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Review Article

A Systematic Review on Lifestyle and Nutritional Management of Polycystic Ovary Syndrome

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Abstract

Here, we present a narrative review of the widely understood changes to the nutrition and lifestyles of women and girls with polycystic ovary syndrome (PCOS). The database was analysed, combining PCOS entries with causes, diseases, diet supplementation, lifestyle, physical activity, and use of herbs. This study explains how different biochemical routes contribute to imbalances in lipid, carbohydrate, and hormone regulation among affected individuals. It also explores links with sleep problems, physiological and psychological shifts, and stress-related inflammation. These conditions consistently lead to the occurrence of severe diseases in patients suffering from diabetes, the fatty degeneration of internal organs, infertility, atherosclerosis, cardiovascular diseases and cancer. Change in lifestyles, diet patterns and proper selection of nutrients, pharmacological and natural supplementation in the form of herbs, and physical activity have been proposed. The progress and consequences of PCOS are largely modifiable and depend on the patient's effort, although we have to take into account the genetic determinants.

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is a prevalent hormonal disorder that affects a significant portion of women during their reproductive years, estimated at roughly one-fifth of this population [1]. In 2003, international reproductive medicine experts meeting in Rotterdam revised the diagnostic standards, leading to broader recognition of the syndrome's diverse clinical forms [2]. This diversity presents challenges for management, yet many patients show overlapping metabolic characteristics that are important for both evaluation and therapy [3].

Many studies have shown that higher hormone levels, gut microbiome composition, and plasma metabolomics are new parameters related to the PCOS phenotypes [4]. The clinical phenotypes can change over the life span with higher weight gain, and can found in the same patient. Individualised treatment remains the main approach, but grouping the phenotypes and following Therapeutic guidance may also have clinical value. Early adoption of well-defined management strategies is essential, particularly for females with PCOS, who face an elevated risk of developing endometrial or ovarian malignancies. [5,6]. Therefore, therapeutic strategies that incorporate anti-inflammatory agents as adjuncts to anticancer treatment are important. Such approaches can disrupt harmful signalling

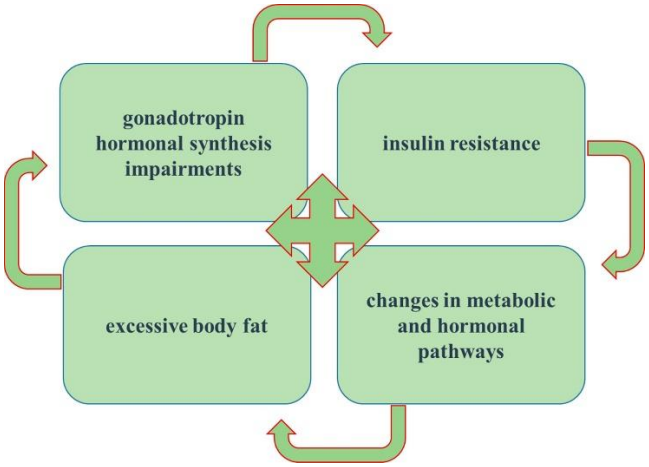
pathways, contributing to improved survival rates, quicker recovery, and enhanced quality of life for patients.

1.1. Physiological Basis

The four main causes of the physiological basis of PCOS include:

- disorders of gonadotropin hormonal synthesis;
- the appearance of insulin resistance;
- the influence of the present excessive body fat; and finally,
- the metabolic pathways involved in PCOS (the secretion and activity of insulin, encoding for steroidogenesis, and other metabolic and hormonal pathways) (Figure 1) [7].

Figure 1. Main pathophysiological basis of polycystic ovary syndrome (PCOS)-disorders of gonadotropin hormonal synthesis, the appearance of insulin resistance, the influence of the present excessive body fat and oblique metabolic pathways involved in PCOS.



Appropriate functioning of the mechanisms responsible for the maturation of the ovarian follicle and its ovulation depends on the proper physiological activity of three organs: the hypothalamus, pituitary gland, and ovaries.

Hormonal control within the hypothalamic–pituitary–ovarian (HPO) axis operates through long, short, and ultrashort negative feedback mechanisms. Neurons in the hypothalamic suprachiasmatic region synthesise gonadotropin-releasing hormone (GnRH), which enters the pituitary portal circulation via the median eminence. Its release depends on neuronal network activity and occurs in pulses that determine gonadotropin output. Slower GnRH pulses favour secretion of follicle-stimulating hormone (FSH), whereas faster pulses promote luteinizing hormone (LH) release from the anterior pituitary. LH drives corpus luteum formation and progesterone synthesis, while FSH supports follicular maturation and estrogen production by activating aromatase in granulosa cells. When LH predominates over FSH, androgen synthesis increases, a pattern often observed in polycystic ovary syndrome (PCOS) [8].

