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The Significance of Plant-Based Foods and Intense Physical Activity on the Metabolic Health of Women with PCOS: A Priori Dietary-Lifestyle Patterns Approach

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Featured Application: This study will help develop dietary recommendations for women with PCOS.

Abstract: The study aimed to analyse dietary-lifestyle patterns (DLPs) and their relation with visceral obesity and other metabolic parameters in women with PCOS. A total of 140 women were diagnosed with PCOS. The KomPAN® and The ShortIPAQ questionnaires analysed the food frequency intake, health habits, economic situation, and physical activity. The dual-energy-x-ray absorptiometry (DXA) measured the visceral and total adipose tissue. The analysis distinguished three DLPs: western (WDLP), prudent (PDLP) and active (ADLP). The WDLP was characterised by high visceral fat, increased intake of animal foods, sweets and sweetened beverages, white grains, junk and fried foods, and low plant foods. High intakes of plant foods and dairy, high daily meal frequency, and intense exercise characterised PDLP. ADLP was characterised by high visceral fat, intake of plant products, intense exercise, and low intake of junk and fried food. Women with LDL > 135 mg/dL had high adherence to WDLP, and with triglycerides >150 mg/dL had high adherence to WDLP [OR 7.73 (CI95% 1.79; 33.2), $p < 0.05$] and [3.70 (1.03; 13.27); $p < 0,05$]. In conclusion, plant-based foods related to PDLP and intense physical activity offer a significantly higher chance of improving metabolic health in women with PCOS.

Keywords: polycystic ovary syndrome; body composition; densitometry; lipoproteins; androgens

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1. Introduction

Polycystic ovary syndrome (PCOS) is an endocrine disorder characterised by ovarian cysts, clinical or biochemical hyperandrogenism, and oligo-ovulation or anovulation [1]. Parallel to the above criteria, PCOS women have a higher chance for metabolic syndrome, central obesity, and insulin resistance than healthy populations [2]. Primary management of PCOS includes hormonal therapy [1]. Nevertheless, PCOS symptoms may be partially alleviated by incorporating healthy behaviours, including changes in diet composition and weight loss [3]. The different pathways of PCOS diagnosis by Rotterdam criteria cause us to distinguish four PCOS phenotypes: (I) Hyperandrogenism, ano- or oligo-ovulation, cystic ovaries; (II) Hyperandrogenism, ano- or oligo-ovulation; (III) Hyperandrogenism, cystic ovaries; (IV) ano- or oligo-ovulation, cystic ovaries. The evidence shows that different phenotypes are associated with diverse dietary patterns and obesity [4–7].

The low glycemic index (GI) diet is a well-known recommendation for PCOS. It has been published widely that Low GI foods consumption and avoidance of high GI products may influence insulin sensitivity and androgen status [4,8–10]. The relations between dietary carbohydrate composition, low GI and hyperandrogenism are still not fully known

in this group. Moreover, it has been shown that sugar limitations and diet modifications often have unsatisfactory effectiveness in diet therapy [11]. High androgens may be caused by hyperinsulinemia, which increases the local testosterone by inhibiting the synthesis of sex hormone-binding globulin (SHGB) [12]. However, the GI list of foods has many drawbacks, and its indiscriminate use severely limits the diversity of the diet. First, a combination of meals composed of high and low GI foods, such as high protein or fibre, can lower the glucose uptake from the intestines, lowering the peak of insulin [13]. Second, despite the low GI, meat products deliver animal fats, including saturated fatty acids, and their intake above recommendations increases the risk of metabolic syndrome. Third, high GI plant products (such as fruits) enrich the diet with high anti-inflammatory status or prebiotic ingredients. Consumption of anti-inflammatory food and synbiotics also shows some beneficial effects in alleviating symptoms of PCOS [14–18]. It has been studied that following Mediterranean dietary patterns, rich in fruits and vegetables, increase the total dietary antioxidant capacity and decreases inflammatory markers [19,20], as shown in PCOS [21]. Still, researchers should be cautious with this recommendation, given the seasonality of consumption in Central Europe [22,23]. Such arguments show that the discussion on PCOS diet guidelines is still needed and should be developed. Simultaneously, many studies suggest that a hygienic lifestyle with a healthy diet and adequate physical activity improves the PCOS endpoints [24].

Most of the studies mentioned above were performed using a single nutrient or, in the case of observational studies, already established diet quality scores and patterns. Dietary lifestyle patterns (DLP) define the quantities, proportions, variety, or combination of different foods and drinks in diets and behaviours and the frequency with which they are habitually consumed or practised evaluated as a single exposure [19,25]. They can be assessed either a priori or a posteriori. A priori concerns an analysis of already established patterns and quality scores, such as healthy eating index and western or Mediterranean dietary patterns. In turn, the a posteriori is based on the food intake analysis after data collection using factor analysis or principal components analysis [26]. According to the author's knowledge, only two studies in the literature involve a posteriori dietary pattern analysis of women with PCOS [27,28]. Their authors compared PCOS with the control group, not recognising the dietary patterns within the specific group of women with PCOS. Moran et al. [28] concluded that women with PCOS were more likely to follow a Mediterranean dietary pattern than the healthy controls. This dietary pattern is based on an increased intake of plant food sources (such as fruits, vegetable legumes), olive oil and fish but decreased meat intake [28]. Women in this study could improve dietary behaviours even prior to diagnosis due to the existing symptoms. Analysing dietary patterns within this group could point out specific nutrition behaviour problems.

Metabolic disorders are also related to lifestyle patterns, such as the amount of physical activity, sleeping time and sedentary behaviours. Currently, no published literature analysed lifestyle patterns among women with PCOS. However, there are multiple studies on the influence of different intensities and types of physical activity on PCOS disease endpoints, and exercise should be the basis of health recommendations [29].

