70% of examined women were at risk of insufficient intake of folic acid. In 36.7% of examined patients there was a risk of insufficient

consumption of protein, but also vitamin C (36.7%), vitamin B12 (26.7%) and magnesium (23.3%). The results showed low risk of deficiency in niacin in 13.3% of examined women (Table 4).

The assessment of the consumption of other nutrients showed, that the intake of total fat, and consequently also SFA, was too high in 70.4% of women. Too high intake of cholesterol was observed in 40.74% of test group. Too low consumption of total carbohydrates was not determined, however, 50% of examined women consumed excessive amounts of saccharose (>40g/day). It was also observed that 83.3% of patients were characterized by too low intake of dietary fibre in their diets (<25 g) (Table 5).

Table 4. The assessment of the consumption using EAR threshold

Determinant	Standard level		Percentage of women	
	EAR	UL	<EAR	>UL
Protein (g)	80	N/A	36.7	-
Phosphorus (mg)	580	4000	0	0
Magnesium (mg)	255	350 <sup>1</sup>	23.3	0
Zinc (mg)	6.8	N/A	10,0	-
Copper (mg)	0.7	N/A	6.7	-
Vitamin A ( $\mu$ g)	500	3000	10.0	0
Vitamin B1 (mg)	0.9	N/A	10.0	-
Vitamin B2 (mg)	0.9	N/A	3.3	-
Niacin (mg)	11	35	13.3	0
Vitamin B6 (mg)	1.1	100	6.7	0
Folate ( $\mu$ g)	320	1000	70.0	0
Vitamin B12 ( $\mu$ g)	2.0	N/A	26.7	-
Vitamin C (mg)	60	2000	36.7	0

Table 5. Percentage of the realisation of the standard nutrient requirements

Determinant	Applied standard	Percentage of women >standard	Percentage of women <standard
Energy (kcal)	1802.5-2846.4	13.0	51.85
Fat (%)	<30 (>35)**	18.82	(50,0)**
SFA* (%)	<10	70,4	29,6
Cholesterol (mg)	200-300	40.74	37.04
Carbohydrates (%)	<45	92,6	7.4
Saccharose (g)	40	50	50
Dietary fibre (g)	25	16.7	83.3

\*SFA-saturated fatty acids; \*\*intake more than 35% energy from fat

## DISCUSSION

Randomized studies carried out in Australia [35], London [18] or in Italy [2] confirmed that overweight or obesity and the excess of abdominal fatty tissue around waist are often the characteristics of women with PCOS. BMI values in our study were similar to

those observed by other researchers: 29.7 $\pm$ 4.8 kg/m<sup>2</sup> [13] and 27.4 $\pm$ 7.3 kg/m<sup>2</sup> [5]. Average WHR value in our study was higher (0.92 $\pm$ 0.08) in comparison to the results of *Wright et al.* [39] (0.837 $\pm$ 0.097), which could be caused by the selection of the patients according to different criteria (one of the required symptoms was clinical or biochemical proof of hyperandrogenism).

The available literature does not provide the assessment of the diet of women with PCOS.

In the meantime, since the introduction of Rotterdam Criteria (2003) the increasing number of women with normal androgen levels is qualified for this disease. In these women were found polycystic ovary syndrome based on ultrasound and menstrual disorders. The authors of this study show that one of the causes of ovarian dysfunction PCOS (except hormonal disorders) is the poor diet of women.