Insulin also contributes to PCOS pathophysiology by acting with LH to elevate androgen output and by reducing hepatic production of sex hormone-binding globulin (SHBG), thereby increasing free testosterone levels [8]. Excess adipose tissue further aggravates these processes because adipocytes release hormones such as leptin and resistin and produce inflammatory mediators, including interleukin-1β and tumour necrosis factor-α [9]. The activity of leptin affects the function of the hypothalamus–pituitary gland–ovary axis by modifying the secretion of GnRH, LH, and FSH. Leptin acts on the hypothalamus to influence the release of gonadotropins, indirectly stimulating luteinizing hormone (LH) secretion and promoting gonadotropin-releasing hormone (GnRH) activity. This mechanism may enhance androgen production. Adipose tissue also releases inflammatory cytokines that sustain oxidative stress and inflammation in PCOS, conditions intensified by hyperglycemia, excess fat mass, and elevated androgens [8].

The clinical diversity of PCOS reflects the involvement of numerous metabolic pathways. These include insulin signaling and its related genes—such as those coding for the insulin receptor (IR), insulin (INS), and insulin-like growth factor (IGF) and its receptor—as well as genes linked to steroid hormone synthesis, cytochrome P450 activity (CYP17, CYP11A1), and hormone receptor function, including androgen receptor (AR), LH receptor, leptin, and follistatin [10]. Dietary habits emphasising anti-inflammatory foods and low glycemic index or reduced-fat intake appear to lower the risk of PCOS development [11,12].

1.2. Improvement in Metabolic Pathways

1.2.1. Insulin Resistance

Weight gain mediates most of its direct medical sequelae through worsening insulin sensitivity.

Insulin resistance (IR) is central to the onset of metabolic disorders such as hypertension, impaired glucose control, and abnormal lipid profiles. Research indicates that mitochondrial impairment contributes to IR, often triggered by excess lipid accumulation in non-adipose tissues. The resulting oxidative stress in skeletal muscle increases reactive oxygen species (ROS) generation, further disrupting mitochondrial function and insulin signalling [13]. This mechanism links IR to obesity-associated conditions, including polycystic ovary syndrome (PCOS). The cellular effects of insulin occur through two main post-receptor pathways: the phosphatidylinositol 3-kinase (PI3K) and the mitogen-activated protein kinase (MAPK) pathways [14]. The PI3K pathway regulates cellular intermediary metabolism, whereas the MAPK pathway controls growth processes and mitoses [14]. AKR1C3 expression in adipocytes leads to the occurrence of insulin resistance and hyperinsulinemia, then drives a vicious circle of intra-adipose androgen activation, lipid accumulation, and hy- hyperinsulinemia [15]. Kauffman et al. suggested that ethnicity has an additive effect on insulin resistance in PCOS. Mexican American women showed significantly higher insulin levels. Resistance compared with Caucasian American women [16].

1.2.2 Oxidative Stress and Chronic Inflammation

The association between body weight and IR is mediated through inflammatory pathways [17]. Obesity causes changes in the release of key cytokines and adipokines, which in turn manifest in paracrine and endocrine effects. The increased levels of leptin

and plasminogen activator inhibitor-1 and the reduced release of adiponectin result in a generalised low-grade inflammatory response. This process is mediated by macrophages and other immune cells.

Elevated oxidative stress markers—such as reactive oxygen species (ROS), p47phox expression, and thiobarbituric acid-reactive substances (TBARS)—have been observed in women with PCOS following consumption of saturated fats, even when obesity is not present. Diets high in refined sugars and saturated fatty acids further intensify ROS formation through several pathways, including alterations in gut microbiota composition [18]. Both circulating immune cells and excess adipose tissue contribute independently to the oxidative imbalance characteristic of PCOS [19]. Lipid-driven oxidative stress appears to play a central role in the onset of insulin resistance and hyperandrogenism, with adipose tissue acting as an additional pro-oxidant source and modulator of insulin signalling [19]. Chronic androgen exposure also increases oxidative stress in pancreatic islet cells, leading to mitochondrial impairment [20,21].

