Original Research Paper

Volume - 15 | Issue - 09 | September - 2025 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Medical Science

THE IMPACT OF DIET ON POLYCYSTIC OVARY SYNDROME: A COMPREHENSIVE REVIEW OF THERAPEUTIC MECHANISMS AND CLINICAL APPLICATIONS

Prisha Sachin Patel Navrachana International School Vadodara.

ABSTRACT Polycystic ovary syndrome (PCOS) affects 4-20% of women globally and represents a complex endocrine disorder characterized by insulin resistance, hyperandrogenism, and metabolic dysfunction. Dietary approaches have emerged as fundamental therapeutic modalities with potential to address PCOS pathophysiology through various biological mechanisms. This comprehensive narrative review examines the impact of dietary interventions on PCOS management, synthesizing current evidence on mechanisms of action, clinical efficacy, and implementation considerations. A literature search was conducted across PubMed/MEDLINE, Scopus, Web of Science, Cochrane Library, and Google Scholar from 1990-2025. Studies involving women with PCOS and dietary interventions were included, including randomized controlled trials, observational studies, systematic reviews, and mechanistic research. Data synthesis used a thematic approach organizing evidence around mechanistic pathways, dietary approaches, and clinical outcomes. Evidence shows that dietary interventions significantly improve PCOS outcomes through gut microbiota modulation, insulin-glucose regulation, anti-inflammatory effects, and hormonal optimization. Low-glycemic index diets show 15-25% improvements in insulin sensitivity and 20-30% testosterone reductions. Mediterranean diet patterns provide anti-inflammatory benefits and cardiovascular protection. Ketogenic interventions show rapid metabolic improvements with 87% reduced PCOS risk with high-quality dietary patterns. Omega-3 fatty acids, polyphenols, fiber, and micronutrients address distinct pathophysiological mechanisms. Implementation challenges include poor adherence, cultural barriers, and limited provider education. Dietary interventions represent evidence-based therapeutic modalities for PCOS management with demonstrable metabolic, reproductive, and psychological benefits. Integrating precise nutrition approaches considering individual biological profiles represents the future of PCOS care. Healthcare providers should prioritize nutritional counseling as first-line therapy, while future research should focus on personalized strategies and digital health integration to optimize outcomes globally.

KEYWORDS : Polycystic ovary syndrome (PCOS), Dietary interventions, Insulin resistance, Gut microbiota, Anti-inflammatory nutrition

INTRODUCTION

Polycystic ovary syndrome (PCOS) is one of the most common hormone disorders affecting women of childbearing age worldwide. About 4-20% of women globally have PCOS, with even higher rates in some countries like India (8.2-22.5%) and developed nations (15-20%). This condition affects millions of women and costs healthcare systems billions of dollars each year.

PCOS causes problems with a woman's hormones, metabolism, and ability to have children. Women with PCOS often struggle with irregular periods, weight gain, insulin resistance (when the body doesn't respond well to insulin), and higher levels of male hormones like testosterone. These issues can make it difficult to get pregnant and can lead to other health problems like diabetes and heart disease.

Recently, scientists have discovered that diet plays a major role in managing PCOS symptoms. Researchers have conducted over 2,000 studies examining how different foods and eating patterns can help women with PCOS feel better and improve their health. The market for PCOS treatments, including nutritional approaches, is expected to grow to $3.9 billion by 2030, showing how important nutrition has become in treating this condition.

Many studies have shown that changing diet can significantly help with PCOS symptoms. The right diet can improve insulin sensitivity (how well the body uses insulin), reduce male hormone levels, help restore normal periods, and improve overall metabolic health. Different types of diets have proven helpful, including low-glycemic index diets, Mediterranean diets, ketogenic diets, and anti-inflammatory diets.

The understanding of how diet affects PCOS has evolved greatly. Instead of just focusing on eating fewer calories, scientists now understand that specific nutrients and eating patterns can target the root causes of PCOS. This includes examining how diet affects gut bacteria, inflammation, and hormone levels.

While many individual studies have examined specific nutrients like omega-3 fatty acids, fiber supplements, and probiotics, few studies have looked at how all these dietary factors work together. Most research focuses on single nutrients or one type of diet, leaving gaps in our understanding of how different foods and nutrients might work together to improve PCOS.

New technologies are helping doctors create personalized nutrition plans based on a person's individual genetics, gut bacteria, and metabolism. This means that doctors can now recommend specific foods and eating patterns that work best for each person's unique situation.

Dietary treatments for PCOS are especially valuable because they are often less expensive and more accessible than medications. This is particularly important in areas where expensive drugs might not be available. Understanding how diet affects different aspects of PCOS is crucial for healthcare providers, researchers, and policymakers to develop effective treatment plans.

This research aims to answer three main questions:
RQ1: How does diet influence PCOS through gut bacteria, hormone regulation, and metabolism?
RQ2: How do different dietary approaches (like low-glycemic, Mediterranean, ketogenic, and anti-inflammatory diets) specifically help with various aspects of PCOS?
RQ3: What challenges do doctors and patients face when trying to implement dietary treatments for PCOS?

This paper will explore these questions by examining how PCOS affects the body, how diet can help, and what practical challenges exist in using food as medicine for PCOS management.

Research Methodology

This research used the narrative literature review method for examining the impact of dietetic interventions on polycystic ovary syndrome (PCOS) management.

Systematic literature searching was conducted in a number of electronic databases for research papers published from January 1990 through February 2025. The databases included PubMed/, Scopus, Web of Science, and Google Scholar. The search strategy used the following keywords in three concept clusters: PCOS-related (e.g., "polycystic ovary syndrome," "PCOS," "hyperandrogenism," "oligomenorrhea"), dietary intervention (e.g., "diet," "nutrition," "Mediterranean diet," "ketogenic diet," "low glycemic index," "omega-3 fatty acids," "polyphenols," "probiotics"), and mechanism/outcome (e.g., "insulin resistance," "gut microbiota," "inflammation," "reproductive outcomes") keywords.

Original research papers, systematic reviews, meta-analyses, clinical trials, and observational studies published in English-language, peer-reviewed journals were included. Excluded were studies that discussed other endocrine diseases only, pharmaceutical treatment only, conference abstracts, editorials, and non-peer-reviewed publications. A total of 60 research papers was used for the narrative review.

A standardized data extraction form used study features, population characteristics, intervention details, outcome variables, and mechanistic data.

Because of heterogeneity of interventions and study designs, narrative synthesis was employed rather than pure meta-analysis. The evidence was synthesized across key thematic issues: mechanistic mechanisms (hormonal regulation, gut microbiota, inflammation), dietary approaches (low-glycemic index, anti-inflammatory, Mediterranean, ketogenic), individual nutrients (micronutrients, fiber, polyphenols, omega-3 fatty acids), and clinical outcomes (metabolic, psychological, and reproductive parameters).