Simultaneously, women with PCOS have a higher incidence of excess visceral adipose tissue (VAT). Such fat distribution is related to insulin resistance, hyperglycaemia, hypercholesterolemia, and dyslipidemia [30–32]. According to research, VAT is an early marker measurement of metabolic syndrome [33]. A comprehensive analysis of VAT and food frequency intake in young women with PCOS could predict metabolic disorders in the future. According to the author's best knowledge, there is no current research concerning the VAT derived from DXA and a posteriori lifestyle dietary patterns of women with PCOS. Therefore, the study aimed to analyse dietary-lifestyle patterns (DLPs) and their relation with visceral obesity and other metabolic parameters in women with PCOS.

2. Materials and Methods

2.1. Study Participants

A total of 140 patients with PCOS were recruited for the nutrition part of the study from the Department of Endocrinology, Metabolism and Internal Diseases, Poznan University of Medical Sciences (Figure 1). The recruitment process was based on the following inclusion and exclusion criteria; PCOS diagnosis according to Rotterdam criteria, age: 18–40 y.o., BMI < 40, no diagnosis of extreme obesity, heart defect, decompensated thyroid dysfunction, severe acute or chronic renal or liver diseases, Cushing's disease, congenital adrenal hyperplasia, or eating disorders, no birth control or hormone replacement therapy, ovulation-inducing agents, anti-androgens over the last 2 months prior to the study. Informed and written consent was obtained from all participants. The clinical examination protocol complied with the Declaration of Helsinki for Human and Animal Rights and its later amendments and received ethical approval from the Board of Bioethics of the University of Medical Science (552/16; 986/17).

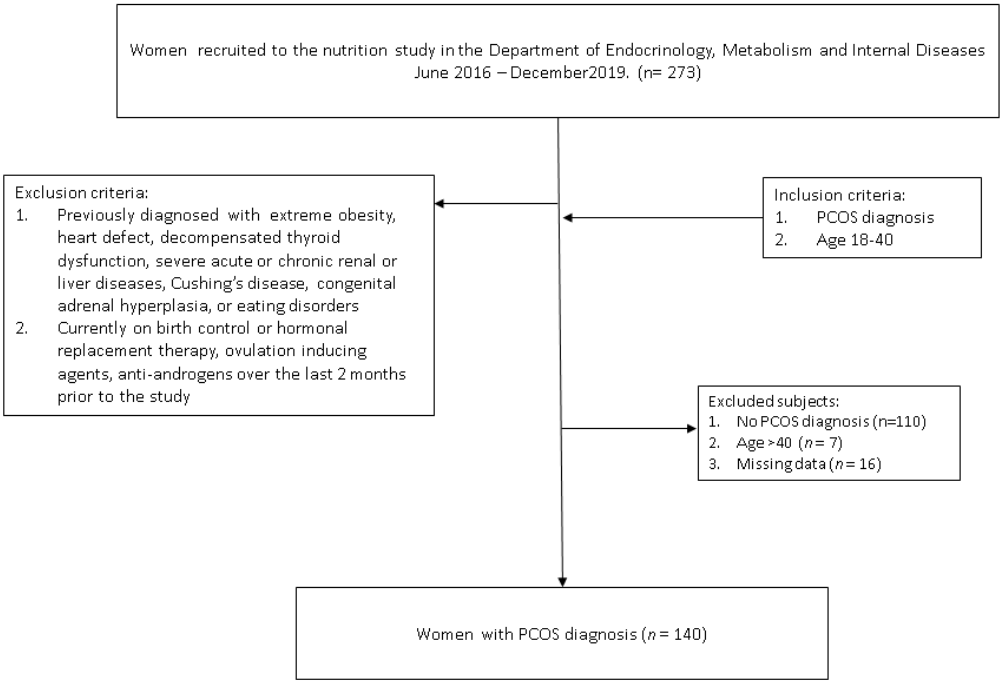

Figure 1. Flowchart of the study.

2.2. Body Composition Parameters

Body composition was measured by the dual-energy x-ray absorptiometry (DXA) method. The Lunar Prodigy™ (GE Healthcare©, Madison, WI, USA, 2013) densitometer was used for evaluation. The quality control was performed according to the user manual on each day of the study visits. VAT was analysed using the software enCORE™ (version 17) and CoreScan™ (GE Healthcare©, Madison, WI, USA). A qualified staff member performed all measures, including weight, height, waist and hip circumference.

2.3. Food Frequency Intake and Lifestyle Habits

Food frequency, lifestyle habits, and socioeconomic status were assessed by validated Dietary Habits and Nutrition Beliefs Questionnaire KomPAN® [34,35]. The food frequency part included twenty-four food groups with six possible answers: (1) never, (2) 1–3 times a month, (3) once a week, (4) 2–3 times a week, (5) once a day, (6) a few times during the day. Those responses were converted into an average frequency of food intake per day [22,34]. The foods were divided into different groups: meats (red meat, white meat, smoked meat, meat conserves), plant foods (vegetables, fruits, legumes), dairy (milk, fermented milk

drinks, cottage cheese, hard cheese), wholegrains (whole bread, wholegrain products such as pasta, rice, groats, oats), white grains (white bread, white pasta, rice) sweets and sweetened beverages (sweets, sweetened beverages). KomPAN® also includes questions that verify reliability (crossing questions concerning the same issue). These involve, for example, the typical day number of meals in two parts of the questionnaire. If the two answers differed by two or more meals, the patient was not included in the study since the low reliability of the answers.