Superoxide is a ROS produced when NADPH is oxidised by membrane-bound NADPH oxidase [22]. Abnormal generation of reactive oxygen species (ROS) by NADPH oxidase contributes to cardiovascular complications—such as endothelial dysfunction, atherosclerosis, and hypertension—that are frequently seen in women with PCOS [23]. Oxidative stress triggered by peroxides activates the nuclear factor κB (NF-κB) pathway, a major regulator of inflammation that enhances the transcription of the tumour necrosis factor-α (TNF-α) gene [24]. Intake of saturated fats can further intensify oxidative responses, promoting TNF-α release from leukocytes [19,25]. Our findings also indicate elevated TNF-α synthesis in women with PCOS [4]. Those with normal or low androgen concentrations, based on total testosterone and the free androgen index (FAI), appear particularly sensitive to TNF-α–driven oxidative and inflammatory changes [26].

1.2.3 Anticancer Protection

Many studies have targeted the inactivation of the transcription factor (NRF2) as a therapeutic approach in various types of cancer [27]. NRF2 was first recognised in anticancer research as an inducer of several antioxidant enzymes. It can protect cells and tissues against many types of toxicants that interrupt essential biochemical processes and carcinogens by increasing the expression of cytoprotective genes [28]. The transcription factor NRF2 exhibits context-dependent behaviour, functioning either as a tumour suppressor or as a promoter of tumour progression, depending on the biological setting in which it is activated [29]. Moderate activation of NRF2 in healthy cells can limit oxidative damage and genomic instability, thereby lowering cancer risk. Conversely, persistent or constitutive NRF2 activity in malignant cells can promote survival advantages, treatment resistance, and poor clinical outcomes, often necessitating therapeutic inhibition of the pathway [29]. NRF2 stability is regulated through at least three distinct mechanisms. One involves the cytoplasmic repressor KEAP1 [30]; another relies on β-transducin repeat-containing protein (β-TrCP) [31]; and a third is mediated by the endoplasmic reticulum–associated E3 ubiquitin ligase HRD1 [32].

The abnormal activation of the NRF2/KEAP1 pathway promotes cancer develop- ment [33], metastasis formation [34], and even resistance to ovarian cancer therapy [35]. Mutations in the KEAP1 gene induce the hyperactivation of the NRF2/KEAP1 pathway. Notably, KEAP1 missense or nonsense mutations were reported in endometrial carcinomas [36], as well as gall bladder [37], breast [38,39], cervical [40], and ovarian [41,42] cancers. MicroRNA miR-141 was the first-identified miRNA to directly repress KEAP1 levels in ovarian carcinoma cell lines [43].

1.3. Gut Microbiota Dysbiosis

The structural and functional dysbiosis of the gut microbiota in high-fat diet (HFD)- induced obesity was demonstrated in a mouse model [44]. Gut microorganisms and their metabolites exert broad influences on appetite regulation, lipid and glucose metabolism, and overall body weight control [44,45]. The intestinal microbiome can modulate roughly 10% of the host transcriptome, affecting immune, metabolic, and proliferative gene networks [46]. Dietary fibre, fermentation processes, and probiotic intake have therefore become major research areas in metabolic health [47]. Evidence shows that dietary fibre can restore microbial balance in individuals with type 2 diabetes [48]. Growth of Bifidobacterium species supports insulin release, enhances glucose tolerance, improves insulin sensitivity, and reduces inflammation. Beneficial microbes also generate short-chain fatty acids (SCFAs), including acetate and butyrate, which influence blood glucose via enteroendocrine hormones such as glucagon-like peptide-1 (GLP-1) and peptide YY (PYY) [45,49]. PYY, acting locally in the gut and centrally in the brain, contributes to appetite regulation. Because SCFAs are integral to lipid and carbohydrate metabolism, maintaining a healthy gut microbiota is an important therapeutic aim for reducing inflammation and preventing urogenital tract infections [50–52].

2. Lifestyle Changes

Lifestyle modification remains the primary therapeutic approach in the management of polycystic ovary syndrome (PCOS), though it complements rather than replaces pharmacological therapy [7]. Consistent physical activity, maintenance of healthy body weight, balanced dietary habits, and the avoidance of smoking are fundamental in the prevention and treatment of metabolic complications. These elements are incorporated into most clinical guidelines addressing metabolic and reproductive health. Attention to psychological well-being and stress reduction is also essential, as sustained behavioural change contributes to overall quality of life. Nutritional counselling has long been a cornerstone of PCOS management. However, studies indicate that severe caloric restriction rarely achieves lasting metabolic or hormonal improvement [53,54]. Even isocaloric diets, when paired with physical activity, may not significantly alter biochemical or anthropometric parameters [55].