Polycystic Ovary Syndrome

Polycystic ovary syndrome (PCOS) is a major endocrine and metabolic disorder in reproductive-aged women with a cluster of reproductive, metabolic, and cosmetic manifestations (Khobragade et al., 2024). According to the Rotterdam criteria, PCOS is diagnosed when two or more of the following three criteria coexist: (1) hyperandrogenism (elevated male sex hormones levels), (2) oligomenorrhea or amenorrhea (irregular or absent menstruation), and (3) polycystic ovarian morphology, ≥12 follicles in each ovary with a diameter of 2-9 mm (Krug et al., 2019; Lizneva et al., 2016).

This polyfacetic endocrine disorder is associated with hypothalamic-pituitary-ovarian axis dysfunction, disrupted ovarian morphology, altered adrenal steroid hormone secretion, metabolic syndrome elements, and genetic predisposition (Khobragade et al., 2024). The syndrome results in fulminant long-term complications in the form of type 2 diabetes mellitus, premature atherosclerosis, infertility, and endometrial cancer and is therefore still a significant public health problem (Khobragade et al., 2024).

Hyperandrogenism becomes evident with a wide range of adverse effects such as hirsutism (excessive hair growth in the face and body), acne, androgenic alopecia (male pattern baldness at the mid-scalp area), and cyst formation in the ovary (Krug et al., 2019; Norman et al., 2004; Teede et al., 2010).

Reproductive failure is a characteristic finding, including oligomenorrhea (less than eight menstrual cycles during one year) or amenorrhea and oligoovulation or anovulation in females (Krug et al., 2019; Yildiz et al., 2012). These defects of reproduction severely compromise fertility along with the psychological morbidity of the condition.

Metabolically, patients with PCOS present with a variety of features such as insulin resistance, glucose intolerance, type 2 diabetes, and cardiovascular risk factors and are much more likely to be overweight or obese (Krug et al., 2019; Lim et al., 2018; Moran et al., 2010). The syndrome is also accompanied by a higher prevalence of eating disorders, depression, and anxiety, rendering it more difficult to manage clinically (Krug et al., 2019).

PCOS is estimated to affect 4-20% of women of reproductive age globally but with very wide ranges of prevalence rates depending on the diagnostic criteria used and the populations studied (Khobragade et al., 2024).

In India, prevalence is speculated to be 8.2% to 22.5% based on data up to December 2019 (Desai & Patel, 2023). Surprisingly, epidemiological investigations propose that PCOS is a more common condition among urban Indian women than rural women, and it may be related to lifestyle factors such as food habit, activity, and environmental exposure (Khobragade et al., 2024).

PCOS pathogenesis is multifactorial and complex with interacting genetic, hormonal, metabolic, and environmental factors. Multiple etiologic hypotheses have been advanced to explain PCOS pathogenesis, including hypothalamic-pituitary-adrenal axis dysfunction, ovarian morphology and steroidogenesis alteration, components of metabolic syndrome, and hereditary predisposition (Khobragade et al., 2024; Nisa et al., 2023; Wei et al., 2022).

Neuroendocrine dysfunction is at the center of pathophysiology for PCOS. The hypothalamic-pituitary-adrenal (HPA) axis, an intricate network regulating interactions between the hypothalamus, pituitary gland, and adrenal glands, is altered in PCOS patients (Khobragade et al., 2024). Dysfunction causes inappropriate secretion of gonadotropin hormone, resulting in luteinizing hormone (LH) and follicle-stimulating hormone (FSH) dysregulation in the proliferative phase of the menstrual cycle (Baskind & Balen, 2016; Stener-Victorin et al., 2020).

The increased LH/FSH ratio, secondary to heightened pituitary sensitivity to gonadotropin-releasing hormone (GnRH), interferes with normal ovarian function (Khobragade et al., 2024). Estradiol and progesterone, the major ovarian steroid hormones, under normal conditions, exert negative feedback to regulate GnRH pulsatile secretion patterns. This compensatory feedback is abnormal in PCOS, leading to persistent anovulation and irregular ovarian steroidogenesis (Khobragade et al., 2024).

Insulin resistance syndrome is another key element of PCOS etiology, forming a vicious circle that facilitates the continuation of the metabolic and reproductive features of the illness. Genetic influences are also significant in PCOS susceptibility, and family aggregation and heritability studies provide evidence to the presence of a powerful genetic factor to syndrome emergence.

Impact of Diet on Health: Mechanisms of Food-Health Interactions

The dietary relationship to human health is much more intricate than simple nutritional needs, involving intricate interactions among components of foods, host physiology, and microbial flora. Diet has been shown in recent studies to impact health through a number of interconnected mechanisms involving modulation of gut microbiota composition, regulation of the immune system, alteration of metabolic pathways, and epigenetic modification.

Gut Microbiota

The human gut microbiota is a dynamic community of trillions of microorganisms of basic functions in human physiology and health homeostasis. The gut microbiota is estimated to be a 3 × 10^13 population of microorganisms that consist of bacteria, viruses, fungi, protozoa, and archaea, and harbor about 3.3 million non-redundant microbial genes compared to the human genome's 21,000 genes—a ratio of about 150:1 (Kelley et al., 2016; Khobragade et al., 2024).

Immune System Modulation

The gut bacteria are key components of numerous physiological processes and hold key roles in the pathological formation of autoimmune and chronic immune disease, with direct disease mechanisms that become more evident by the day (Zhou et al., 2024). The gut flora is accountable for the development and maturation of the immune system of the intestinal mucosa, forming a complex defense system in addition to local immunity.

When exposed to invasion by pathogens, gut flora triggers humoral immunity along the intestinal mucosa and immediately sets up immune tolerance (Furusawa et al., 2013). Gut microbiota achieves systemic immune homeostasis through a variety of mechanisms: induction of proper inflammation, controlling T lymphocyte differentiation, immunoglobulin encoding, promoting B cell maturation, and activating antibody-secreting B cell populations (Zhou et al., 2024).

The control of systemic immune responses by gut microbiota through modulation of chronic inflammation pathways has relevance to a broad range of diseases with an immunological component.

Microbial Diversity and Individual Variation

Human gut microbiomes may differ by 80–90% between individuals, even though human beings have 99.9% of their genomic sequences in common. This high degree of diversity impacts responses to treatments, disease susceptibility, and general health outcomes (Tomova et al., 2019). Many things, including genetics, early life exposures, antibiotic use, lifestyle, and most importantly-dietary habits-contribute to this variation in the composition of gut microbiota in individuals.

Several health conditions, such as neurological disease, metabolic disease, autoimmune disease, cardiovascular disease, and more, have been associated with disruption in gut microbial populations

(Khobragade et al., 2024). Such broad impact underscores the gut microbiota's central role as a mediator between nutrition and disease endpoints in several organ systems.

Gut-Brain Axis: Bidirectional Communication Networks

Neural, hormonal, and immunologic mechanisms are implicated through which the gut-brain axis is a dynamic two-way communication process that connects the central nervous system and the gastrointestinal system. The axis provides for continuous communication between the brain and the gut, which affects a wide range of physiological processes such as mood, appetite, stress response, and cognition.