2.4. Biochemical Parameters

Blood samples were collected from all patients after an overnight fast. Insulin, follicle-stimulating hormone (FSH), luteinising hormone (LH), and total testosterone (T), androstenedione (A) were analysed using a Cobas 6000 (Roche Diagnostics, GmbH, Mannheim, Germany). Kits available from the manufacturer were used. Total cholesterol (TC), high-density lipoprotein cholesterol (HDL), and triglycerides (TG) were evaluated by the enzymatic colourimetric method. The Friedewald formula calculated low-density lipoprotein cholesterol (LDL). Serum glucose was assessed with the hexokinase method (Roche Diagnostics) and a coefficient of variation (CV) of 3%. The formula (HOMA-IR) calculated the homeostasis model assessment for insulin resistance: HOMA-IR = (fasting plasma glucose (mg/dL) $\times$ fasting plasma insulin (mU/L))/405. The threshold of HOMA-IR > 2.5 was used.

2.5. Statistics

The software Statistica v.13.1 (StatSoft Polska sp. z o.o., Kraków, Poland) was used for the calculations. The study group characteristics involved calculating means, standard deviation, medians and confidence intervals set for 95%. Each variable was checked for normality. The dietary patterns were analysed by Principal Component Analysis (PCA) method, with varimax normalised rotation. A total of twenty-four food frequency intake variables were included in the PCA. The dietary patterns were identified by considering the following criteria: (1) the eigenvalues of the variable correlations >1.0, (2) the plot of eigenvalues, and (3) the total variance explained [36]. Rotated factor loadings with an absolute value $\geq |0.50|$ were considered specific to the given pattern. For each patient and each pattern, the scores were calculated as a product of factor loading and food frequency consumption. Next, for each dietary pattern, tertile intervals were calculated to measure the adherence to the patterns of each patient.

Logistic regression was used to analyse the associations between dietary patterns and metabolic parameters. The odds ratios (ORs) and 95% confidence intervals (95%CIs) were calculated between upper tertiles of dietary patterns and recommended values for metabolic parameters.

3. Results

3.1. Lifestyle-Dietary Patterns and Patient's Characteristics

The patient's characteristics can be seen in Table 1. The study group's mean age was 26 $\pm$ 5 years old, and BMI was 25.4 $\pm$ 5.2 kg/m^2. Three lifestyle-dietary patterns have been distinguished among women with PCOS: (1) Western (WDLP), (2) Prudent (PDLP) (3) Active (ADLP). The factor loadings of each dietary pattern are depicted in Figure 2 and Supplementary materials Table S1. The WDLP was characterised by high visceral fat, high-frequency intake of animal foods, sweets and sweetened beverages, white grains, junk and fried foods, and low-frequency intake of plant foods. PDLP was characterised by high-frequency intake of plant foods and dairy, high meal frequency per day, and intense exercise. ADLP was characterised by high visceral fat, high-frequency intake of plant products, intense exercise and low intake of junk and fried food.

Table 1. Patient's characteristics (n = 140).

	Mean	±SD	Median	CI (95%)	
Age (years)	26	5	25	25	27
Height (cm)	165	15	167	163	168
Body mass (kg)	70.8	14.9	68.0	68.3	73.3
BMI (kg/m^2)	25.4	5.2	24.1	24.5	26.3
Waist circumference (cm)	81.5	13.0	79.5	79.3	83.7
Hips circumference (cm)	100.5	11.4	100.0	98.6	102.4
WHR (−)	0.81	0.08	0.80	0.80	0.82
FM (%)	36	8	35	35	37
VAT (g)	467	530	283	378	555
TC (mg/dL)	178	31	176	173	184
HDL (mg/dL)	66	16	63	63	68
LDL (mg/dL)	95	30	93	90	100
TG (mg/dL)	87	58	69	77	97
Fasting glucose (mg/dL)	89	7	88	88	9
Fasting insulin (uU/mL)	11.09	7.15	8.89	9.84	12.34
HOMA-IR (−)	2.52	1.81	1.94	2.20	2.84
FSH (mlU/mL)	6.6	8.00	5.9	5.1	8.00
LH ((mIU/mL)	10.4	6.1	8.4	9.3	11.5
LH/FSH (−)	1.8	1.1	1.5	1.6	2.0
T nmol/L	1.9	0.9	1.6	1.7	2.0

Abbreviations: BMI—Body Mass Index; WHR—Waist-to-Hip Ratio; WHtR—Waist-to-Height Ratio; FM—fat mass; VAT—Visceral Fat Tissue; TC—Total cholesterol; HDL—High-density lipoprotein; LDL—Low-density lipoprotein; TG—Triglycerides; HOMA-IR—Homeostatic Model Assessment—Insulin Resistance; FSH—Follicle Stimulating Hormone; LH—luteinising hormone, T—Total testosterone.