2.1. Diet

Examination of macronutrient composition—relative proportions of protein, fat, and carbohydrates—has revealed no major differences in key metabolic indicators. Instead, total caloric reduction and the adoption of a diet with a low glycemic index (GI) are the most consistent predictors of clinical improvement [56,57]. Low-GI (LGI) diets have been shown to reduce insulin resistance (HOMA-IR), fasting insulin, total and LDL cholesterol, triglycerides, waist circumference, and total testosterone compared with high-GI (HGI) diets, without major changes in fasting glucose, HDL, body weight, or the free androgen index [58]. Combining an LGI diet with moderate caloric restriction, physical activity, and omega-3 fatty acid supplementation further increases HDL levels, promotes synthesis of sex hormone–binding globulin (SHBG), and reduces adiposity [8].

Dietary patterns rich in saturated fatty acids (SFA) can elevate circulating tumour necrosis factor-α (TNF-α) and leukocytic suppressor of cytokine signalling-3 (SOCS-3) expression, suggesting that limiting SFA intake is particularly important for PCOS management [25]. Sources of α-linolenic acid, such as flaxseed oil, have demonstrated favourable effects on hormonal and inflammatory markers in animal models, implying potential benefits in humans as well [59].

Soluble, fermentable dietary fibre promotes short-chain fatty acid (SCFA) formation, which supports a healthy gut microbiome and improves metabolic outcomes [60]. Diets with a low GI also modulate appetite-regulating hormones, decreasing ghrelin and increasing glucagon secretion in women with PCOS [12,61]. Conversely, excessive fructose intake may exacerbate endocrine abnormalities, worsening hormonal imbalances despite limited metabolic changes [62]. Meta-analyses confirm that LGI diets are safe, practical, and effective for improving insulin resistance, underscoring the need for individualised dietary guidance in all PCOS patients [63,64].

The ketogenic diet (KD) represents another form of carbohydrate restriction that replaces a portion of dietary carbohydrates with plant-derived fats. In women with PCOS—especially those with obesity or fatty liver disease—KD has been associated with improved menstrual regularity, reduced body mass, lower glucose and insulin levels, and better liver function [65]. A 12-week trial demonstrated significant reductions in body weight (≈9.4 kg), BMI (≈3.3 units), and fat mass (≈8.3 kg), along with improved lipid profiles, reduced triglycerides and LDL cholesterol, elevated HDL cholesterol, and normalisation of hormonal ratios, including LH/FSH and androgens [66]. Estradiol, progesterone, and SHBG levels increased, while the Ferriman–Gallwey score showed a modest decline, indicating partial improvement in hirsutism. The absence of correlation between hirsutism and the visceral adiposity index (VAI) suggests that hair growth disorders are not directly driven by visceral fat dysfunction [67].

For women with obesity or metabolic syndrome, a ketogenic or low-GI diet may produce greater metabolic and hormonal benefits than standard dietary interventions. Overall, adherence to a nutrient-balanced, calorie-controlled eating plan remains key for restoring physiological equilibrium and promoting recovery in PCOS.

2.1.1 Physical Activity

Exercise training in the management of PCOS is becoming more recognised and accepted among professionals in the health sector and patients. Physical training potentiates the effects caused by insulin sensitivity through the optimisation of glucose transport and metabolism [68].

Recent evidence indicates that the intensity of physical activity has a stronger impact on health outcomes than total exercise volume. Vigorous-intensity activity appears to produce the largest improvements in cardiorespiratory capacity, insulin resistance, and body composition among women with PCOS [69]. Significant reductions in insulin resistance, measured by HOMA-IR, and in BMI have been observed following moderate- and high-intensity programs (MD −0.57; 95% CI −0.98 to −0.16, $p = 0.01$; and MD −1.90; 95% CI −3.37 to −0.42, $p = 0.01$, respectively) [70]. Systematic reviews recommend incorporating both aerobic and resistance exercise to optimise insulin sensitivity and androgen balance in this population [71]. A minimum of approximately 120 minutes of aerobic activity per week is generally advised [69].

2.1.2. Sleep

Psychological disturbances such as anxiety, depression, and sleep abnormalities occur more frequently in women with PCOS [72]. Sleep dysregulation contributes to both the onset and progression of metabolic and emotional disturbances; therefore, treating sleep issues is a critical component of PCOS management [72]. Insufficient or fragmented sleep is linked to a higher risk of insulin resistance, obesity, and type 2 diabetes [73–75]. The mechanisms appear to involve autonomic dysregulation, hormonal fluctuations in leptin and ghrelin, and inflammatory signalling. Experimental models show that chronic sleep fragmentation leads to inflammation in white adipose tissue and aggravates insulin resistance, partly through disruption of intestinal barrier function and lipopolysaccharide-mediated inflammation (“gut leakage”) [51,76]. These findings suggest that microbiota-targeted therapy may mitigate some of the metabolic effects of poor sleep [76].

