
RESEARCH ARTICLE

Effectiveness of Dietary Interventions on Metabolic Profile and Hormonal Profiles in Women with Polycystic Ovary Syndrome (PCOS): A Systematic Review Study

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ABSTRACT

Polycystic ovary syndrome (PCOS) is the most common endocrine disorder in women of reproductive age, characterized by insulin resistance, visceral fat accumulation, dyslipidemia, and hyperandrogenism. The combination of metabolic and hormonal disorders worsens reproductive function, increases the risk of cardiometabolic disease, and decreases quality of life. Dietary interventions have become a promising non-pharmacological strategy for improving metabolic and hormonal regulation in PCOS patients. *This systematic review* was prepared following the guidelines of PRISMA 2020. Literature searches were conducted on PubMed, ScienceDirect, and the Cochrane Library until August 2025. Inclusion criteria included *randomized controlled trials* (RCTs) in PCOS women with a BMI ≥ 25 kg/m², which evaluated the effects of dietary interventions on metabolic and hormonal profiles. Of the 437 articles screened, seven studies (n = 406 participants) met the eligibility criteria and were critically analyzed. A review of seven RCT studies showed that dietary interventions such as ketogenic, VLCKD, Mediterranean, and low-calorie diets had a positive impact on metabolic and hormonal profiles in women with PCOS. The combination of licorice + a low-calorie diet is effective in lowering insulin resistance while the PMCD diet is more effective in lowering LDL and TG. Hormonal improvements were reflected in increased SHBG (p = 0.042) and decreased LH (p < 0.05), especially in PMCD and ketogenic diets. The low-AGEs diet also helps with weight loss, waist circumference, and HOMA-IR, although data are still limited. The KEMEPHY diet and Low-AGEs showed that both diets had a significant impact on VAT and VFL (p < 0.005), with only two studies explicitly describing these outcomes, so more research is needed. Dietary interventions are effective in lowering metabolic profiles, balancing hormonal regulation, and having a positive impact on visceral fat, in women with PCOS. KD and VLCKD are the most promising strategies, while PMCD and low-AGEs diets are beneficial for specific metabolic phenotypes. The principle of personal nutrition needs to be prioritized, although long-term clinical trials are still needed to confirm the effects on reproductive outcomes.

KEYWORDS

Diet intervention, visceral adipose tissue, lipid profile, hormone profile, polycystic ovary syndrome

ARTICLE INFORMATION

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1. Introduction

Polycystic ovary syndrome (PCOS) is the most common endocrine disorder found in women of reproductive age, with prevalence in Indonesia estimated at around 6.8% in women of reproductive age, while the prevalence in the world is estimated at 6-13% in women of reproductive age. PCOS is characterized by chronic anovulation, hyperandrogenism, and polycystic ovarian morphology, and is closely related to insulin resistance, central obesity, and an increased risk of metabolic syndrome (Azziz et al., 2016). Pathophysiologically, PCOS is characterized by metabolic disorders in the form of insulin resistance and compensatory hyperinsulinemia, which increase ovarian androgen production and interfere with follicle maturation (Dunaif, 2012). This insulin resistance also contributes to dyslipidemia (high triglycerides, low HDL, increased LDL) and the risk of type 2 diabetes and cardiovascular disease (Dapas & Dunaif, 2021). Hormonal disorders such as hyperandrogenism, increased luteinizing hormone

(LH), and abnormal LH/FSH ratios exacerbate reproductive dysfunction, trigger chronic anovulation, and worsen clinical symptoms such as hirsutism and infertility (Conway et al., 2014). Thus, this combination of metabolic and hormonal disorders not only increases the risk of chronic diseases, but also decreases the quality of life of PCOS patients.

One of the important aspects of PCOS is the accumulation of visceral fat, which plays a more important role than subcutaneous fat in worsening insulin resistance and systemic inflammation (Carmina et al., 2020). Visceral fat increases the release of free fatty acids and pro-inflammatory cytokines, which further exacerbate metabolic dysfunction and hormonal disorders (Ibrahim, 2010). Dietary interventions are one of the main non-pharmacological approaches to address this condition. Various dietary patterns have been researched, including low-carb, high-protein diets, Mediterranean diets, and calorie restriction. Studies show that energy restriction can reduce body weight and visceral fat, improve insulin sensitivity, and reduce hyperandrogenism (Moran et al., 2013). The Mediterranean diet has been shown to reduce inflammation, improve lipid profiles, and improve reproductive function (Barrea et al., 2019). In addition, a low-glycemic diet has been reported to improve the menstrual cycle and increase the likelihood of spontaneous ovulation in women with PCOS (Marsh et al., 2010).