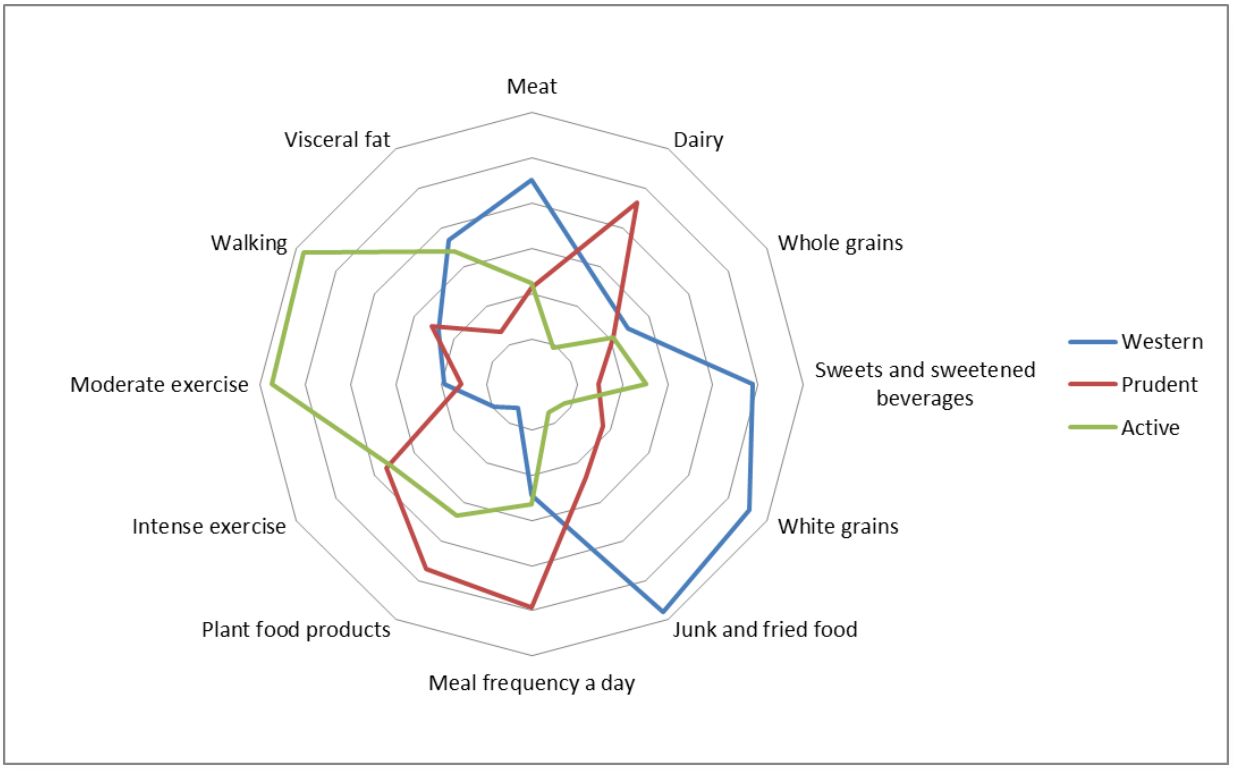

Figure 2. Factor loadings diagram characterising dietary-lifestyle patterns distinguished among women with PCOS.

The mean frequency intakes of different food groups for each adherence can be found in Supplementary Tables S2–S5.

3.2. Metabolic Parameters

Women with PCOS, with high adherence to WDLP, were prone to increase (>135 mg/dL and >150 mg/dL) levels of LDL and triglycerides, respectively (Table 1). The low adherence to WDLP decreased the chance (70%) of BMI over 30 kg/m^2. They also had a lower chance of WHtR above 0.5 and HDL below 50 mg/dL.

Women with high adherence to PDLP had almost 58% less chance of having a BMI above 25 kg/m^2 and 54% less chance of having body fat above 35% (Table 2). In turn, middle adherence to prudent DLP was related to a higher chance of BMI over 25 kg/m^2 and total cholesterol below 200 mg/dL. Low adherence to prudent DLP was associated with over three times higher chance of total cholesterol over 200 mg/dL than the rest of the participants (Table 2).

Table 2. The adherence to the western dietary-lifestyle pattern (DLP) and its relation with different metabolic and endocrine markers.