Melatonin, the main hormone secreted by the pineal gland, regulates circadian rhythm and exhibits strong antioxidant properties. In PCOS, reduced melatonin concentrations have been reported in follicular fluid [77]. Melatonin receptors within ovarian tissue influence steroidogenesis during follicular maturation, and sufficient melatonin levels help protect developing oocytes from oxidative damage [77]. Overall, sleep disorders may represent an early contributor to diminished physiological resilience and worsening insulin resistance in PCOS.

2.1.3. Supplementation

Dietary surveys reveal that many women with PCOS consume unbalanced diets lacking fibre, omega-3 fatty acids, and key micronutrients such as calcium, magnesium, zinc, folate,

vitamins C, B12, and D, while showing excessive intake of sucrose, sodium, saturated fat, and cholesterol [8]. Calorie-controlled, low-glycemic-index diets can correct some deficiencies, especially of water-soluble vitamins [78,79]. Improved plasma concentrations of most B vitamins have been noted after dietary adjustment, though vitamin B3 responses remain suboptimal [79]. Inadequate niacin (B3) intake has been associated with inflammation and higher cardiovascular risk [80,81].

Metformin therapy, while beneficial for glycemic control, may reduce thiamine and cobalamin stores, warranting supplementation [82]. Thiamine enhances transketolase activity and supports vascular protection, potentially reducing cardiovascular complications [83,84]. Coenzyme Q10 (CoQ10) supplementation for eight weeks has been shown to improve inflammatory status and endothelial function in overweight and obese women with PCOS [85]. Vitamin D plays multiple metabolic roles, enhancing insulin synthesis, receptor expression, and response to glucose [86]. It indirectly modulates carbohydrate metabolism by maintaining calcium–parathyroid hormone balance and by suppressing pro-inflammatory cytokine expression [87]. Weekly supplementation with 20,000 IU cholecalciferol improved fasting glucose, triglycerides, estradiol levels, and menstrual regularity, although androgen levels remained unchanged [88].

Combined supplementation with magnesium, zinc, calcium, and vitamin D produced reductions in hirsutism and total testosterone but did not affect SHBG or the free androgen index [89]. Similarly, vitamin D administered with fish oil reduced serum C-reactive protein, downregulated interleukin-1 expression, lowered testosterone levels, and improved mood scores in women with PCOS [90]. Overall, nutritional optimisation, targeted vitamin and mineral replacement, and antioxidant support represent important adjuncts to lifestyle and pharmacologic therapy in PCOS.

Current evidence indicates that myo-inositol provides metabolic and hormonal improvements in women with PCOS comparable to those achieved with metformin, particularly regarding insulin sensitivity and glucose regulation, but without the gastrointestinal side effects often associated with metformin therapy [91,92]. Supplementation with inositol enhances tissue responsiveness to insulin, lowers circulating androgen concentrations, improves glycemic control, and positively affects several markers of metabolic syndrome [93,94]. In PCOS, an excessive conversion of myo-inositol (MI) to D-chiro-inositol (DCI) has been observed in the ovaries under the influence of insulin, leading to a local shortage of MI and an excess of DCI. This imbalance may impair follicle-stimulating hormone (FSH) signalling and compromise oocyte maturation and quality [95]. Clinical studies suggest that using inositol isomers—either individually or in combination—can restore spontaneous ovulation, support folliculogenesis, and improve conception rates in women with PCOS. Literature reviews consistently identify inositol supplementation as a safe and effective therapeutic option that enhances ovarian function, oocyte development, and pregnancy outcomes [96].

Traditional and complementary medicine approaches have also explored the use of natural compounds such as isoquinoline alkaloids to regulate androgen synthesis and lipid and carbohydrate metabolism. Berberine, one such alkaloid, has attracted attention for its multiple beneficial effects in PCOS management [97–99]. Its metabolic action resembles that of metformin, largely through activation of adenosine monophosphate-activated protein kinase (AMPK), leading to improvements in glucose and lipid profiles, reduction in body mass, and increased insulin sensitivity [100]. Berberine additionally influences the hypothalamic–pituitary–ovarian axis by reducing the synthesis of steroid hormones and downregulating ovarian aromatase, which contributes to improved ovulatory function, menstrual regularity, and higher pregnancy and live-birth rates. Long-term use has been associated mainly with mild and transient adverse effects such as nausea or constipation, indicating a favourable safety profile [98,101,102].