Thus, dietary interventions have great potential to lower visceral fat, improve metabolic profiles (glucose, lipids, insulin resistance) and hormonal profiles (androgens, LH/FSH) in PCOS patients. However, the available evidence is still diverse and often inconsistent, especially regarding the most effective types of diets. Therefore, a systematic review is needed to summarize the current scientific evidence regarding the effectiveness of dietary interventions on visceral fat, metabolic profile, and hormonal profile in women with PCOS.

2. Method

2.1 The Research Questions

This research was compiled based on the Preferred Reporting Items for *Systematic Reviews and Meta-Analyses* (PRISMA) 2020 guidelines to ensure transparency and quality of reporting (Page et al., 2021). The main question in the study was: "Can specific dietary interventions improve metabolic profiles and evaluate their impact on hormonal profiles, as well as visceral fat in women with Polycystic Ovary Syndrome (PCOS)?" To answer this question, this study uses the *Population, Intervention, Comparison and Outcome* (PICO) framework, as shown in **Table 1**.

Table 1. The framework of Population, Intervention, Comparison, and Outcomes (PICO)

Component	Detail
<i>Population</i>	Women with PCOS of reproductive age (18-45 years), diagnosed by Rotterdam, NIH, or AE-PCOS criteria.
<i>Intervention</i>	Dietary interventions: low-calorie diets, specific diets such as <i>the Ketogenic Diet (KD)</i> , <i>the Very Low-Calorie Ketogenic Diet (VLCKD)</i> , <i>(Portfolio Moderate-Carb Diet (PMCD)</i> , <i>Low Advanced Glycation End Products (Low-AGEs)</i> or Mediterranean diet.
<i>Comparator</i>	Standard diet, placebo, or no specific dietary interventions.
<i>Outcomes</i>	Primary: metabolic profile (FPG, insulin, HOMA-IR, lipids profile, <i>Visceral Adipose Tissue (VAT)</i>) and hormonal profile (LH, FSH, total testosterone, SHBG)

2.2 Resources

The study used three main databases, namely PubMed, ScienceDirect, and the Cochrane Library, to obtain relevant literature. The literature search was conducted using a combination of keywords and *Medical Subject Headings* (MeSH) as follows:

"Polycystic Ovary Syndrome" OR "PCOS" AND "diet" OR "dietary intervention" OR "low-calorie diet" OR "low-carb diet" OR "Mediterranean diet" OR "ketogenic diet" AND "metabolic profile" OR "insulin resistance" OR "HOMA-IR" OR "glucose" OR "HbA1c" OR "visceral adipose tissue" OR "visceral fat" OR "adipose tissue" OR "lipid profile" OR "IMT" OR "waist circumference" OR "LH" OR "FSH". OR "SHBG".

2.3 Study Selection

This study only included studies assessing the effectiveness of dietary interventions against metabolic and hormonal profiles in women with polycystic ovary syndrome (PCOS). The selection was carried out using *eligibility criteria* in the form of inclusion and exclusion criteria compiled based on the PICO framework. The inclusion criteria for this study were studies with a *Randomized Controlled Trial (RCT) design*, which involved participants in the form of women of reproductive age with a diagnosis of PCOS based on the Rotterdam criteria and had a body mass index (BMI) of $\geq 25 \text{ kg/m}^2$ (overweight or obese category). The interventions

included were dietary modifications, either given singly or in combination with additional supplements or metabolic pharmacological agents.

The minimum duration of the intervention was four weeks, with *Outcome* reporting including at least one of the following indicators: BMI, body weight, insulin resistance (HOMA-IR or insulin levels), lipid profile (LDL, HDL, triglycerides, total cholesterol), as well as hormonal profiles (LH, FSH, testosterone, DHEA-S, SHBG) and visceral Fat (VAT). In addition, only articles written in English and accessible in full-text form are considered. The exclusion criteria included studies with non-RCT designs (cohort, *case-control*, *case report*, *review*, or editorial), participants who were not women with PCOS or were not in the *overweight/obese* category, and studies evaluating pharmacotherapy as the primary intervention without dietary components (e.g. oral contraceptives or single metformin). Articles are also excluded if they do not report quantitative data on results (e.g. no SD \pm mean or median/IQR available), or articles that are not available in full-text form.