In this study the energy requirements were properly fulfilled. Only 13% of the examined women consumed higher amount of energy than the required intake. Even if we assume, that some of women with PCOS underestimated their dietary ratios, which was reported in obese people *Chandon & Wansik* [9], then we can conclude that the problem is related to the expenditure of consumed energy, i.e. too little physical activity of the patients. The percentage of the energy coming from protein in own study and the studies of *Altieri* and *Wright* [2,39] was similar, and amounted to 15.59, 14.61 and 16.62, respectively. The percentage of the energy in diets coming from carbohydrates was appropriate, unfortunately the major share came from simple sugars, including saccharose. They contribute to the decrease in the HDL cholesterol fraction, the increase in the concentration of triglycerides and insulin resistance. The average content of fat in diets was not high, but 50% of examined women consumed excessive amounts of fat. When analyzing its quality, we determined high consumption of SFA and cholesterol, with simultaneous low intake of dietary fibre, which increases the risk of metabolic diseases related to dyslipidemia, cardio-vascular diseases and type II diabetes. It was previously reported in the literature that there is a positive correlation between the concentration of androstendion, testosterone and free testosterone in blood and the consumption of total fat, SFA, MUFA and cholesterol. Moreover, cholesterol is a precursor of steroid hormones, including androgens distorting hormonal balance in polycystic ovary syndrome.

Despite the fact that PCOS was recognized 80 years ago, no studies have been carried out on the components of diets of women with PCOS, considering the content of vitamins and minerals. The intake of antioxidative vitamins (A, C, E) was at an appropriate level, therefore their deficiency should not occur in PCOS. However, the data from the literature suggest, that the excess in vitamin A may contribute to menstrual disorders and cause scanty menstruations. On the other hand, vitamin D was consumed in too small amounts. Proper intake of this vitamin is crucial, because the studies showed the correlation between its deficiency and insulin resistance, hirsutism and infertility, which are characteristic symptoms of PCOS

[34]. The deficiency in vitamin D were reported in people with abdominal obesity, diabetics and in the group of people at risk of insulin release disorders. It was also shown that, due to the presence of VDR (vitamin D receptor) in epithelial cells of uterine glands, the administration of vitamin D significantly affected the thickness of endometrium in women with PCOS, which contributed to higher chances of success of intrauterine insemination [3]. In this study we observed insufficient consumption of group B vitamins, especially folic acid. Only 30% of women consumed it at EAR. In the test group the highest deficiencies were noted in case of folic acid - average intake 271  $\mu\text{g}$  (EAR 320  $\mu\text{g}$ ). Folic acid is one of the coenzymes taking part in homocysteine metabolism. Its deficiency inhibits proliferation and growth of cells on nucleic acids level and leads to the increase in the concentration of homocysteine in blood. It was shown that hyperhomocysteinaemia has a negative effect on reproduction, leads to pregnancy complications and disorders in foetus development. High concentration of homocysteine in follicular fluid may distort the interaction between an ovum and a sperm cell, decreasing the chance of fertilization. Moreover, the excessive amount of homocysteine disturbs the mechanism of implantation of fertilized egg and has a negative influence of embryogenesis, which can result in the inhibition of foetus development, embryonic death and miscarriage [36]. High deficiencies in folic acid in women can lead to disorders in the occurrence of proper mucous symptom. Moreover, proper intake of folic acid contributes to the increase in the number of maturing egg cells [29]. Studies performed by other authors suggest that the frequency of ovulations and births may be higher with the intake of folic acid at the level of 700  $\mu\text{g}/\text{day}$  (with the general daily intake of 400  $\mu\text{g}/\text{day}$ ) [10].

Considering the consumption of mineral elements, we noted too low intake of calcium ( $634.3 \pm 228.9$  mg) and potassium ( $3493.4 \pm 502.5$  mg) in the diets of women with PCOS. Proper contents of calcium is very important, because its deficiency has an inhibiting effect on the maturation of ovary follicles and lowers the number of developing follicles [8]. Moreover, crystal structure studies not only revealed calcium-binding sites in the human SHBG but also demonstrated that SHBG is a zinc binding protein [4]. It had previously been shown that the presence of calcium is essential for maintaining homodimer stability and steroid-binding activity [6], but the presence of a zinc molecule was found to help orient the exposed loop over the entrance to the steroid-binding site and to alter the binding affinity of estrogens versus androgens [16]. The contents of other nutrients in the study (magnesium 300.9 mg, iron 11.7 mg, zinc 9.0 mg, copper 1.1 mg) did not pose the risk

of deficiencies, however, as far as dietary procedure is concerned, it seems proper to supplement the diet with zinc. The intake of sodium was too high. Sodium, together with chlorine, increases blood pressure and lowers insulin sensitivity, which is crucial for frequent coexistence of type II diabetes and high blood pressure in PCOS [24]. Thus it seems reasonable to limit the consumption of salt and products containing it.