Chromium, an element involved in carbohydrate and lipid metabolism, has long been included in dietary supplements marketed for metabolic health in the United States [103]. Although the essentiality of chromium remains debated, some findings suggest it can enhance insulin signalling, promote AMPK activity, and increase cellular glucose uptake, thereby benefiting patients with PCOS and type 2 diabetes [104–106]. Furthermore, alterations in the expression of steroidogenic enzymes—specifically 3β-hydroxysteroid dehydrogenase and 17β-hydroxysteroid dehydrogenase—in adipose tissue have been linked to dehydroepiandrosterone metabolism, suggesting that chromium may indirectly affect androgen balance [107].

Evidence from clinical and experimental research indicates that supplementation with trace minerals such as zinc and selenium may be beneficial for some women with PCOS. Zinc is involved in numerous intracellular processes, serving both structural and signalling functions that influence glucose and lipid metabolism as well as reproductive health [108]. Insufficient zinc intake, particularly among individuals with obesity, has been linked to hyperinsulinemia, chronic low-grade inflammation, and an adverse lipid profile. In adipose tissue, zinc ions can act similarly to insulin by promoting glucose uptake via translocation of the glucose transporter GLUT4 to the plasma membrane and by stimulating lipogenesis [109]. Several studies report that women with PCOS have lower serum zinc concentrations than healthy controls, and those with impaired glucose tolerance exhibit the lowest levels [110]. Selenium, another essential micronutrient, demonstrates potent anti-inflammatory and antioxidant actions and shows an inverse correlation with circulating C-reactive protein (CRP) levels [111]. Maintaining adequate selenium and zinc status may therefore support metabolic equilibrium and reduce the inflammatory burden characteristic of PCOS.

Omega-3 fatty acids are also frequently deficient in the diets of women with PCOS. When an overall balanced diet is followed, targeted omega-3 supplementation may be required only seasonally or during periods of dietary imbalance [112]. Polyunsaturated fatty acids (PUFAs) contribute to improved ovarian function by enhancing the expression of steroidogenic

enzymes—such as CYP51, CYP19, StAR, and 3β-HSD—which regulate hormone synthesis and reproductive performance [113]. Supplementation should always be individualised and monitored by a qualified dietitian to ensure patient adherence and safety. Active participation by the patient remains crucial for achieving a lasting improvement in metabolic homeostasis. A nutritionally adequate diet, combined with regular exercise and stress control, continues to represent the cornerstone of PCOS therapy.

2.2. Herbs Supporting Treatment

A nutritionally balanced plan that stabilises insulin response can be effectively complemented by selected medicinal herbs. Botanical preparations such as Aloe vera, cinnamon (Cinnamomum verum), green tea (Camellia sinensis), chamomile (Matricaria chamomilla), and white mulberry (Morus alba) have demonstrated beneficial effects on glucose and lipid metabolism and may also exert mild anti-inflammatory activity [114,115]. Because of these properties, such herbal agents can be applied to different PCOS phenotypes, especially where metabolic disturbances dominate. Green tea [116] and marjoram (Majorana hortensis) have been shown to improve insulin sensitivity, restore hormonal balance, and enhance antioxidant and anti-inflammatory parameters in both clinical and preclinical studies [117,118].

For women with elevated androgen levels, specific antiandrogenic herbs may be helpful. Spearmint (Mentha spicata L.) reduces circulating testosterone concentrations and supports follicular development in ovarian tissue [119,120]. Liquorice (Glycyrrhiza glabra), long used in traditional medicine, contains phytoactive molecules that exert both estrogen-like and antiandrogenic actions. Glycyrrhetinic acid, one of its principal metabolites, inhibits 11β-hydroxysteroid dehydrogenase type 2 and binds to mineralocorticoid receptors, thereby influencing steroid metabolism [121,122]. Despite its therapeutic potential, liquorice can increase blood pressure and alter potassium balance; individuals with high cortisol levels or cardiovascular disease should therefore use it cautiously [123]. Other botanical extracts possess 5-α-reductase inhibitory activity and may counteract androgen-driven hair loss. Serenoa repens, Camellia sinensis, Rosmarinus officinalis, and Glycyrrhiza glabra have demonstrated the capacity to reduce androgen concentrations and slow the progression of androgenetic alopecia [124].