The literature search strategy was carried out systematically on three international databases, namely PubMed, Science Direct, and the Cochrane Library, until August 2025 without restrictions on the year of initial publication. The search keywords use a combination of free terms and *Boolean search* as follows:

("polycystic ovary syndrome" OR PCOS) AND (overweight OR obese) AND (diet OR "calorie restriction" OR "ketogenic diet" OR "Mediterranean diet" OR "dulaglutide" OR "licorice") AND ("randomized controlled trial" OR RCT).

To narrow down the results, the search was limited to additional filters in the form of human studies, female, adult (18–45 years), and *English language*.

2.4 Data Extraction

The data extraction process is carried out by collecting information such as the title of the study, the year of publication, the type of study, the population, the intervention and controls used, and the results of the study. The data is then organized in the Microsoft Excel application to make analysis easier. Afterwards, the selected studies undergo a critical assessment to evaluate the quality of the study before being included in this systematic review.

3. Result

3.1 Study Selection

The selection and identification process of the study is shown in **Figure 1**. In the initial search, 437 studies were found. After the process of duplication and screening of irrelevant titles, the number of studies obtained amounted to 424 studies. The study was then selected by reviewing the title and abstract so that it was found that as many as 396 studies did not meet the *eligibility criteria* so that there were 28 studies that would be searched in full. A total of 8 studies were not found in complete articles, leaving 20 studies that will be reviewed in detail according to the *eligibility criteria* based on the complete text. In the end, there were 7 studies that met the *eligibility criteria* and were used in *this systematic review study*. The risk assessment of bias in this study used Review Manager (RevMan) software according to Cochrane guidelines, the results of the assessment included aspects of randomization, *blinding*, completeness of outcome data and reporting selectivity, which are shown in **Figure 1**.

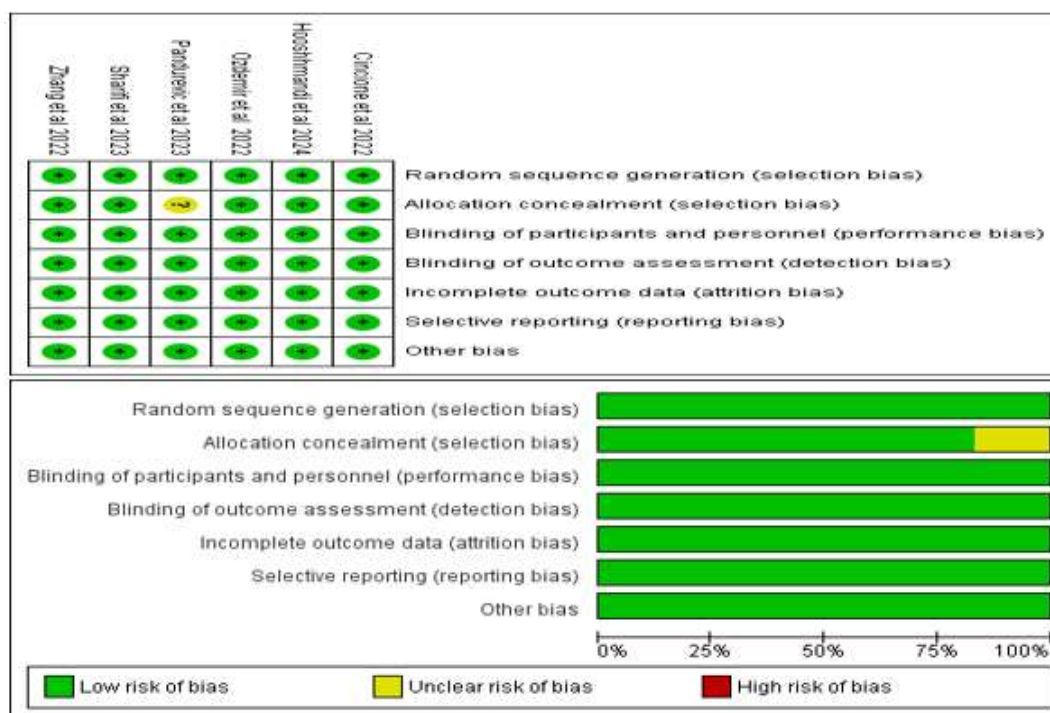


Figure 2. Risk assessment of bias in RCT studies

Evaluation of risk bias in randomized clinical trials (RCTs) using *the Risk of Bias 2 Tool* showed that most studies had a low risk of bias in almost all major domains, with potential bias limited to the allocation concealment aspect that was classified as an *unclear risk of bias*. Overall, the methodological quality of the study was considered adequate and the results were reliable, although one study that was evaluated using *ROBINS-I* with a single-arm study design had limitations in participant reporting, but the data obtained remained consistent and objective in measuring outcomes. His findings contribute as supporting evidence in the narrative synthesis and are carefully considered in the overall interpretation of the results.