	High Adherence to WDLP		Middle Adherence WDLP		Low Adherence to WDLP	
	n	**OR (CI95%), *p***	***n***	**OR (CI95%), *p***	***n***	**OR (CI95%), *p***
BMI > 30 kg/m^2	12	2.35 (0.94; 5.88), $p = 0.06$	8	1.13 (0.44; 2.92), $p = 0.79$	4	0.30 (0.09; 0.97), $p = 0.04$ *
BMI > 25 kg/m^2	22	1.27 (0.62; 2.63), $p = 0.51$	22	1.58 (0.76; 3.30), $p = 0.21$	16	0.49 (0.23; 1.05), $p = 0.06$
WHR > 0.80	24	1.69 (0.79; 3.60), $p = 0.17$	21	1.17 (0.55; 2.46), $p = 0.68$	18	0.51 (0.24; 1.09), $p = 0.07$
WhtR > 0.5	21	1.37 (0.65; 2.87), $p = 0.39$	22	1.77 (0.84; 3.72), $p = 0.12$	14	0.40 (0.19; 0.88), $p = 0.02$ *
Fat > 35%	24	1.17 (0.56;2.41), $p = 0.66$	24	1.47 (0.70; 3.08), $p = 0.29$	21	0.58 (0.28; 1.51), $p = 0.15$
T. Chol. > 200 mg/dL	11	1.91 (0.74; 4.92), $p = 0.17$	9	1.23 (0.48; 3.19), $p = 0.66$	6	0.40 (0.13; 1.15), $p = 0.08$
LDL > 135 mg/dL	8	7.73 (1.79; 33.2), $p < 0.00$ *	1	0.19 (0.20; 1.64), $p = 0.13$	2	0.32 (0.06; 1.67), $p = 0.17$
HDL < 50 mg/dL	9	2.24 (0.80; 6.25), $p = 0.12$	7	1.49 (0.52; 4.20), $p = 0.44$	2	0.19 (0.04;0.89), $p = 0.03$ *
TG > 150 mg/dL	7	3.70 (1.03; 13.27), $p = 0.04$ *	3	0.71 (0.17; 2.90), $p = 0.64$	2	0.28 (0.05; 1.44), $p = 0.12$
HOMA > 2.5	21	1.93 (0.91; 4.07), $p = 0.08$	16	1.11 (0.52; 2.38), $p = 0.77$	13	0.44 (0.20; 0.99), $p = 0.04$ *
Fasting gluc. > 100 mg/dL	5	2.39 (0.62; 9.20), $p = 0.19$	3	0.95 (0.23; 4.04), $p = 0.95$	2	0.37 (0.07; 1.95), $p = 0.24$
Fasting ins. > 10 mU/mL	25	1.53 (0.75; 3.16), $p = 0.24$	24	1.75 (0.84; 3.66), $p = 0.12$	16	0.37 (0.17; 0.78), $p = 0.01$ *
LH > upper tertile	14	1.05 (0.48; 2.31), $p = 0.90$	16	1.67 (0.76; 3.64), $p = 0.19$	11	0.56 (0.24; 1.28), $p = 0.16$
FSH > upper tertile	18	2.30 (1.03; 5.11), $p = 0.04$ *	9	0.56 (0.24; 1.35), $p = 0.19$	13	0.72 (0.32; 1.63), $p = 0.43$
LH/FSH > upper tertile	14	0.98 (0.44; 2.17), $p = 0.97$	15	1.35 (0.62; 2.96), $p = 0.44$	13	0.74 (0.34; 1.65), $p = 0.47$
T > upper tertile	7	0.35 (0.15; 0.83), $p = 0.01$ *	13	2.01 (0.94; 4.30), $p = 0.07$	8	1.28 (0.60; 2.71), $p = 0.51$
A > upper tertile	10	0.37 (0.16; 0.88), $p = 0.02$ *	21	2.57 (1.21; 5.48), $p = 0.01$ *	16	0.96 (0.45; 2.05), $p = 0.99$
DHEA-s > upper tertile	14	0.69 (0.32; 1.51), $p = 0.35$	18	1.68 (0.79; 3.57), $p = 0.17$	15	0.86 (0.40; 1.83), $p = 0.68$
PCOS type 1	24	0.86 (0.42; 1.78), $p = 0.69$	22	0.90 (0.43; 1.88), $p = 0.78$	28	1.27 (0.61; 2.61), $p = 0.51$
PCOS type 2	7	0.79 (0.29; 2.14), $p = 0.65$	9	1.58 (0.61; 4.04), $p = 0.33$	8	0.78 (0.29; 2.09), $p = 0.62$
PCOS type 3	4	0.79 (0.22; 2.78), $p = 0.71$	4	1.01 (0.29; 3.56), $p = 0.97$	5	1.23 (0.37; 4.08), $p = 0.73$
PCOS type 4	12	1.68 (0.70; 4.04), $p = 0.24$	7	0.75 (0.28; 1.97), $p = 0.56$	8	0.75 (0.30; 1.89), $p = 0.54$

Abbreviations: BMI—Body Mass Index, WHR—Waist-to-Hip Ratio, WHtR—Waist-to-Height Ratio, T. Chol.—total cholesterol; HDL—High-density lipoprotein, LDL—Low-density lipoprotein, TG—Triglycerides, HOMA-IR—Homeostatic Model Assessment—Insulin Resistance, Fasting gluc.—fasting glucose, Fasting ins.—fasting insulin, FSH—Follicle Stimulating Hormone, LH—luteinising hormone, T—total testosterone, A—androstenedione; DHEA-s—Dehydroepiandrosterone sulfate. The *p* values below the threshold of statistical significance are marked with the * $p < 0.05$.

High and low adherence to ADLP was not significantly related to any metabolic markers. However, middle adherence was related to body fat above 35% and WHtR above 0.5.

3.3. Endocrine Parameters

Women with high adherence to western DLP had over twice the higher probability of having FSH above the third tertile. High adherence to WDLP was also related to a 65% and 63% lower chance of total T and A, respectively, above the third tertile (Table 2).

Women with high adherence to prudent PDLP were nearly two and a half more likely to have PCOS type 2 (Table 3). In turn, the low adherence to that pattern was related to androstenedione above 245 ng/dL.

High adherence to active DLP was related to PCOS type 2, and low adherence to that pattern was related to androstenedione above 245 ng/dL (Table 4).

Table 3. The adherence to the prudent dietary-lifestyle pattern (PDLP) and it's relation with different metabolic and endocrine markers.