The production of neurotransmitters and neuroactive compounds by intestinal microbiota is responsible for this interaction. Mood, behavior, and neurological function are affected by neurotransmitters like serotonin, gamma-aminobutyric acid (GABA), dopamine, and acetylcholine, produced by certain bacterial species. Besides, metabolites from microbiota, most notably short-chain fatty acids (SCFAs), have the ability to cross the blood-brain barrier and modulate neurotransmitter production and neuroinflammation directly.

Stress Response And Hormonal Regulation

In addition to the HPA axis, the gut-brain axis also facilitates stress responses through the hypothalamus. Although microbial dysbiosis can modulate the production of stress hormones and stress sensitivity, chronic stress can alter the gut microbiota composition. Such bidirectional interaction demonstrates how interventions that support gut microbiota health may improve mental health outcomes and stress resilience.

Nutrient Absorption And Metabolic Integration

By a range of mechanisms, the gut microbiota significantly contributes to metabolic events and nutrient utilization. The bioavailability of essential nutrients, including vitamin K, B vitamins (particularly B12, folate, and biotin), and various minerals, is influenced by particular species of bacteria. While certain bacteria are capable of competing for nutrients or altering their chemical structure, others are able to produce these vitamins de novo, enhancing the host's nutritional status.

In order to hydrolyse intricate dietary constituents that human enzymes cannot digest, microbial enzymes come into play. For example, the bacterial enzyme is responsible for degrading dietary fibre into SCFAs (acetate, propionate, and butyrate), which colonocytes utilize as energy substrates and also as signalling molecules influencing immunological response, lipid metabolism, and glucose homeostasis.

Metabolic Pathway Modulation

Gut microbiota influence host metabolism by modulating the metabolic pathway through multiple mechanisms, including the control of lipid metabolism, amino acid biosynthesis, and bile acid metabolism. These microbial effects may contribute to the development of metabolic disorders, metabolic efficiency, and energy extraction from food. Microbial production of metabolites creates a rich biochemical signaling network that integrates host metabolic responses and nutrient inputs.

Bioactive Compounds: Polyphenols and Plant Secondary Metabolites

Polyphenols are highly accumulated in plant tissue, a broad class of secondary metabolites produced by the acetic acid and shikimic acid pathways (Zhou et al., 2024). Flavonoids and non-flavonoids are the two generic types of polyphenols in plants that can be distinguished based on their chemical structures. They exist in two major forms: free aglycones lacking sugar moiety or bound to sugars to form well-soluble glycosides (Santhakumar et al., 2018). Flavonoids are a crucial class of plant polyphenols that are widely distributed in fruits, vegetables, tea, legumes, and other edible plant foods, as noted by Zhou et al. (2024). This class has several subgroups including isoflavones, flavonols, flavanones, flavanols, and anthocyanidins. Because of the variability in their chemical structures, every one of these subgroups possesses unique physiological and biochemical activities, like anti-inflammatory, antioxidant, and antimicrobial effects (Wang et al., 2022; Xia et al., 2022).

Another crucial class is the non-flavonoid polyphenols, which consist of phenolic acids (e.g., caffeic acid, ferulic acid, and chlorogenic acid), tannins, stilbenes, and lignans (Zhou et al., 2024). Through anti-inflammatory, antioxidant, and metabolically health-promoting mechanisms, these substances foster health (Banez et al., 2020; Barre & Mizier-Barre, 2019).

Furthermore, polyphenols act directly on the gut microbiota and are prebiotic substances that inhibit pathogenic microbes and stimulate growth in beneficial bacteria. Non-flavonoid polyphenols play a critical role in ensuring total intestinal health, protecting against damage to the intestinal mucosa, and maximizing the structure of intestinal flora (Li et al., 2023; Molino et al., 2021; Zhang et al., 2021). Gut microbiota metabolism plays a significant role in determining the bioavailability of polyphenols. Intestinal bacteria biotransform many polyphenols, producing metabolites that are more bioavailable and biologically active than the parent compound. For optimal realization of the health benefits of dietary polyphenols, this microbial processing is required.

A dynamic regulative network is created by the reciprocal interaction between polyphenols and gut microbiota, where the former alter microbial form and the latter are metabolized by microbes to produce bioactive metabolites (Zhou et al., 2024).

Dietary Intervention as a Therapeutic Tool

Modulation of Gut Microbiota by Diet

Of several therapeutic methods, nutritional therapy has been held as the most powerful and easily modifiable method for influencing gut flora composition and function (Wan et al., 2021). Food-derived bioactive compounds, especially polyphenols, are ideal potential targets for gut microbiota modulation. They are natural regulators of inflammation and oxidative stress, thus ideal tools for prevention and treatment of chronic ailments (Tzounis et al., 2008).

The evidence from a variety of research models, including in vitro experiments, in vivo experiments, and clinical trials, consistently validates the health effects of polyphenols and other bioactive food constituents (Durazzo et al., 2019; Fraga et al., 2019). Such an evidence base supports the development of evidence-based dietary interventions for promoting health and preventing disease.

The striking heterogeneity in gut microbiota between individuals suggests that personalized nutrition approaches may be necessary to realize optimal health outcomes. The discovery of individual microbial signatures, genetic determinants, and metabolic phenotypes may enable the design of individualized dietary interventions that take into account individual variation in nutrient metabolism, microbial ecology, and responsiveness to therapy.

Geographical and cultural dietary practices also affect gut microbiota composition, so effective dietary interventions would potentially have to be customized to geographic and cultural environments.

Integration of Mechanisms: Systems-Level Effects

Interconnected Pathways

Instead of being distinct pathways through which diet influences health, the different ways in which diet influences health are part of an integrated network of interactions. The character of the gut microbiota determines nutrient uptake, and nutrient uptake determines metabolic pathways, and metabolic pathways determine inflammation and immune status. Similarly, stress reactions determine composition and barrier integrity of the gut microbiota, which determine signalling along the gut-brain axis.

There are diverse pathways that are concurrently acted upon by polyphenols and other bioactive compounds: they modify metabolic signalling, protect against oxidative stress, modulate inflammation, and alter gut microbiota.

Temporal Dynamics

Dietary impacts on health include acute and chronic impacts. Acute dietary changes can modify gut microbiota composition quickly over the span of days, whereas chronic patterns of intake establish more persistent microbial communities and metabolic adaptation.

Dietary Impact on PCOS: Food Groups and Their Biochemical Mechanisms

The relationship between food intake and polycystic ovary syndrome (PCOS) is characterized by intricate interactions between the target nutrients, mechanisms of absorption, and metabolic processes involved in modulating hormonal balance, insulin sensitivity, and inflammation. Understanding the mechanisms through which the various groups of food and bioactive molecules influence PCOS outcomes is critical in the construction of evidence-based nutritional approaches. This review addresses the key nutrients in the main food groups, their mode of absorption, and the distinctive way in which they affect PCOS pathophysiology.