Study Characteristics

The studies included in this systematic review were published between 2020 and 2025 totaling 7 studies with a total sample of 406 patients. The studies included in this study have a *Randomized Controlled Trial* (RCT) research design located in Iran, Italy, Turkey, and China. All of the studies included in this systematic review assessed the effectiveness of dietary interventions on the metabolic, or hormonal and visceral fat profiles in women with Polycystic Ovary Syndrome (PCOS). The characteristics of the studies included in this study are shown in **Table 2**.

Table 1 Summary of controlled clinical trials (RCTs) in women of reproductive age with PCOS evaluating dietary and/or pharmacological interventions on anthropometric, metabolic, lipid, and hormonal indicators.

No	Author (Year)	Study Design	Age	Number of Participants (Intervention/Control)	Duration	Intervention/ Control	Primary Outcomes	Research Results
1	Hooshmandi et al. (2022)	RCT, double-blind	18-45 year	66 (33/33)	8 weeks	Intervention: <i>Licorice extract</i> (1.5 g/day) + low-calorie diet Control: Placebo + low-calorie diet	BMI, BF, FPG, insulin, HOMA-IR, lipid profile	This study assessed the effects of <i>licorice</i> supplementation combined with a low-calorie diet in female patients with PCOS. Decrease in BMI (kg/m ²) -1.64 ± 0.07 ; p<0.001 (I) 0.01±-0.0; p 0.25(K), BF -2.45 ± -0.82; p<0.001 (I) -0.01±0.00; p 0.97 (K), lipid profile (mg/dl) in group (I) TG, TC (p<0.001), LDL(p=0.01), and in group (K) TG (p=0.16), TC (p=0.21), LDL (p=0.71). The increase means HDL (p<0.001 (I) p=0.85 (K), FPG, insulin levels, Homa-IR and Homa-B, while in group (I) (p<0.001) and FPG (p=0.07), insulin (p=0.98), Homa-IR (p=0.16), HOMA-B (p=0.09) in group (K). The results of this study showed that the combination of <i>licorice</i> and a low-calorie diet led to improvements in obesity index, glucose homeostasis, lipid profile compared to the placebo group.
2	Cincione et al. (2022)	RCT	18-45 years old	144 (73/71)	6 weeks	Intervention: Ketogenic Diet (KD) Control: Mediterranean Diet (MD)	BMI, FPG, lipid profile, insulin, HOMA-IR, hormone profile	Significant changes before and after treatment in Anthropometric parameters (BMI (kg/m ²) – 4,15 ± 1.31; p<0.001), Metabolic (FPG – 13.23 ± 5.64 (KD) and – 4.53 ± 4.11 (MD), Insulin – 21.64 ± 15.34 (KD) and – 6.87 ± 7.52 (MD), HOMA-IR (– 5.70 ± 3.94 (KD) and – 1.90 ± 1.97 (MD)), Hormones (LH – 5.51 ± 3.23 (KD) and – 3.07 ± 1.80(MD) with p values <0.003), TT – 7.40 ± 4.01 (KD) – 5.30 ± 4.07 (MD) p 0.02, FSH increase of 2.59 ± 1.04 (KD) 1.26 ± 0.63(MD) p <0.001, and SHBG (18.08 ± 8.84 (KD) 9.10 ± 3.89 (MD) p < 0.001), These results