	High Adherence to PDLP		Middle Adherence to PDLP		Low Adherence to PDLP	
	n	**OR (CI95%), *p***	***n***	**OR (CI95%), *p***	***n***	**OR (CI95%), *p***
BMI > 30 kg/m^2	6	0.60 (0.22; 1.65), $p = 0.32$	8	1.04 (0.40; 2.68), $p = 0.93$	10	1.52 (0.61; 3.79), $p = 0.36$
BMI > 25 kg/m^2	14	0.42 (0.20; 0.89), $p = 0.02$ *	25	2.09 (1.00; 4.34), $p = 0.04$ *	21	1.11 (0.54; 2.28), $p = 0.77$
WHR > 0.80	23	1.24 (0.59; 2.58), $p = 0.56$	19	0.84 (0.40; 1.77), $p = 0.64$	21	0.95 (0.46; 1.98), $p = 0.95$
WhtR > 0.5	15	0.54 (0.25; 1.14), $p = 0.10$	21	1.46 (0.70; 3.05), $p = 0.30$	21	1.25 (0.60; 2.59), $p = 0.53$
Fat > 35%	18	0.45 (0.21; 0.94), $p = 0.03$ *	23	1.11 (0.54; 2.30), $p = 0.76$	28	1.97 (0.95; 4.10), $p = 0.06$
T. Chol. > 200 mg/dL	8	0.81 (0.30; 2.16), $p = 0.68$	4	0.29 (0.09; 0.94), $p = 0.04$ *	14	3.27 (1.28; 8.39), $p = 0.01$ *
LDL > 135 mg/dL	3	0.68 (0.16; 2.83), $p = 0.59$	3	0.77 (0.18; 3.16), $p = 0.71$	5	1.78 (0.49; 6.43), $p = 0.37$
HDL < 50 mg/dL	4	0.51 (0.16; 1.69), $p = 0.27$	5	0.77 (0.25; 2.33), $p = 0.64$	9	2.21 (0.80; 6.08), $p = 0.12$
TG > 150 mg/dL	4	0.94 (0.25; 3.47), $p = 0.92$	2	0.38 (0.08; 1.89), $p = 0.23$	6	2.22 (0.64; 7.67), $p = 0.20$
HOMA > 2.5	14	0.59 (0.27; 1.29), $p = 0.18$	15	0.87 (0.40; 1.85), $p = 0.71$	21	1.88 (0.90; 3.95), $p = 0.09$
Fasting gluc. > 100 mg/dL	4	1.30 (0.69; 5.09), $p = 0.70$	2	0.50 (0.10; 2.52), $p = 0.39$	4	1.37 (0.35; 5.34), $p = 0.64$
Fasting ins. > 10 mU/mL	19	0.62 (0.30; 1.28), $p = 0.19$	23	1.33 (0.65; 2.75), $p = 0.42$	23	1.20 (0.59; 2.45), $p = 0.61$
LH > upper tertile	12	0.74 (0.33; 1.66), $p = 0.46$	15	1.29 (0.59; 2.81), $p = 0.51$	14	1.03 (0.47; 2.26), $p = 0.93$
FSH > upper tertile	12	0.69 (0.30; 1.58), $p = 0.38$	14	1.22 (0.55; 2.72), $p = 0.61$	14	1.15 (0.52; 2.56), $p = 0.71$
LH/FSH > upper tertile	15	1.13 (0.52; 2.45), $p = 0.75$	14	1.05 (0.48; 2.29), $p = 0.90$	13	0.83 (0.38; 1.84), $p = 0.66$
T > upper tertile	14	0.68 (0.31; 1.49), $p = 0.32$	17	1.34 (0.63; 2.86), $p = 0.44$	16	1.08 0.51; 2.30), $p = 0.84$
A > upper tertile	14	0.76 (0.35; 1.63), $p = 0.47$	16	1.12 (0.53; 2.38), $p = 0.76$	17	1.17 (0.55; 2.46), $p = 0.67$
DHEA-s > upper tertile	17	1.17 (0.55; 2.46), $p = 0.68$	15	0.98 (0.46; 2.08), $p = 0.95$	15	0.87 (0.41; 1.86), $p = 0.72$
Pcos type 1	22	0.64 (0.31; 1.32), $p = 0.22$	25	1.08 (0.52; 2.24), $p = 0.82$	27	1.44 (0.69; 3.00), $p = 0.32$
Pcos type 2	13	2.45 (0.98; 6.16), $p = 0.05$ *	5	0.51 (0.17; 1.50), $p = 0.22$	6	0.67 (0.24; 1.87), $p = 0.45$
Pcos type 3	6	1.72 (0.54; 5.53), $p = 0.32$	3	0.59 (0.15; 2.31), $p = 0.45$	4	0.89 (0.26; 3.11), $p = 0.86$
Pcos type 4	7	0.60 (0.23; 1.58), $p = 0.30$	12	1.88 (0.78; 4.50), $p = 0.15$	8	0.82 (0.33; 2.09), $p = 0.69$

Abbreviations: BMI—Body Mass Index, WHR—Waist-to-Hip Ratio, WHtR—Waist-to-Height Ratio, T. Chol.—total cholesterol; HDL—High-density lipoprotein, LDL—Low-density lipoprotein, TG—Triglycerides, HOMA-IR—Homeostatic Model Assessment—Insulin Resistance, Fasting gluc.—fasting glucose, Fasting ins.—fasting insulin, FSH—Follicle Stimulating Hormone, LH—luteinising hormone, T—total testosterone, A—androstenedione; DHEA-s—Dehydroepiandrosterone sulfate. The *p* values below the threshold of statistical significance are marked with the * $p < 0.05$.

Table 4. The adherence to the active dietary-lifestyle pattern (DLP) and it's relation with different metabolic and endocrine markers.