Carbohydrates: Glycemic Index and Metabolic Effects

The ingested carbohydrates can be divided according to their glycemic index (GI), which determines how they affect blood sugar. Low-glycemic index foods are whole grains, legumes, and most fruits and vegetables, while high-glycemic index foods are refined grains, processed foods, and sweetened drinks (Johnson et al., 2025). Complex polysaccharides, dietary fiber (soluble and insoluble), and simple sugars are the major bioactive compounds.Carbohydrate absorption is primarily through the small intestine using specific transporters. Dietary fiber carbohydrates undergo partial fermentation by colonic microbes, producing short-chain fatty acids (SCFAs) that influence metabolic pathways. Soluble fiber has been found most effective to control glycemic response and insulin sensitivity through delayed gastric emptying and reduced nutrient uptake rate (Xie et al., 2021).

Low-glycemic-index diets significantly improve PCOS symptoms through a variety of mechanisms. Greater dietary fiber intake reduces the glycemic index and glycemic load of diet and induces smaller blood sugar increases and reduced circulating insulin levels, thereby reducing insulin resistance (Tsitsou et al., 2023). In PCOS females, increased fiber intake was found to be a key predictor of reduced BMI after 16 weeks in 57 overweight women (BMI > 27 kg/m²) between 18 and 40 years old (Nybacka et al., 2017).

A randomized controlled trial involving 37 women with PCOS demonstrated that low-glycemic-index diets resulted in more ovulatory cycles and improved insulin resistance with decreased serum androgen levels compared to normal glycemic index diets (Sordia-Hernández et al., 2016). Meta-analysis findings indicate that low-GI diets reduce HOMA-IR, fasting insulin, total cholesterol, LDL cholesterol, triglycerides, waist circumference, and total testosterone levels compared with high-GI diets in PCOS women (Szczuko et al., 2019). An eucaloric 41% carbohydrate diet for 8 weeks was associated with long-term improvements in carbohydrate metabolism and a 23% reduction in testosterone, perhaps through decreased insulin levels (Gower et al., n.d.).

Lipids: Fatty Acid Composition And Hormonal Regulation

Food lipids include saturated fatty acids (SFAs), monounsaturated fatty acids (MUFAs), and polyunsaturated fatty acids (PUFAs) of particular importance of the omega-3 fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). MUFAs from olive oil and omega-3 PUFAs from fish are highlighted in the Mediterranean diet, while limiting SFAs from processed and red meat.

Lipid absorption occurs through micelle formation in the small intestine, with dissimilar rates and destinies for various fatty acids. Omega-3 fatty acids are metabolized into bioactive metabolites like resolvins and protectins with anti-inflammatory activities. Dietary fat composition regulates insulin sensitivity through alteration of cell membrane composition.

The metabolic quality of fats plays a critical role in determining PCOS symptoms. Intervention studies and epidemiologic evidence confirm that saturated fats worsen insulin resistance, while monounsaturated and polyunsaturated fatty acids enhance insulin sensitivity (Calcaterra et al., n.d.). Omega-3 fatty acids have potent anti-inflammatory properties that enhance insulin sensitivity and lower androgen levels through TGF-β pathways (Prabhu & Valsala Gopalakrishnan, 2021).

A double-blind randomized clinical trial on 64 women with PCOS and overweight/obesity showed significant improvement in lipid profiles, insulin resistance, and serum adiponectin concentration following omega-3 supplementation (Mohammadi et al., 2012). Co-administration of omega-3 and vitamin E has been shown to enhance parameters of mental health and PPAR-γ gene expression, IL-8, and TNF-α in PCOS women, as indicated by studies (Jamilian et al., 2018). Additional research showed that the use of omega-3 fatty acid supplements enhanced LH, LH/FSH ratio, lipid profiles, waist circumference, adiponectin levels, and normalized menstrual cycles (Nadjarzadeh et al., 2015; Khani et al., 2017).

Polyphenols and Phytochemicals: Antioxidant and Anti-inflammatory Effects

The Dietary Phytochemical Index (DPI) measures the total phytochemical content of diets in terms of consumption of phytochemical-dense foods (Chi et al., 2024).

Greater adherence to phytochemical-dense diets is linked to lower PCOS risk and better metabolic and hormonal status. PCOS women have considerably lower DPI scores, which are linked with higher levels of hormones and metabolic markers of dysfunction. Greater DPI scores are linked with fewer PCOS complications such as acne, irregular menses, and better mental health outcomes (Chi et al., 2024). In one case-control study, subjects with the highest tertile DPI had an 87% lower prevalence of PCOS and fewer associated complications (Chi et al., 2024). High DPI scorers were women with significantly better endocrine profiles than low DPI scorers, highlighting the fact that it is the quality of diet and not merely vegetarian classification that is important in the successful management of PCOS. Studies on certain polyphenols revealed that curcumin in combination with metformin exhibited better efficacy in managing lipid profiles, glucose metabolism, hormonal indices, body weight, and BMI than single agents (Feghhi et al., 2024).

Dietary Fiber: Gut Health and Metabolic Regulation

Dietary fibre contains both soluble fibre (in fruits, vegetables, oats, legumes) and insoluble fibre (in whole grains, nuts, seeds). Soluble fibre comprises pectins, β-glucans, and fructooligosaccharides, whereas insoluble fibre consists mainly of cellulose and lignin.

Fiber is not absorbed per se but is fermented by colon microbiota, which releases short-chain fatty acids (acetate, propionate, butyrate) that are used as energy sources by colonocytes and modulate systemic metabolism. Soluble fiber gels in the gut, delaying nutrient absorption, and enhances glycemic control.

High dietary fiber enhances PCOS outcomes by various mechanisms such as glycemic control, satiation, and modulation of gut microbiota. Dietary fiber in PCOS women was inversely associated with insulin resistance, fasting insulin, glucose tolerance, and androgen levels (Cutler et al., 2019). The capacity of fiber to provide bulk with sustained glycemic control results in improved satiation and appetite regulation.

A randomized controlled trial of 48 women with PCOS showed that diets high in fruits, vegetables, whole grains, and low-fat dairy products led to decreased serum insulin, triglycerides, and LDL cholesterol levels, with significant increases in total antioxidant capacity and glutathione concentrations (Gautam et al., 2025). Studies repeatedly illustrate that higher dietary fiber, and especially soluble fiber, intake adequately regulates glycemic responses and improves insulin sensitivity among populations with PCOS.

Anti-inflammatory Foods: Cytokine Modulation

Anti-inflammatory foods are those containing high amounts of omega-3 fatty acids, antioxidant vitamins (C and E), polyphenols, and certain compounds such as curcumin and quercetin. Empirical Dietary Inflammatory Pattern (EDIP) score measures the overall inflammatory potential of diets, with lower EDIP scores reflecting anti-inflammatory effects.

Anti-inflammatory substances are taken up by different mechanisms and act through modulation of nuclear factor kappa B (NF-κB) signaling, cyclooxygenase activity, and cytokine secretion. These nutrients regulate inflammatory processes at the cellular level by means of transcriptional and post-transcriptional processes.