								showed a significant decrease in all parameters after receiving the KD and MD dietary interventions, where the decrease was more significant in the KD group than in the MD group
3	Sharif et al, (2023)	RCT, open-label	18-45 years old	40 (21/19)	8 weeks	Intervention: Portfolio Moderate-Carb Diet (PMCD) Control: Ketogenic Diet (KD)	BMI, FPG, insulin, HOMA-IR, lipid profile, hormonal profile	The mean difference in the KD group showed a more significant improvement compared to the (PMCD) group on the decrease in BMI (kg/m ²) (2.73±0.35 (KD)) compared to 1.71±0.775 (PMCD) with values (p <0.05) and LH and LH (4.13±1.375 (KD) compared to 2.46±1.105 (PMCD), while the lipid profile including LDL and TG showed a more significant improvement in the PMCD group compared to the KD group (33.95±7.345 compared to 23.34±14.136 and 38.20±10.757 compared to 57.62±21, 688). These results showed that both diets showed improvement, but were more effective in the KD group in improving metabolic factors, anthropometry, and reproductive hormone levels compared to the PMCD diet in patients with PCOS women with obesity. In addition, the PMCD diet is more beneficial in PCOS women with fat profile disorders.
4	Pandurevic et al, (2023)	RCT, open-label	18-45 years old	30 (15/15)	16 weeks	Intervention: VLCKD (8 weeks) + LCD (8 weeks) Control: Mediterranean LCD (16 weeks)	BMI, FPG, lipid profile, hormone profile	Group (I) showed superiority over group (K) in terms of reducing BMI (kg/m ²) (-4.60±0.10; p<0.001 and- 1.70±0.60; p=0.03) with a selective decrease in fat mass (-9.30±1.60; p=0.0014 and - 3.10±2.00; p=1.00), insulin resistance (group (I) p=0.024, group (K) p>0.05) and significant differences in Free testosterone (FT) (pg/mL) and SHBG from the two groups after 16 weeks (p=0.0009 and p=0.0004). Ovulation increased significantly from 38.5% to 84.6% (p=0.031), compared to the Mediterranean diet which only increased from 14.3% to 35.7% (p>0.05). The study expands the possibilities of therapeutic approaches in obese women with PCOS.
5	Paoli et al, (2020)	RCT (non-blind)	18-45 years old		12 weeks	Intervention:	BMI, FPG, VAT, lipid profile, hormone profile	Significant decreases occurred in the metabolic profile of FPG (mg/dl) (91.8 ± 4.5 →83.5 ± 4.3 p <0.0001), BMI (- 3.35; p < 0.0001), insulin and HOMA-IR (p<0001), FBM (- 8.29 kg; p <

						Ketogenic Mediterranean Diet with Phytoextracts (KEMEPHY) Controls : -	0.0001) including VAT (1750 ± 181.58 grams $\rightarrow 1110.36 \pm 189.23$ grams; $p < 0.0001$) and decreased lipid profile (TC, TG, LDL experienced a significant decrease in $p < 0.0001$) accompanied by an increase in HDL $p < 0.0146$) in group (I). Hormone profile (decrease in statistical significance in LH levels ($10.24 \pm 1.43 \rightarrow 6.41 \pm 1.46$; $p < 0.0001$), TT (47.43 ± 6.08 ng/dL $\rightarrow 40.71 \pm 5.77$ ng/dL; $p < 0.0001$), while FSH and SHBG levels increased ($p = 0.0258$ and; $p < 0.0001$). The results of this study show that the Mediterranean Diet Ketogenic + <i>phytoextract</i> (KEMEPHY) can improve metabolic profile, lipid profile, improvement of hormone profile and loss of visceral fat which can be considered as a non-pharmacological therapy for PCOS.
6	Ozdemir et al, (2025)	RCT11	19-35 years old	44 (22/22)	12 weeks	Intervention: Low-AGEs Diet Control: Standard AGEs Diet	BMI, BF,VFL, FPG, lipid profile, hormone profile, TNF- α Group (I) showed a more significant decrease in BMI (kg/m^2), BF (%), VFL, Insulin Resistance (-3.2 (95% IK -4.0 to -2.0 ; $p = 0.001$), -2.2 (95% IK -3.3 to -1.3 ; $p = 0.001$), -2.4 (95% IK -4.4 to 1.8 ; $p = 0.001$), -0.8 (-1.4 to 0.1); $p = 0.016$) compared to the group (K), but there was no significant difference in the two groups ($p > 0.05$). Meanwhile, group (I) was better at reducing FPG (mg/dl) -8.5 (95% IK -11.5 to 3.5 ; $p = 0.064$) than group (K) ($p = 0.346$) with a difference in value ($p = 0.027$) between the 2 groups. The decrease in LDL(mg/dL), TNF- α (ng/L), TT (ng/dL) was significant in group (I) than in group (K) with values ($p = 0.046$, $p = 0.004$, $p = 0.009$), while SHBG (nmol/L) showed a significant increase (0.042 in group (I)). Meanwhile, this parameter showed no significant improvement in the control group ($p = 0.856$, $p = 0.063$, $p = 0.215$, $p = 0.285$). Reducing dietary intake AGEs provides significantly better results in metabolic and hormonal profiles, especially those accompanied by signs of inflammation in PCOS Phenotype A patients.
7	Dulaglutide Trial RCT, 2022	open-label	18-45 years old	68 (35/33)	Up to 7% weight loss or max 6 months	Intervention: Dulaglutide + Calorie-Restricted Diet	BMI, Hba1c, PPG, VAT area, lipid profile, hormone profile Compared to group (K), the average time of group (I) was faster in achieving a 7% BB reduction and significantly in lowering Hba1c(%) $p = 0.027$ as well as PPG $p = 0.021$ was also significantly different from $p < 0.05$. Both groups managed to significantly reduce the VAT area (I): -18.44 cm^2 , IK 95%: -27.44 to -9.43 , (K): -17.47 cm^2 (IK 95%: -27.37 to -7.56). There was no significant