	High Adherence to ADLP		Middle Adherence to ADLP		Low Adherence to ADLP	
	n	**OR (CI95%), *p***	***n***	**OR (CI95%), *p***	***n***	**OR (CI95%), *p***
BMI > 30 kg/m^2	11	1.83 (0.74; 4.53), $p = 0.18$	9	1.35 (0.53; 3.41), $p = 0.52$	4	1.09 (0.42; 2.81), $p = 0.80$
BMI > 25 kg/m^2	23	1.39 (0.68; 2.87), $p = 0.36$	23	1.67 (0.80; 3.46), $p = 0.16$	14	0.87 (0.41; 1.81), $p = 0.70$
WHR > 0.80	24	1.34 (0.64; 2.78), $p = 0.43$	21	1.22 (0.58; 2.57), $p = 0.60$	18	0.84 (0.40; 1.78), $p = 0.65$
WhtR > 0.5	21	1.20 (0.58; 2.48), $p = 0.62$	24	2.37 (1.12; 5.01), $p = 0.02$ *	12	0.97 (0.46; 2.03), $p = 0.93$
Fat > 35%	24	0.97 (0.47; 1.99), $p = 0.94$	29	2.71 (1.27; 5.77), $p = 0.01$ *	16	1.60 (0.77; 3.33), $p = 0.21$
Cholersterol > 200 mg/dL	13	2.35 (0.93; 5.89), $p = 0.07$	4	0.32 (0.10; 1.06), $p = 0.06$	9	1.82 (0.72; 4.63), $p = 0.20$
LDL > 135 mg/dL	6	2.51 (0.70; 9.05), $p = 0.15$	1	0.20 (0.02; 1.68), $p = 0.13$	4	1.23 (0.33; 4.63), $p = 0.75$
HDL < 50 mg/dL	8	1.65 (0.60; 4.58), $p = 0.32$	7	1.42 (0.50; 4.01), $p = 0.50$	3	1.88 (0.68; 5.22), $p = 0.22$
TG > 150 mg/dL	5	1.39 (0.40; 4.84), $p = 0.60$	5	1.78 (0.50; 6.31), $p = 0.36$	3	1.60 (0.46; 5.60), $p = 0.46$
HOMA > 2.5	18	1.03 (0.49; 2.18), $p = 0.92$	17	1.21 (0.58; 2.48), $p = 0.61$	15	1.09 (0.53; 2.27), $p = 0.80$
Fasting gluc. > 100 mg/dL	5	1.28 (0.33; 4.96), $p = 0.71$	1	0.22 (0.03; 1.92), $p = 0.17$	4	0.88 (0.21; 3.76), $p = 0.81$
Fasting ins. > 10 mU/mL	26	1.52 (0.75; 3.12), $p = 0.24$	21	1.05 (0.51; 2.17), $p = 0.88$	18	1.10 (0.53; 2.27), $p = 0.79$
LH > upper tertile	16	1.35 (0.63; 2.92), $p = 0.43$	11	0.70 (0.31; 1.61), $p = 0.40$	14	1.00 (0.47; 2.12), $p = 0.99$
FSH > upper tertile	14	0.93 (0.87; 2.09), $p = 0.87$	12	0.94 (0.41; 2.12), $p = 0.88$	14	1.10 (0.49; 2.48), $p = 0.80$
LH/FSH > upper tertile	16	1.28 (0.59; 2.77), $p = 0.52$	12	0.79 (0.35; 1.77), $p = 0.57$	14	0.80 (0.36; 1.80), $p = 0.60$
T > upper tertile	18	1.20 (0.57; 2.55), $p = 0.62$	13	0.77 (0.35; 1.69), $p = 0.51$	16	1.06 (0.49; 2.29), $p = 0.87$
A > upper tertile	17	1.17 (0.56; 2.47), $p = 0.67$	14	1.57 (0.47; 5.26), $p = 0.46$	16	1.01 (0.47; 2.17), $p = 0.96$
DHEA-s > upper tertile	16	1.02 (0.48; 2.17), $p = 0.95$	18	1.48 (0.70; 3.13), $p = 0.30$	13	0.75 (0.34; 1.64), $p = 0.47$
Pcos type 1	31	2.10 (1.00; 4.40), $p = 0.04$ *	18	0.46 (0.22; 0.96), $p = 0.04$ *	25	1.83 (0.85; 3.90), $p = 0.11$
Pcos type 2	6	0.47 (0.16; 1.39), $p = 0.17$	13	3.47 (1.37; 8.82), $p = 0.00$ *	5	0.58 (0.19; 1.69), $p = 0.31$
Pcos type 3	3	0.55 (0.14; 2.14), $p = 0.39$	5	1.35 (0.41; 4.44), $p = 0.62$	5	0.65 (0.17; 2.56), $p = 0.54$
Pcos type 4	8	0.77 (0.30; 1.94), $p = 0.58$	8	0.86 (0.34; 2.17), $p = 0.75$	11	0.75 (0.29; 1.96), $p = 0.56$

Abbreviations: BMI—Body Mass Index, WHR—Waist-to-Hip Ratio, WHtR—Waist-to-Height Ratio, T. Chol.—total cholesterol; HDL—High-density lipoprotein, LDL—Low-density lipoprotein, TG—Triglycerides, HOMA-IR—Homeostatic Model Assessment—Insulin Resistance, Fasting gluc.—fasting glucose, Fasting ins.—fasting insulin, FSH—Follicle Stimulating Hormone, LH—luteinising hormone, T—total testosterone, A—androstenedione; DHEA-s—dehydroepiandrosterone sulfate. The *p* values below the threshold of statistical significance are marked with the * $p < 0.05$.

4. Discussion

The study met its aim and distinguished three dietary-lifestyle patterns (DLP): western (WDLP), prudent (PDLP) and active (ADLP). The WDLP was characterised by a high-frequency intake of animal foods, sweets and sweetened beverages, white grains, junk and fried foods, and a low-frequency intake of plant foods. This DLP was also characterised by high visceral fat mass. PDLP was characterised by high-frequency intake of plant foods and dairy, high meal frequency per day, and intense exercise. ADLP was characterised by high visceral fat, high-frequency intake of plant products, intense exercise and low intake of junk and fried food.

4.1. The Relation of DLPs with Metabolic and Endocrine Markers

High adherence to WDLP was related to high LDL, triglycerides, and FSH but low total testosterone and androstenedione. Patients with low adherence to the WDLP were at lower risk of being obese and having insulin resistance. This result agrees with other studies showing that western dietary patterns harm metabolic health in different populations [26,37].

In turn, high adherence to PDLP was related to the low chance of overweight and excess fat tissue. At the same time, they were more likely to have PCOS type II. Nevertheless, this result contradicts the previous study, which found that PCOS type II is inversely associated with healthy eating [38]. However, most studies on PCOS phenotypes, nutrition status and dietary intake show that obesity and adherence to western dietary patterns tend to be related to classic PCOS phenotype (type I) [4–6]. This statement agrees with our findings that type I of PCOS was related to ADLP. It is worthwhile to underline that this pattern, although characterised by high physical activity, also had high visceral fat volume and some unhealthy dietary behaviours.