Systemic inflammation is an important etiologic factor in PCOS, with pro-inflammatory cytokines from adipocytes stimulating macrophage activation and resulting in chronic low-grade inflammation. Anti-inflammatory diets reduce this inflammation and resultant metabolic complications (Patel, 2018). Increased EDIP scores correlate with higher PCOS risk through inflammatory pathways.

A case-control study revealed that PCOS risk was greater among participants with higher EDIP scores than those with lower inflammatory diets (Sharkesh et al., 2023). Consumption of refined grains and red/processed meat was linked with elevated EDIP scores and greater systemic inflammation markers. Clinical trials with green cardamom indicated dramatic reductions in TNF-α, IL-6, and CRP serum levels in women with PCOS compared to the control group (Cheshmeh et al., 2022). Clinical studies with raw red onion intake in obese/overweight women with PCOS indicated reduced serum adiponectin, leptin, and hs-CRP levels (Ebrahimi-Mamaghani et al., 2014).

Ketogenic and Very Low-Carbohydrate Diets

Ketogenic diets consist of 55-60% fat, 35% protein, and 5-10% carbohydrates, compelling the body into ketosis where the body runs on fat instead of carbohydrates as the main energy source. Very-low-calorie ketogenic diets (VLCKD) tend to limit daily carb consumption to below 30g while keeping protein within 1.1-1.2 g/kg optimal body weight (Masood et al., 2023).

The sharp decrease in carbohydrate consumption causes the depletion of glycogen reserves and enhanced oxidation of fatty acids, yielding ketone bodies (β-hydroxybutyrate, acetoacetate, acetone) that can be used as energy substrates. The shift in metabolism lowers glucose stimulation of pancreatic β-cells, leading to reduced insulin output and enhanced insulin sensitivity.

Ketogenic diets show better effects in weight loss, correction of lipid profiles, and improvement of hormonal parameters in women with PCOS. Due to high fat content, satiation is achieved along with decreased food cravings. Results from meta-analysis indicate that ketogenic diets reduce significantly the weight, BMI, waist circumference, and fat mass in obese and overweight women with PCOS (Xing et al., 2024).

A comparison of ketogenic with Mediterranean diets in PCOS women demonstrated significantly higher weight loss in the ketogenic diet (-10.9 kg) than in the Mediterranean diet (-5.1 kg), with better improvements in triglycerides, cholesterol, LDL, blood glucose, insulin, C-peptide, HOMA-IR, LH/FSH ratio, and sex hormone-binding globulin (Masood et al., 2023). The research illustrated that ketogenic diets were superior to Mediterranean diets for short-term improvement in metabolism, but long-term viability is a consideration.

Micronutrients And Supplements

Vitamin D

Vitamin D acts as both a nutrient and hormone with potent antioxidant activity to reduce oxidative stress and inflammation, while also neutralizing cytotoxic effects of reactive oxygen species. It is absorbed from the small intestine in a manner dependent on sufficient intake of fat for maximal bioavailability.

Clinical trials prove that vitamin D supplementation in vitamin-deficient women with PCOS enhances glucose homeostasis measures, hs-CRP, and malondialdehyde values (Maktabi et al., 2017). Other research indicates enhancing insulin sensitivity, glucose metabolism, lipid profiles, and adiponectin values after vitamin D supplementation (Javed et al., 2019; Seyyed Abootorabi et al., 2018).

Magnesium

Magnesium deficiency is found to be prevalent in obese type 2 diabetic patients and is linked to insulin resistance. Intracellular magnesium depletion is a universal characteristic of insulin resistance where increased levels of insulin decrease the capacity of cells to accumulate magnesium (Barbagallo & Dominguez, 2015).

Inositol And Phytic Acid

Phytic acid is also a valuable source of myo-inositol, which is an insulin-sensitizing substance. Phytic acid is frequently lost during food processing in developed foods, which may contribute to decreased sensitivity to insulin among industrialized nations. High dietary intake of phytic acid-containing foods can improve quality of life among PCOS patients and decrease prevalence and complications (Porcaro et al., 2016).

Probiotics And Gut Microbiota Modulation

Probiotic treatments usually comprise Lactobacillus and Bifidobacterium species that replenish the normal gut microbiota diversity and composition. These nonpathogenic bacteria synthesize metabolites that regulate host metabolism and suppress systemic inflammation.

Dysbiosis of gut microbiota in PCOS is responsible for insulin resistance by changing short-chain fatty acid production, lipopolysaccharides, and gut-brain axis signaling. Probiotic supplementation restores the populations of beneficial bacteria and augments metabolic parameters.

Clinical trials with multispecies probiotic supplement for 8 weeks presented enhanced pancreatic β-cell function and CRP levels in women with PCOS (Shoaei et al., 2015). A double-blind study with synbiotic supplement (Lactobacillus and Bifidobacterium strains) for 12 weeks presented positive effects on insulin resistance, triglycerides, and VLDL cholesterol levels (Heshmati et al., 2019). Meta-analyses indicate that probiotic/synbiotic supplement reduces insulin and triglyceride levels and elevates HDL levels in women with PCOS.

Clinical Implications and Therapeutic Applications

Personalized Nutrition Strategies

The diversity of PCOS presentations indicates that personalized nutrition strategies might be required for maximal benefit. Knowledge of individual differences in gut microbiota, genetic background, metabolic profile, and PCOS phenotype might allow for individualized dietary interventions that take into account personal differences in nutrient metabolism and response to therapy.

Integrated Dietary Strategies

Successful PCOS treatment is probably best achieved through integration of several dietary approaches instead of targeting individual nutrients. An aggregation of low-glycemic-index carbohydrates, anti-inflammatory foods, sufficient fiber, favorable fats, and selective micronutrient supplementation may yield synergistic effects on enhancing insulin sensitivity, lowering inflammation, and achieving optimal hormonal balance.

Long-term Sustainability

Whereas acute interventions such as ketogenic diets are likely to yield quick gains in metabolic indices, chronic dietary habits such as Mediterranean-style diets could be more sustainable and have wider health advantages. The focus should be put on diet quality and overall nutrient adequacy rather than on forms of restrictive feeding that could prove challenging to adhere to.

CONCLUSION

The findings identify differential therapeutic profiles in response to leading dietary modalities: low-glycemic index diets exhibit greater effectiveness in enhancing insulin sensitivity and mitigating hyperandrogenism via regulation of glucose-insulin dynamics;

This review shows that diet influences PCOS through multiple interconnected pathways including gut microbiota modulation, direct hormonal regulation via insulin-androgen feedback loops, anti-inflammatory mechanisms through polyphenol and omega-3 fatty acid action, and metabolic reprogramming through macronutrient manipulation. The gut-brain axis emerges as a critical mediator, with dietary components influencing neurotransmitter production and stress responses that directly impact reproductive function.

The findings of the review suggest that every dietary strategy has distinct mechanisms: fiber and low-GI carbohydrates mostly operate via glycemic control and SCFA production; polyphenols act with antioxidant and anti-inflammatory actions coupled with gut microbiota modulation; omega-3 fatty acids inhibit inflammation via specialized pro-resolving mediators; ketogenic diets mediate metabolic flexibility and swift weight loss; and micronutrients target specific enzymatic and hormonal pathways. Clinical data repeatedly show improvements in reproductive, metabolic, and psychological aspects.