	Control: Calorie-Restricted Diet	difference between the two groups in the change in area from the VAT reduction (p=0.884). Group (K) showed a statistically significant decrease in total testosterone parameters of -0.22 (95% CI -0.43 to -0.01) compared to group (I). No significant changes were found in the parameters (LH, FSH, or SHBG either in either group.
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4. Discussion

The results of a *systematic review* of seven RCTs showed that dietary interventions had a significant impact on metabolic and hormonal profiles in women with PCOS. Consistently, low-carb diets, especially ketogenic diets (KD) and *very low calorie ketogenic diets* (VLCKD), provide the most noticeable improvements compared to other dietary interventions. In the study of Zhang et al., the *calorie restricted diet* (CRD) intervention with or without dulaglutide showed a significant decrease in area VAT (dulaglutide + CRD: -18.44 cm^2 , 95% CI: -27.44 to -9.43 , CRD: -17.47 cm^2 (95% CI: -27.37 to -7.56). There was no significant difference between the two groups in area change from the VAT decrease ($p=0.884$), but the dulaglutide group showed significant improvements in HbA1c ($p = 0.027$) and glucose 2 hours post prandial ($p=0.021$) (Zhang et al., 2023). The results of the analysis showed differences in changes in metabolic profile, hormones and body composition but no significant differences between the 2 groups. The results of this study support the importance of dietary interventions as the primary therapy and GLP-1RA provides additional therapy used to achieve weight targets in women with PCOS. This confirms that calorie restriction remains fundamental in the management of PCOS, while GLP-1 agonists only act as adjuvant for glycemic improvement.

In line with that, the study of Paoli et al, (2020) using the *Ketogenic Mediterranean Diet* (KEMEPHY) also showed a significant decrease in VAT along with body weight (-9.43 kg , $p<0.001$), BMI (-3.35 , $p<0.01$), and fat body mass (-8.29 kg , $p<0.001$). This decrease in VAT is important because visceral fat tissue plays a role in the production of pro-inflammatory cytokines and worsens insulin resistance, so improvements in these fat compartments contribute directly to decreased hyperandrogenism and increased insulin sensitivity.

In contrast, the combination of *licorice extract* and *low-calorie diet* (LCD) resulted in greater improvements than LCD alone. The study of Hooshmandi et al, found a decrease in BMI (kg/m^2) -1.64 ± 0.07 ; $p<0.001$ (I) 0.01 ± 0.01 ; $p=0.25$ (K.), BF -2.45 ± -0.82 ; $p<0.001$ (I) -0.01 ± 0.00 ; $p=0.97$ (K). Lipid profile (mg/dl) in the triglyceride intervention group, total cholesterol, LDL ($p<0.05$), and in the triglyceride control group, total cholesterol, LDL ($p>0.05$), as well as a decrease in HOMA-IR by -1.7 in the *licorice* group compared to -0.8 in the control group ($p=0.03$) (Hooshmandi et al., 2024). The glycyrrhizic acid content in licorice is known to have *insulin-sensitizing* and anti-inflammatory properties, which support the improvement of these metabolic variables.