## CONCLUSIONS

1. Too high intake of total fat, saturated fatty acids, cholesterol and saccharose, and low consumption of dietary fibre observed in the study, may contribute to ovaries dysfunction through down regulation of metabolic processes.
2. It may be necessary to supplement the diets of some women with PCOS with potassium, zinc and magnesium. Because of deficiencies and excesses of nutrients in the diet, come to mind also additional implications:
3. One of the most important dietary reasons for disorders in reproductive processes is the deficiency of folic acid in a diet.
4. Proper intake of vitamin D and calcium may positively affect the improvement of menstruation cycle regularity and maturation of ovarian follicle.
5. High consumption of cholesterol, as a substrate for androgens' synthesis, may contribute to hormonal imbalance in PCO syndrome.

## Conflict of interest

All authors declare no conflict of interest. Informed consent was obtained from all individual participants included in the study.

## REFERENCES

1. *Al-Azemi M.K., Omu A.E., Fatnikun T., Mannazhath N., Abraham S.*: Factors contributing to gender differences in serum retinol and alpha-tocopherol in infertile couples. *Reprod Biomed* 2009, 19, 583-590.
2. *Altieri P., Cavazza C., Pasqui F., Morselli A-M., Gambiner 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(1), 52-59.
3. *Asadi M., Matin N., Frootan M., Mohamadpour J., Qorbani M., Tanha F.D.*: Vitamin D improves endometrial thickness in PCOS women who need intrauterine insemination: a randomized double-blind placebo-controlled trial. *Arch Gynecol Obstet* 2014, 289(4), 865-870.
4. *Avvakumov G.V., Muller Y.A., Hammond G.L.*: Steroid-binding specificity of human sex hormone-binding globulin is influenced by occupancy of a zinc-binding site. *J Biol Chem* 2000, 275, 25920-25925.
5. *Barr S., Hart K., Reeves S., Sharp K., Jeanes Y.M.*: Habitual dietary intake, eating pattern and physical activity of women with polycystic ovary syndrome. *Eur J Clin Nutr* 2011, 65(10), 126-132.
6. *Bocchinfuso W.P., Hammond G.L.*: Steroid-binding and dimerization domains of human sex hormone-binding globulin partially overlap: steroids and Ca<sup>2+</sup> stabilize dimer formation. *Biochemistry* 1994, 33, 10622-10629.