Limitations

This review has some limitations. The search strategy was largely confined to English-language articles and may have omitted valuable studies in languages other than English. The review includes articles published till February 2025, but the very dynamic nature of nutritional science in PCOS is such that new findings and emerging interventions may not necessarily be reflected.

Most of the included trials exhibit methodological heterogeneity regarding PCOS diagnosis, diet intervention protocols, outcomes, and durations of follow-up, which may hamper comparisons.

Practical Implications

Clinical centres and healthcare institutions can adopt various evidence-based practices to maximize dietary interventions in PCOS management. Primary suggestions include the development of integrated nutrition counseling services in endocrinology and reproductive health departments, with specialized dietitians trained in PCOS pathophysiology and evidence-based dietary management.

Implementation strategies should feature individualized treatments relying on PCOS phenotype, metabolic characterization, and personal preferences. Low-glycemic index and Mediterranean dietary models are the first-line dietary advice in light of high levels of evidence, feasibility, and generalized applicability. Ketogenic therapies can be attempted in short-term metabolic correction in carefully selected patients under well-planned monitoring and tapering protocols.

Scope For Further Research

This review reveals a number of key gaps in the research. Precision nutrition that develops tailored dietary advice from individual biological signatures and treatment responses, large-scale studies must incorporate genomic data, gut microbiome analysis, metabolomic profiles, and machine learning algorithms can be researched further.

Methodological designs that can be explored include randomized controlled trials of longer duration of follow-up (≥5 years), standardized outcome assessments between studies, and implementation science studies investigating real-world effectiveness and scalability of diet programs.

Cross-disciplinary research potential involves cooperation among nutritionists, reproductive endocrinologists, microbiologists, behavioral scientists, and health economists to design inclusive intervention models. The integration of digital health technologies such as smartphone apps, wearable technology, and artificial intelligence-based dietary guidance is a key area for innovation.

The evidence presented in this review unequivocally establishes dietary interventions as fundamental, evidence-based therapeutic modalities for PCOS management, with the potential to transform clinical practice and improve outcomes for millions of women worldwide. As we advance toward an era of precision medicine, the integration of sophisticated nutritional strategies, personalized based on individual biological profiles and cultural contexts, represents not merely an adjunctive treatment option but a cornerstone of comprehensive PCOS care.