Meanwhile, a comparison of the *Moderate-Carbohydrate Diet* (PMCD) and KD Portfolios (Sharif et al., 2024) shows different patterns of results. KD is superior in reducing BMI (kg/m^2) with an MD value of 2.90 (IK95 2.73, 3.07; $p<0.05$) versus PMCD 1.71 (95% IK 1.36, 2.07; $p<0.05$), LH 4.38 (95% IK 3.72, 5.05; $p<0.05$) compared to PMCD 2.48 (95% IK 1.98, 2.98; $p<0.05$), and increased FSH (nmol/L) -0.68 (IK95 0.69, 0.40; $p<0.05$) compared to PMCD -0.24 (95% IK 95%, -0.54 , -0.35 $p<0.05$), while PMCD was better at correcting LDL-C (mg/dL) with an MD value of 33.8 (95% IK 30.46-37.15; $p<0.05$) compared to KD 21.52, (IK95%, 14.71-28.33 $p<0.05$). Biologically, this corresponds to the mechanism: the ketogenic diet lowers insulin and improves hyperandrogenism, while plant-based PMCDs with soluble fiber and phytosterol content have more effect on lipid profiles.

The effectiveness of KD was further strengthened by Paoli et al, (2020), who used the Ketogenic Mediterranean Diet (KEMEPHY). After 12 weeks, there was a significant decrease in body weight (-9.43 kg , $p<0.001$), BMI (-3.35 , $p<0.01$), fat body mass (-8.29 kg , $p<0.001$), and improvement in HOMA-IR (-3.1 , $p<0.01$). Reproductive hormones also improve, with a decrease in *free testosterone* (-0.4 ng/mL , $p=0.0009$) and LH/FSH ratio (-1.2 , $p<0.05$). These findings are in line with translational studies showing that ketosis lowers insulin levels, increases SHBG, and lowers free testosterone (Moran et al., 2013), A significant decrease also occurs in VAT (-640 grams , $p<0.0001$). Biologically, the decrease in visceral fat decreases systemic inflammation and insulin resistance, thereby reducing ovarian hyperandrogenism and improving ovulation (Legro et al., 2015).

The most significant results were found in the group that followed a diet low in the compound Advanced Glycation End Products (Low-AGEs). This group experienced a significant decrease in BMI (-3.2 kg/m^2 ; 95% CI: -4.0 to -2.0 ; $p=0.001$), body fat percentage (-2.2% ; 95% IK: -3.3 to -1.3 ; $p=0.001$), visceral fat (-2.4% ; 95% IK: -4.4 to -1.8 ; $p=0.001$), and insulin resistance (HOMA-IR) (-0.8 ; 95% IK: -1.4 to -0.1 ; $p=0.016$). A decrease in fasting blood glucose (FPG) of -8.5 mg/dL ($p=0.064$) was not statistically significant, but was more significant in the diet group Low-AGEs were compared to groups with standard diets. There was a significant difference between groups in fasting blood glucose ($p=0.027$). In addition, there was a significant decrease in LDL ($p=0.046$), TNF- α ($p=0.004$), and TT ($p=0.009$), as well as a significant increase in SHBG ($p=0.042$). Meanwhile, the control group showed no significant changes in these parameters. These findings indicate that diets low in AGEs are more effective in improving visceral, metabolic (FPG and LDL), hormonal (TT, and SHBG) fat profiles and also of interest are inflammatory signs (TNF- α) in PCOS phenotype A patients compared to standard diets. The Low-AGEs diet, for example by avoiding high-temperature cooking methods has been associated with improvements in metabolic and hormonal profiles as well as signs of inflammation including increased insulin sensitivity and decreased oxidative stress in PCOS patients (Garg D et al., 2015).

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The most striking findings came from VLCKD (Pronokal method), Pandurevic et al., (2023) showing a decrease in BMI (−13.7% vs −5.1%, $p=0.0003$), WC (−11.4% vs −2.9%, $p=0.0008$), BF % (−24.0% vs −8.1%, $p=0.0176$), and FT (−30.4% vs −12.6%, $p=0.0009$) after 16 weeks. In addition, ovulation increased significantly from 38.5% to 84.6% ($p=0.031$), compared to the Mediterranean diet which only increased from 14.3% to 35.7% ($p>0.05$). This suggests that VLCKD not only improves metabolic and hormonal variables, but also its impact on reproduction.