4.2. Plant Products Intake

The distinguished DLPs support the results of other studies controlling the intake of sweets and sweetened beverages, where increasing the vegetable intake was related to a lower incidence of insulin resistance and dyslipidemia [8,12]. This result supports studies showing the importance of the GI of diet for women with PCOS. A low GI diet has been recommended for women with PCOS, especially with increased insulin resistance [39]. Low GI foods are primarily plant-based foods containing complex carbohydrates, such as unprocessed grains, legumes and vegetables. However, there are some drawbacks to following only the list of low GI products [13]. As mentioned earlier, the proper combination of products with high and low GI in one meal will reduce glucose absorption, and restriction in different fruits may influence the antioxidant dietary capacity. In our study, a high intake of fruits did not negatively affect metabolic markers. Moreover, the combination of vegetables and legumes had a positive effect. In turn, meat intake in our study, which has a low GI, negatively affected metabolic markers in women with PCOS.

4.3. Meat Intake

In our study, the intake of meats (white and red meat) has been a significant factor in distinguishing DLPs. High-frequency intake of meat as part of WDLP was related to metabolic disorders in PCOS. Patients with high adherence to WDLP consumed meat products on average over once a day while comparing it to those with low adherence, where meat intake was on average a few times a week (Supplement Table S2). Excessive intake of meat is related to multiple chronic diseases. It has been shown that saturated fatty acid intake, presented in meat, was related to heart rate variability [40]. Some studies suggest that women with PCOS consume more saturated fatty acids than recommended [24]. A high saturated fat intake is present in red meats, but sweets and fried food in WDLP could lower insulin sensitivity. Excess of saturated fatty acids, especially palmitate, influences the increase of white adipose tissue and apoptosis through oxidative or endoplasmic reticulum stress, generation of ceramide and reactive oxygen species, and protein kinase C signalling [41]. Information concerning meat intake is very limited in the dietary manage-

ment of PCOS. Servings of fruits and vegetables are carefully explained, yet there is no recommendation concerning the number of servings and portion sizes of meats. Setting the proper animal product servings in diet therapy is also essential to establish the most optimal diet to alleviate the PCOS symptoms and protect the environment.

4.4. Dairy Intake

One of the alternative protein sources is dairy. The high frequency of dairy consumption in our study was a part of PDLP and has been related to a more desirable metabolic profile in women with PCOS. The literature shows that high-frequency dairy intake is related to a lower risk of obesity and cardiovascular risks [42]. However, dairy intake and its relation to PCOS, fertility and insulin resistance are unclear. Some studies suggest that consuming dairy products was negatively associated with the risk of type 2 diabetes mellitus, insulin resistance and ovulation disorders [43]. The fermented dairy had a significant part in this group. Fermented dairy, such as yoghurt, has been associated with lowering body mass [44], and its probiotic properties improve anti-inflammatory response [45]. Consumption of fermented products could reduce pancreatic lipase activity, decreasing fatty acid absorption [46].

It is essential to mention that dairy restrictions could increase the consumption of phytoestrogens from dairy alternatives such as soy products. Phytoestrogens could be beneficial in PCOS therapy; however, more studies in this field are needed [47,48].

4.5. Meal Frequency

Additionally, women following the PDLP had a high frequency of meals eaten during the day, and women following WDLP had a low frequency of meals per day. These results emphasise the importance of regular meals in diet therapy. The high frequency of meals has been related to lower body fat and a high fat-free mass among the adult population [49].

4.6. Physical Activity

Intensive exercise has been a significant factor in PDLP, and low exercise, in turn, has been a characteristic of WDLP, which supports the importance of physical activity for health. However, the surprise has been the ADLP, which, even though significantly high medium intensity exercise and walking, also had high VAT and was more prone to complete PCOS symptoms [50].

4.7. Limitations

Even though the study reached its aims, it had some limitations. First, the sample size has been limited; however, the smaller sample size allowed us to include more endocrine and metabolic markers analysis. Second, the physical activity was self-reported in our study by an internationally validated questionnaire. Physical activity tracking by online devices could be used in future research to support the results of our study. Third, the food frequency questionnaire was used. Even though the validated questionnaire allowed the calculation of multiple nutrition scores, it could be valuable to see the daily energy intake and the nutritional density in future research. However, the food frequency questionnaire allowed us to analyse long-term nutrition habits, which are not depending on seasonal variability. The KomPAN questionnaire specifically asks about the intake in the past year. A 24-h recall cannot show a typical annual intake [51].

5. Conclusions

Three dietary-lifestyle patterns (DLPs) were distinguished and related to the visceral fat tissue in women with PCOS: Western, prudent, and active. Low adherence to WDLP was additionally related to desirable levels of metabolic markers. High adherence to PDLP was linked to type two PCOS. The PDLP was characterised by increased frequency intake of plant-based food and intense physical activity. In turn, the least desirable WDLP was

characterised by a high intake of meat products and low physical activity. The role of plant-based foods could be underestimated in diet therapy, and it needs further investigation.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/app13042118/s1, Table S1. Factor loadings of three dietary-lifestyle patterns (DLP): western (WDLP), prudent (PDLP), and active (ADLP). Table S2. Food frequency intake of the study group (n = 140) described in means and medians. Table S3. Mean food frequency intake per day for high, medium and low adherence to the western dietary-lifestyle pattern (WDLP). table S4. Mean daily food frequency intake for high, medium and low adherence to the prudent dietary-lifestyle pattern (PDLP). Table S5. Mean food frequency intake per day for high, medium and low adherence to the active dietary-lifestyle pattern (ADLP).

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