7. *Brown J.M., Boysen M.S., Jensen S-S., Morrison R.F., Storkson J., Lea-Currie R., Pariza M., Mandrup S., McIntosh M.K.*: Isomer-specific regulation of metabolism and PPARgamma signaling by CLA in human preadipocytes. *J Lipid Res* 2003, 44, 1287-1300.
8. *Brzozowska M., Karowicz-Blińska A.*: The role of vitamin D deficiency in the pathophysiology of disorders of polycystic ovary syndrome. *Ginekol Pol* 2013, 84, 456-460 (in Polish).
9. *Chandon P., Wansik B.*: Is Obesity Caused by Calorie Underestimation? A Psychophysical Model of Meal Size Estimation. *J Mark Res* 2007, 84 (XLIV), 84-99.
10. *Chavarro J.E., Rich-Edwards J.W., Rosner B.A., Willatt W.C.*: Diet and life style in the prevention of ovulatory disorder infertility. *Obstet. Gynecol.*, 2007, 110, 1050-1058.
11. *Chavarro J.E., Rich-Edwards J.W., Rosner B.A., Willatt W.C.*: Protein intake and ovulatory infertility. *AM J Obstet Gynecol* 2008, 198, 210, e1-e7.
12. *Dniak-Nikolajew A.*: Zespół policystycznych jajników jako przyczyna niepłodności kobiecej [Polycystic ovary syndrome as a cause of female infertility]. *Położna Nauka i Praktyka* 2012, 1(17), 14-17 (in Polish).
13. *Douglas C.C., Gower B.A., Darnell B.E., Ovalle F., Oster R.A., Azziz R.*: Role of diet in the treatment of polycystic ovary syndrome. *Fertil Steril* 2006, 85, 679-688.
14. *Druckmann R., Rohr U.*: IGF-1 in gynecology and obstetrics: update 2002 *Maturitas.*, 2002, 41(1), 65-83.
15. *Elsheikh E., Genead R., Danielsson Ch., Wärdeell E., Andersson A., Kjaeldgaard A., Sundström E., Sutlu T., Grinnemo K.H., Sylvén Ch.*: Isolation and Characterization of the SSEA-1+ Progenitor Cells from the Human Embryonic Heart. *J Cytol Histol* 2011, 2, 4, 122.
16. *Hammond G.L.*: Diverse Roles for Sex Hormone-Binding Globulin in Reproduction. *Biol. Reprod* 2011, 85, 431-44.
17. *Hawrysz I., Krusińska B., Słowińska M.A., Wądołowska L., Czerwińska A., Maciej Biernacki M.*: Nutritional knowledge, diet quality and breast or lung cancer risk: a case-control study of adults from Warmia and Mazury region in Poland. *Rocz Panstw Zakł Hig* 2016;67(1):9-15, PMID: 26953576, <https://www.ncbi.nlm.nih.gov/pubmed/26953576>
18. *Humphreys L., Costarelli V.*: Implementation of diet and general life style advice among women with polycystic ovaria syndrome. *J R Soc Promo Health* 2008, 128(4), 190-195.
19. *Kalme T., Koistinen H., Loukovaara M., Koistinen R., Leinonen P.*: Comparative studies on the regulation of insulin-like growth factor-binding protein