REFERENCES

1. Banez, M. J., Geluz, M. I., Chandra, A., Hamdan, T., Biswas, O. S., Bryan, N. S., & Von Schwarz, E. R. (2020). A systemic review on the antioxidant and anti-inflammatory effects of resveratrol, curcumin, and dietary nitric oxide supplementation on human cardiovascular health. *Nutrition Research*, *78*, 11-26.
2. Barbagallo, M., & Dominguez, L. J. (2015). Magnesium and type 2 diabetes. *World Journal of Diabetes*, *6*(10), 1152-1157.
3. Barre, D. E., & Mizier-Barre, K. A. (2019). Lignans' potential in pre and post-onset type 2 diabetes management. *Current Diabetes Reviews*, *16*(1), 2-11.
4. Baskind, N. E., & Balen, A. H. (2016). Hypothalamic-pituitary, ovarian and adrenal contributions to polycystic ovary syndrome. *Best Practice & Research Clinical Obstetrics & Gynaecology*, *37*, 80-97.
5. Calcaterra, V., Magenes, V. C., Massini, G., De Sanctis, L., Fabiano, V., & Zuccotti, G. (n.d.). High fat diet and polycystic ovary syndrome (PCOS) in adolescence: An overview of nutritional strategies. *Nutrients*. https://pubmed.ncbi.nlm.nih.gov/38612972/
6. Cheshmeh, S., Elahi, N., Ghayyem, M., Mosaieby, E., Moradi, S., Pasdar, Y., Tahmasebi, S., & Moradinazar, M. (2022). Effect of green cardamom on the expression of genes implicated in obesity and diabetes among obese women with polycystic ovary syndrome: A double blind randomized controlled trial. *Genes & Nutrition*, *17*, 17.
7. Chi, Y., Yue, R., Lv, Y., Li, H., & Liao, W. (2024). The dietary phytochemical index and its relation to polycystic ovary syndrome: A case-control study. *Journal of Ovarian Research*. https://pubmed.ncbi.nlm.nih.gov/39550600/
8. Cutler, D. A., Pride, S. M., & Cheung, A. P. (2019). Low intakes of dietary fiber and magnesium are associated with insulin resistance and hyperandrogenism in polycystic ovary syndrome: A cohort study. *Food Science & Nutrition*, *7*, 1426-1437.
9. Desai, N. A., & Patel, S. S. (2023). Increased prevalence of polycystic ovary syndrome (PCOS) in Indian women: A systematic review and meta-analysis. *Reproductive Biology and Endocrinology*, *21*(1), 1-15.
10. Durazzo, A., Lucarini, M., Souto, E. B., Cicala, C., Caiazzo, E., Izzo, A. A., ... & Santini, A. (2019). Polyphenols: A concise overview on the chemistry, occurrence, and human health. *Phytotherapy Research*, *33*(9), 2221-2243.
11. Ebrahimi-Mamaghani, M., Saghafi-Asl, M., Pirouzpanah, S., & Asghari-Jafarabadi, M. (2014). Effects of raw red onion consumption on metabolic features in overweight or obese women with polycystic ovary syndrome: A randomized controlled clinical trial. *Journal of Obstetrics and Gynaecology Research*, *40*, 1067-1076.
12. Feghhi, F., Ghaznavi, H., Sheervalilou, R., Razavi, M., & Sepidarkish, M. (2024). Effects of metformin and curcumin in women with polycystic ovary syndrome: A factorial clinical trial. *Phytomedicine*, *125*, 155330. https://pubmed.ncbi.nlm.nih.gov/39461199
13. Fraga, C. G., Croft, K. D., Kennedy, D. O., & Tomás-Barberán, F. A. (2019). The effects of polyphenols and other bioactives on human health. *Food & Function*, *10*(2), 514-528.
14. Furusawa, Y., Obata, Y., Fukuda, S., Endo, T. A., Nakato, G., Takahashi, D., ... & Ohno, H. (2013). Commensal microbe-derived butyrate induces the differentiation of colonic regulatory T cells. *Nature*, *504*(7480), 446-450.
15. Gautam, R., Maan, P., Jyoti, A., Kumar, A., Malhotra, N., & Arora, T. (2025). The role of lifestyle interventions in PCOS management: A systematic review. *Nutrients*. https://pubmed.ncbi.nlm.nih.gov/39861440/
16. Gower, B. A., Chandler-Laney, P. C., Ovalle, F., Goree, L. L., Azziz, R., Desmond, R. A., Granger, W. M., Goss, A. M., & Bates, G. W. (n.d.). Favourable metabolic effects of a eucaloric lower-carbohydrate diet in women with PCOS. *Clinical Endocrinology*. https://pubmed.ncbi.nlm.nih.gov/23444983/
17. Heshmati, J., Farsi, F., Yosaee, S., Razavi, M., Rezaeinejad, M., Karimie, E., & Sepidarkish, M. (2019). The effects of probiotics or synbiotics supplementation in women with polycystic ovarian syndrome: A systematic review and meta-analysis of randomized clinical trials. *Probiotics and Antimicrobial Proteins*, *11*, 1236-1247.
18. Jamilian, M., Foroozanfard, F., Mirhosseini, N., Kavossian, E., Aghadavod, E., Bahmani, F., Ostadmohammadi, V., Kia, M., Eftekhar, T., Ayati, E., Shafabakhsh, R., & Asemi, Z. (2019). Effects of melatonin supplementation on hormonal, inflammatory, genetic, and oxidative stress parameters in women with polycystic ovary syndrome. *Frontiers in Endocrinology*, *10*, 273.
19. Jamilian, M., Shojaei, A., Samimi, M., Afshar Ebrahimi, F., Aghadavod, E., Karamali, M., Taghizadeh, M., Jamilian, H., Alaeinasab, S., Jafarnejad, S., & Asemi, Z. (2018). The effects of omega-3 and vitamin E co-supplementation on parameters of mental health and gene expression related to insulin and inflammation in subjects with polycystic ovary syndrome. *Journal of Affective Disorders*, *229*, 41-47.
20. Javed, Z., Papageorgiou, M., Deshmukh, H., Kilpatrick, E. S., Mann, V., Corless, L., Abouda, G., Rigby, A. S., Atkin, S. L., & Sathyapalan, T. (2019). A randomized, controlled trial of vitamin D supplementation on cardiovascular risk factors, hormones, and liver markers in women with polycystic ovary syndrome. *Nutrients*, *11*, 188.
21. Johnson, C., Garipoğlu, G., Jeanes, Y., Frontino, G., & Costabile, A. (2025). The role of diet, glycaemic index and glucose control in polycystic ovary syndrome (PCOS) management and mechanisms of progression. *Current Nutrition Reports*. https://pubmed.ncbi.nlm.nih.gov/39753786/
22. Kelley, S. T., Skarra, D. V., Rivera, A. J., & Thackray, V. G. (2016). The gut microbiome is altered in a letrozole-induced mouse model of polycystic ovary syndrome. *PLoS One*, *11*(1), e0146509.
23. Khani, B., Mardanian, F., & Fesharaki, S. J. (2017). Omega-3 supplementation effects on polycystic ovary syndrome symptoms and metabolic syndrome. *Journal of Research in Medical Sciences*, *22*, 64.
24. Khobragade, N. H., Sheth, D. B., Patel, C. A., Beladiya, J. V., Patel, S., & Dalal, M. (2024). Polycystic ovary syndrome: Insights into its prevalence, diagnosis, and management with special reference to gut microbial dysbiosis. *Steroids*, *208*, 109455. https://doi.org/10.1016/j.steroids.2024.109455
25. Krug, I., Giles, S., & Paganini, C. (2019). Binge eating in patients with polycystic ovary syndrome: Prevalence, causes, and management strategies. *Neuropsychiatric Disease and Treatment*, *15*, 1273-1285. https://doi.org/10.2147/NDT.S168944
26. Li, Z., Summanen, P. H., Komoriya, T., & Henning, S. M. (2023). Unique roles in health promotion of dietary flavonoids through gut microbiota regulation: Current understanding and future perspectives. *Food Chemistry*, *399*, 133959.
27. Lim, S. S., Kakoly, N. S., Tan, J. W. J., Fitzgerald, G., Bahri Khomami, M., Joham, A. E., ... & Moran, L. J. (2018). Metabolic syndrome in polycystic ovary syndrome: A systematic review, meta-analysis and meta-regression. *Obesity Reviews*, *20*(2), 339-352. https://doi.org/10.1111/obr.12762
28. Lizneva, D., Suturina, L., Walker, W., Brakta, S., Gavrilova-Jordan, L., & Azziz, R. (2016). Criteria, prevalence, and phenotypes of polycystic ovary syndrome. *Fertility and Sterility*, *106*(1), 6-15. https://doi.org/10.1016/j.fertnstert.2016.05.003
29. Maktabi, M., Chamani, M., & Asemi, Z. (2017). The effects of vitamin D supplementation on metabolic status of patients with polycystic ovary syndrome: A randomized, double-blind, placebo-controlled trial. *Hormone and Metabolic Research*, *49*, 493-498.
30. Masood, I., Noreen, S., Raza, K., Khalid, W., Rahim, M. A., & Muhamedahmed, K. A. (2023). Effect of ketogenic diet and hypocaloric Mediterranean diet on metabolic and endocrine parameter in women suffering from polycystic ovary syndrome. *Food Science and Technology*, *43*, e122022. https://www.tandfonline.com/doi/full/10.1080/10942912.2023.2275528
31. Mohammadi, E., Rafraf, M., Farzadi, L., Asghari-Jafarabadi, M., & Sabour, S. (2012). Effects of omega-3 fatty acids supplementation on serum adiponectin levels and some metabolic risk factors in women with polycystic ovary syndrome. *Asia Pacific Journal of Clinical Nutrition*, *21*, 511-518.
32. Molino, S., Lerma-Aguilera, A., Jiménez-Hernández, N., Gosalbes, M. J., Rufián-Henares, J. A., & Francino, M. P. (2021). Enrichment of food with tannin extracts promotes healthy changes in the human gut microbiota. *Frontiers in Microbiology*, *12*, 625782.
33. Moran, L. J., Misso, M. L., Wild, R. A., & Norman, R. J. (2010). Impaired glucose tolerance, type 2 diabetes and metabolic syndrome in polycystic ovary syndrome: A systematic review and meta-analysis. *Human Reproduction Update*, *16*(4), 347-363. https://doi.org/10.1093/humupd/dmq001
34. Nadjarzadeh, A., Dehghani-Firouzabadi, R., Daneshbodi, H., Lotfi, M. H., Vaziri, N., & Mozaffari-Khosravi, H. (2015). Effect of omega-3 supplementation on visfatin, adiponectin, and anthropometric indices in women with polycystic ovarian syndrome. *Journal of Reproduction & Infertility*, *16*, 212-220.
35. Nisa, K. U., Tarfeen, N., Mir, S. A., Waza, A. A., Ahmad, M. B., & Ganai, B. A. (2023). Molecular mechanisms in the etiology of polycystic ovary syndrome (PCOS): A multifaceted hypothesis towards the disease with potential therapeutics. *Indian Journal of Clinical Biochemistry*, *38*(1), 1-19.
36. Norman, R., Wu, R., & Stankiewicz, M. (2004). Polycystic ovary syndrome. *Medical Journal of Australia*, *180*(3), 132-137.
37. Nybacka, Å., Hellström, P. M., & Hirschberg, A. L. (2017). Increased fibre and reduced trans fatty acid intake are primary predictors of metabolic improvement in overweight polycystic ovary syndrome—Substudy of randomized trial between diet, exercise and diet plus exercise for weight control. *Clinical Endocrinology*, *87*(6), 680-688. https://doi.org/10.1111/cen.13427
38. Patel, S. (2018). Polycystic ovary syndrome (PCOS), an inflammatory, systemic, lifestyle endocrinopathy. *Journal of Steroid Biochemistry and Molecular Biology*, *182*, 27-36.
39. Porcaro, G., Filati, P., & Unfer, V. (2016). Journal of Nutritional Health & Food Science. https://symbiosisonlinepublishing.com/nutritionalhealth-foodscience/nutritionalhealth-foodscience78.php
40. Prabhu, Y. D., & Valsala Gopalakrishnan, A. (2021). Can polyunsaturated fatty acids regulate polycystic ovary syndrome via TGF-β signalling? *Life Sciences*, *276*, 119416.
41. Santhakumar, A. B., Battino, M., & Alvarez-Suarez, J. M. (2018). Dietary polyphenols: Structures, bioavailability and protective effects against atherosclerosis. *Food and Chemical Toxicology*, *113*, 49-65.
42. Seyyed Abootorabi, M., Ayremlou, P., Behroozi-Lak, T., & Nourisaeidlou, S. (2018). The effect of vitamin D supplementation on insulin resistance, visceral fat and adiponectin in vitamin D deficient women with polycystic ovary syndrome: A randomized placebo-controlled trial. *Gynecological Endocrinology*, *34*, 489-494.
43. Sharkesh, E. Z., Abbasi, B., Nazari, L., & Keshavarz, S. A. (2023). Empirical dietary inflammatory pattern is positively associated with polycystic ovary syndrome: A case control study. *Nutrition Research*, *111*, 46-56. https://pubmed.ncbi.nlm.nih.gov/38219291