Vitex agnus-castus remains one of the best-studied herbal agents for restoring menstrual cyclicity and alleviating premenstrual symptoms through dopaminergic and pituitary modulation [125]. Phytoestrogen-rich sources such as flaxseed (Linum usitatissimum), abundant in lignans, may regulate aromatase activity and influence estrogen metabolism, thereby helping to normalise sex hormone ratios [126,127]. Turmeric (Curcuma longa) and its main polyphenolic constituent, curcumin, have well-documented antioxidant and anti-inflammatory effects. In PCOS, curcumin supplementation has been associated with reductions in oxidative stress markers and inhibition of NF-κB–dependent inflammatory signalling [128–132]. Urtica dioica (nettle) displays wide-ranging pharmacological activities, including antioxidant, antimutagenic, and anti-inflammatory effects that may aid metabolic regulation [133,134]. Plant-derived flavonoids, present in many of these herbs, can neutralise reactive oxygen species such as peroxides and hydroxyl radicals, protecting tissues from oxidative damage and supporting cellular resilience [135]. In more advanced PCOS accompanied by metabolic syndrome or non-alcoholic fatty liver disease, hepatoprotective plants can be useful adjuncts. Extracts from milk thistle (Silybum marianum), which contain silymarin, have shown antioxidant and membrane-stabilising effects that protect hepatocytes from oxidative injury [136–138]. Artichoke (Cynara cardunculus) provides sesquiterpene lactones and phenolic acids with similar hepatoprotective properties [139,140]. Compounds from dandelion (Taraxacum officinale), particularly taraxasterol, activate SIRT1-dependent pathways that safeguard liver cells [141]. Nigella sativa (black cumin) also exhibits antioxidant and anti-inflammatory actions, and may reduce hepatic steatosis and insulin resistance in obese women with PCOS [142].

In summary, herbal and micronutrient supplementation offer numerous complementary strategies for PCOS management. The combined actions of antioxidant, antiandrogenic, and hepatoprotective mechanisms contribute to restoring metabolic and hormonal equilibrium. Individualised selection of herbal mixtures and nutritional support—guided by clinical assessment and evidence-based practice—can enhance the efficacy of standard medical therapies and improve the overall health outcomes of women living with PCOS. Summary information has been added in Table 1.

Table 1: Overview of herbal interventions and their reported outcomes in PCOS

A Symptom Accompanying PCOS	Diet	Physical Activity	Sleep	Supplementation	Microbiota	Herbs
Hirsutism	Reduced Diet [26,44,45,54,58]	Daily Physical Activity [68–71]	Improving Sleep [72–77]	Magnesium, Zinc, Calcium [89,108–111], Vitamin D [86–90], Myo-Inositol [93–96]	Microbiota And Metabolites [46,47]	Gl Glycyrrhiza Glabra, Serenoa Repens, Camellia Sinensis [120,121], Rosmarinus Officinalis [122]
The androgen levels	Diet With Reduced GI And Calorie [26,44,45,54,58], Ketogenic Diet [64]	Daily Physical Activity [68–71]	Improving Sleep [72–77]	Magnesium, Zinc, Calcium [89,108–111], Vitamin D [86–90], Berberine [97–102], Chromium [105–107], Zinc [110]	SCFA [47,52], Microbiota and Metabolites [50]	Mentha Spicata [120,121], Glycyrrhiza Glabra [122], Serenoa Repens, Camellia Sinensis, Rosmarinus Officinalis [124]
Ovulation disorders	Diet With Reduced GI And Calorie [26,44,45,54,58], Ketogenic Diet [64]	Daily Physical Activity [71–74]	Improving Sleep [72–77]	Vitamin D [86–90], Myo-Inositol [97,98], Berberine [99], Zinc [108], Pufas [112,113]	Bifidobacteria [45,50]	Glycyrrhiza Glabra [121], Vitex Agnus-Castus [124], Flaxseed (Linum Usitatissimum) [59,125,126]
Fat mass reduction	High-Fibre Diet with Reduced GI And Calorie [28,46,47,56,60], Ketogenic Diet [64], Elimination Of SFA [22,58]	Daily Physical Activity [68–71]	Melatonin [77]	Vitamin B1 [82–84], Vitamin D [86–90], Myo-Inositol [91–96], Berberine [97–102], Chromium [105–107], Zinc [109]	SCFA [47,52]; Microbiota and Metabolites [50]	Aloe Vera, Cinnamomum Verum, Camellia Sinensis [115], Matricaria Chamomilla, Morus Alba [117]
Carbohydrate metabolism disorders	High-Fibre Diet With Reduced GI And Calorie [26,44,45,54,58], Ketogenic Diet [64]	—	—	Vitamin D [86–90], Myo-Inositol [91–96], Berberine [97–102]	—	Aloe Vera, Cinnamon, Green Tea [115], Chamomile, White Mulberry [117]
Insulin resistance	High-Fibre Diet With Reduced GI And Calorie [26,44,45,54,58], Elimination SFA [22,58]	Intensity Exercise [72]	—	Omega-3 [112,113], Berberine [97–102], Zinc [110]	—	Milk Thistle [137,138], Artichoke Extract [139,140], Dandelion [141], Black Cumin [142]
Lipids metabolism disorders	High-Fibre Diet With Reduced GI And Calorie [26,44,45,54,58], Elimination Of SFA [25,60]	—	—	—	Bifidobacteria [45,50]	Milk Thistle [137,138], Artichoke Extract [139,140], Dandelion [141], Black Cumin [142]
Steatosis of organs – liver profile	High-Fibre Diet With Reduced GI And Calorie [46,47,56,60]	—	—	A-Linolenic Acid [59], Vitamin B3 [80,81], Vitamin B1 [82–84], Coenzyme Q10 [85]	—	—
Cardiovascular diseases	High-Fibre Diet With Reduced GI And Calorie [46,47,56,60]	—	—	—	—	—
Intestinal dysbiosis	High-Fibre Diet [49,50]	—	—	—	—	—
A Symptom Accompanying PCOS	Diet	Physical Activity	Sleep	Supplementation	Microbiota	Herbs
Chronic inflammation	High-Fibre Diet With Reduced GI And Calorie [28,46,47,56,60]	Daily Physical Activity [71–74]	Melatonin [79]	A-Linolenic Acid [59], Vitamin B3 [80,81], Coenzyme Q10 [85], Vitamin D [88,89], Selenium [112], Flavonoids [135]	Bifidobacteria [45,50]	Marjoram [117–119], Turmeric [128–131], Nettle [133,134], Milk Thistle [137,138], Artichoke Extract [139,140], Dandelion [141], Black Cumin [142]
Limiting predisposition to cancer	Elimination SFA [25,27]; High-Fiber Diet [49,50]	—	Improving Sleep [75]	A-Linolenic Acid [59]	—	Turmeric [128–131], Nettle [133,134]