- 1(IGFBP-1) and sex hormone – binding globulin (SHBG) production by insulin and insulin-like growth factors in human hepatoma cells. *J Steroid Biochem Mol Biol* 2003, 86, 197-200.
20. *Kasim-Karakas S.E., Almario R.U., Gregory L., Wong R., Todd H., Lasley B.L.*: Metabolic and endocrine effects of a polyunsaturated fatty acid-rich diet in polycystic ovary syndrome. *J Clin Endocrinol Metab* 2004, 89, 615-620.
21. *Kunachowicz H., Nadolna I., Przygoda B., Iwanow K.*: Tabele składu i wartości odżywczych żywności [Tables of composition and nutritional value of food]. Wyd. Lekarskie PZWL, Warszawa 2005 (in Polish).
22. *Larner J.*: D-chiro-inositol-its functional role in insulin action and its deficit in insulin resistance. *Int J Exp Diabetes Res* 2002, 3(1), 46-60.
23. *Matilla P., Kumpulainen J.*: Coenzymes Q<sub>9</sub> and Q<sub>10</sub>, Contents in foods and dietary intake. *J Food Comp Anal* 2001, 14, 409-417.
24. *McCarty M.F.*: Should we resist chloride rather than sodium? *Med. Hypotheses.*, 2004, 63(1), 138-148.
25. *Molloy A.M., Kirke P.N., Brody L.C., Scott J.M., Mills J.L.*: Effects of folate and vitamin B<sub>12</sub> deficiencies during pregnancy on fetal, infant and child development. *Food Nutr. Bull.*, 2008, 9(supl.2), 101-111.
26. *Nestler J., Jakubowicz D., de Vargas A., Brik C., Quintero N., Medina F.*: Insulin stimulates testosterone biosynthesis by human thecal cells from women with polycystic ovary syndrome by activating its own receptor and using inositoglycan mediators as the signal transduction system. *J Clin Endocrinol Metab* 1998, 83, 2001-2005.
27. *Ovalle F., Azziz R.*: Insulin resistance, polycystic ovary syndrome, and type 2 diabetes mellitus. *Fertil Steril* 2002, 77, 1095-1105.
28. *Ruder E.H., Hartman T.J.*: Goldman M.B., Impact of oxidative stress on female fertility. *Curr Opin Obstet Gynecol* 2009, 21, 219-222.
29. *Shannon M.M.*: Fertility cycles and nutrition. Can what you eat affect your menstrual cycle and your fertility. *The couple to couple Ohio*, 1996.
30. *Sicińska E., Wýka J.*: Folate intake in Poland on the basis of literature from the last ten years (2000-2010). *Rocz Panstw Zakl Hig* 2011, 62(3), 247-256, PMID: 22171513, <https://www.ncbi.nlm.nih.gov/pubmed/22171513>
31. *Szostak-Węgierek D., Waśkiewicz A.*: Metabolic disorders in women at procreative age living in Warsaw. *Rocz Panstw Zakl Hig* 2015;66(3):245-251, PMID: 26400121, <https://www.ncbi.nlm.nih.gov/pubmed/26400121>
32. *Szponar L., Wolnicka K., Rychlik E.*: Album fotografii produktów i potraw [Photo album products and dishes]. Wyd. Instytut Żywności i Żywienia, Warszawa 2000 (in Polish).
33. *Thierry Van Dessel H.J., Lee P-D., Faessen G.*: Elevated serum levels of free insulin-like growth factor I In polycystic ovary syndrome. *J Clin Endocrinol Metab* 1999, 84, 3030-3035.
34. *Thomson R.L., Buckley J.D., Noakes M., Clifton P.M., Norman R.J., Brinkworth G.D.*: The Effect of a Hypocaloric Diet with and without Exercise Training on Body Composition, Cardiometabolic Risk Profile, and Reproductive Function in Overweight and Obese Women with Polycystic Ovary Syndrome. *The journal of the Royal Society for Promotion of Health* 2008, 128(4), 190-195.
35. *Thomson R.L., Spedding S., Buckley J.D.*: Vitamin D in the aetiology and management of polycystic ovary syndrome. *Clin. Endocrinol*, 2012, 77, 343-350.
36. *Twigt J.M., Hammiche F., Sinclair K.D.*: Preconceptional folic acid use modulates estradiol and follicular responses to ovaria stimulation. *J Clin Endocrinol Metab* 2011, 96, 322-329.
37. *Uriu-Adams J.Y., Keen C.L.*: Zinc and reproduction: Effects of zinc deficiency on prenatal and early postnatal development. *Birth Defects Res B Dev Reprod Toxicol* 2010, 89, 313-325.
38. *World Health Organization*: Body Mass Index- BMI [online]. 2003 [dostęp: 14 marzec 2015]. <http://www.euro.who.int/en/health-topics/disease-prevention/nutrition/a-healthy-lifestyle/body-mass-index-bmi>.
39. *Wright C.E., Zborowski J.V., Talbott E.O., McHugh-Pemu K., Youk A.*: Dietary intake, physical activity, and obesity in women with polycystic ovary syndrome. *Int J Obes* 2004, 28, 1026-1032.
40. *Zhang G., Garmey J., Veldhuis J.*: Interactive stimulation by luteinizing hormone and insulin of the steroidogenic acute regulatory (StAR) protein and 17 alpha hydroxylase/17,20-lyase (CYP17) genes in porcine theca cells. *Endocrinology* 2000, 141, 2735-2742.

Received: 11.03.2016

Accepted: 08.06.2016