The consistency of KD's superiority is also seen in short-term studies, Cincione et al., (2022) reported that over 6 weeks, KD resulted in greater reductions in HOMA-IR (−2.8 compared to controls −1.1, $p<0.001$), LH/FSH ratio (−0.9 compared to controls −0.3, $p<0.05$), and FT (−1.2 compared to controls −0.5 ng/mL, $p<0.05$) compared to Mediterranean diets. Another study by Paoli et al., (2020) also corroborates this evidence with a decrease in insulin (−6.2 $\mu\text{U/mL}$, $p<0.01$) and an increase in SHBG (+12 nmol/L, $p<0.05$), which plays a role in lowering free testosterone. When compared to the previous literature, these results reinforce the meta-analysis of Al Khalifah et al., (2021) who stated that a low-carbohydrate diet provides significant improvement in HOMA-IR (WMD −0.63, $p<0.001$) and total testosterone (WMD −0.21 nmol/L, $p=0.04$). However, this review further shows that VLCKD even increases ovulation by 46%, which gives an idea of positive effects on reproduction that have not been shown in previous meta-analyses.

Overall, the main biological mechanisms of these findings are: (1) a low-carb diet lowers insulin and glucose levels which can reduce ovarian stem cell stimulation, which in turn decreases androgen production; (2) the reduction of visceral fat lowers inflammatory cytokines (IL-6, TNF- α) which can improve insulin sensitivity; (3) ketosis improves fat oxidation, activates AMPK/SIRT1, and suppresses androgen production; (4) plant-based diet (PMCD) lowers lipids through soluble fiber and phytosterols; (5) licorice provides insulin-sensitizing effect; and (6) GLP-1 agonist (dulaglutide) improves glycemic control without affecting androgens, (7) Low-AGEs diet can improve oxidative stress especially inflammatory signs (TNF- α)

The clinical implications of these findings are quite clear: KD and VLCKD can be considered as non-pharmacological first-line therapies for obese PCOS women with severe insulin resistance, especially if infertility is a major problem. PMCD may be more appropriate in patients with dominant dyslipidemia, while licorice or dulaglutide act as adjuvant. The LOW-AGES diet corresponds to signs of inflammation in PCOS women. The principle of personalized nutrition must be prioritized to adjust dietary patterns to the metabolic and reproductive profile of each patient. However, it is worth noting a number of limitations: most RCTs had small sample sizes (14–72 participants), short durations (8–16 weeks), and difficult dietary protocol variations. Long-term outcomes such as *pregnancy rates* or *live births* are also rarely studied. In addition, dietary adherence is often measured through self-reports, which is at risk of bias. The next direction of research should be focused on long-term multicenter RCTs (≥ 12 months) with large samples, which evaluate primary reproductive outcomes. It is also necessary to conduct *direct comparative effectiveness trials* between KD, VLCKD, and PMCD, as well as translational research on the molecular mechanisms of ketosis on ovarian function.

5. Conclusion

Dietary interventions such as the ketogenic diet, VLCKD, Mediterranean diet, PMCD and low-calorie diets have a positive impact on women with PCOS. Improvements in metabolic profile were seen in the combination of licorice + low-calorie diet and PMCD diet, which lowered TG more significantly than the ketogenic diet. BMI reduction was effective with the seven interventions of the licorice diet + low-calorie diet, PMCD, VLCKD, KD, Low-AGEs and KEMEPHY. The ketogenic diet and VLCKD lead to more dominant improvements in metabolic and hormonal profiles in women with PCOS, with VLCKD even showing unique benefits in increasing ovulation. The hormone profile showed an increase in SHBG and a decrease in LH, especially in the VLCKD and ketogenic diet groups. Inflammatory signs such as TNF- α show a decrease in the LOW-AGES diet. Calorie-restricted dietary interventions with or without Dulaglutide also showed potential in reducing BB by 7% and were superior in controlling HbA1c (%) and PPG levels compared to calorie-restricted diets alone. The KEMEPHY diet and Low-AGEs indicate a positive effect on visceral fat (VAT and VFL) which may contribute to lowering the symptoms of women with PCOS. Only a handful of studies explicitly evaluated visceral fat as a primary outcome, so the effectiveness of diet on this variable still requires further research. Despite methodological limitations, this evidence supports that dietary interventions are not merely complementary but a key pillar of PCOS therapy. The integration of *personalized* diet with selective pharmacotherapy has the potential to be an optimal strategy to be effective, safe, and sustainable in improving quality of life, metabolic function, and fertility in PCOS.

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