SCFA—short-chain fatty acids;
GI—glycemic index;
SFA—saturated fat acids;
PUFA—Polyunsaturated fatty acid.

3. CONCLUSIONS

Metabolic dysregulation associated with polycystic ovary syndrome (PCOS) requires a multifaceted therapeutic approach that simultaneously addresses hormonal, reproductive, and metabolic disturbances. These disturbances stem from several interacting biochemical pathways; therefore, clinical management should emphasize improving fertility outcomes, minimizing androgen-related symptoms, and restoring glucose–lipid metabolism and insulin response. Lifestyle modification remains central to therapy, with evidence supporting the benefits of a calorie-controlled, low-glycemic-index diet, adequate sleep, and consistent physical activity in reducing cardiometabolic risk and enhancing overall health in affected women.

Adjunct nutritional and phytotherapeutic strategies have gained attention as supportive interventions in PCOS. Bioactive compounds with antioxidant and hepatoprotective properties may contribute to reduced inflammation and improved metabolic status. Specific agents—such as extracts from Curcuma longa or Silybum marianum and probiotics aimed at restoring gut microflora—have shown preliminary benefits in clinical and experimental contexts. In the present study, nutrient-intake patterns and supplementation practices were evaluated to understand their influence on micronutrient balance among women with PCOS. Emerging evidence also highlights potential roles for inositols, thiamine, coenzyme Q10, vitamin D, zinc, and selenium in optimizing metabolic and reproductive outcomes.

Although these findings indicate encouraging trends, definitive conclusions cannot yet be drawn. Larger, well-designed trials are needed to validate the efficacy, safety, and long-term clinical significance of these nutritional and herbal interventions when combined with standard medical care..

4. Methods of Searching

This review focused on non-pharmacological approaches to the management of PCOS. A comprehensive literature search was carried out in the PubMed and Embase (Elsevier) databases, covering research published within the past twenty years.All retrieved records were screened at the abstract level. Publications unrelated to the primary topic, duplicated between the two databases, or limited to conference proceedings were excluded. Only articles written in English were included for full-text review.The discussion also draws on the authors' decade of clinical and research experience with PCOS patients. From this work, studies corresponding to each stage of the interventions discussed were selected. The literature search emphasized physiological mechanisms linking PCOS with insulin resistance, chronic inflammation, endocrine dysfunction, cancer development, and gut microbiota alterations. Lifestyle modification studies were analyzed first, followed by those investigating diet and supplementation—including inositol, berberine, vitamin D, chromium, zinc, selenium, and melatonin—along with reports evaluating herbal adjunct therapies. In cases of overlapping information across publications, the most comprehensive and relevant studies were prioritised for inclusion.

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