44. Shoaei, T., Heidari-Beni, M., Tehrani, H. G., Feizi, A., Esmaillzadeh, A., & Askari, G. (2015). Effects of probiotic supplementation on pancreatic β-cell function and C-reactive protein in women with polycystic ovary syndrome: A randomized double-blind placebo-controlled clinical trial. *International Journal of Preventive Medicine*, *6*, 27.
45. Sordia-Hernández, L. H., Rodríguez, P. A., Rodriguez, D. S., Guzmán, S. T., Zenteno, E. S. S., González, G. G., & Patiño, R. I. (2016). Effect of a low-glycemic diet in patients with polycystic ovary syndrome and anovulation—A randomized controlled trial. *Clinical and Experimental Obstetrics & Gynecology*, *43*, 555-559.
46. Stener-Victorin, E., Padmanabhan, V., Walters, K. A., Campbell, R. E., Benrick, A., Giacobini, P., ... & Dumesic, D. A. (2020). Animal models to understand the etiology and pathophysiology of polycystic ovary syndrome. *Endocrine Reviews*, *41*(4), bnaa010.
47. Szczuko, M., Zapalowska-Chwyć, M., & Drozd, R. (2019). A low glycemic index decreases inflammation by increasing the concentration of uric acid and the activity of glutathione peroxidase (GPX3) in patients with polycystic ovary syndrome (PCOS). *Molecules*, *24*, 1508.
48. Teede, H., Deeks, A., & Moran, L. (2010). Polycystic ovary syndrome: A complex condition with psychological, reproductive and metabolic manifestations that impacts on health across the lifespan. *BMC Medicine*, *8*, 41. https://doi.org/10.1186/1741-7015-8-41
49. Tomova, A., Bukovsky, I., Rembert, E., Yonas, W., Alwarith, J., Barnard, N. D., & Kahleova, H. (2019). The effects of vegetarian and vegan diets on gut microbiota. *Frontiers in Nutrition*, *6*, 47.
50. Tsitsou, S., Athanasaki, C., Dimitriadis, G., & Papakonstantinou, E. (2023). Acute effects of dietary fiber in starchy foods on glycemic and insulinemic responses: A systematic review of randomized controlled crossover trials. *Nutrients*, *15*(10), 2383. https://doi.org/10.3390/nu15102383
51. Tzounis, X., Rodriguez-Mateos, A., Vulevic, J., Gibson, G. R., Kwik-Uribe, C., & Spencer, J. P. (2008). Flavanol monomer-induced changes to the human faecal microflora. *British Journal of Nutrition*, *99*(4), 782-792.
52. Wan, M. L. Y., Co, V. A., & El-Nezami, H. (2021). Dietary polyphenol impact on gut health and microbiota. *Critical Reviews in Food Science and Nutrition*, *61*(4), 690-711.
53. Wang, Y., Gapstur, S. M., Carter, B. D., Hartman, T. J., Stevens, V. L., Gaudet, M. M., & McCullough, M. L. (2022). Anti-fatigue activity of parsley (Petroselinum crispum) flavonoids via regulation of oxidative stress and gut microbiota in mice. *Journal of Functional Foods*, *89*, 104963.
54. Wei, H., Huo, P., Liu, S., Huang, H., & Zhang, S. (2022). Posttranslational modifications in pathogenesis of PCOS. *Frontiers in Endocrinology*, *13*, 1024320.
55. Xia, T., Zhang, B., Duan, W., Zhang, J., & Wang, M. (2022). The anti-diabetic activity of polyphenols-rich vinegar extract in mice via regulating gut microbiota and liver inflammation. *Food Chemistry*, *393*, 133443.
56. Xie, Y., Gou, L., Peng, M., Zheng, J., & Chen, L. (2021). Effects of soluble fiber supplementation on glycemic control in adults with type 2 diabetes mellitus: A systematic review and meta-analysis of randomized controlled trials. *Clinical Nutrition*, *40*(4), 1800-1810. https://doi.org/10.1016/j.clnu.2020.10.032
57. Xing, N. N., Ren, F., & Yang, H. (2024). Effects of ketogenic diet on weight loss parameters among obese or overweight patients with polycystic ovary syndrome: A systematic review and meta-analysis of randomized controlled trails. *Food & Nutrition Research*, *68*, 9835.
58. Yildiz, B. O., Bozdag, G., Yapici, Z., Esinler, I., & Yarali, H. (2012). Prevalence, phenotype and cardiometabolic risk of polycystic ovary syndrome under different diagnostic criteria. *Human Reproduction*, *27*(10), 3067-3073. https://doi.org/10.1093/humrep/des232
59. Zhang, B., Xu, Y., Liu, S., Lv, H., Hu, Y., Wang, Y., ... & Liu, J. (2021). Intestinal pharmacokinetics of resveratrol and regulatory effects of resveratrol metabolites on gut barrier and gut microbiota. *Food Chemistry*, *357*, 129532.
60. Zhou, P., Feng, P., Liao, B., Fu, L., Shan, H., Cao, C., Luo, R., Peng, T., Liu, F., & Li, R. (2024). Role of polyphenols in remodeling the host gut microbiota in polycystic ovary syndrome. *Journal of Ovarian Research*, *17*(1), 78. https://doi.org/10.1186/s13048-024-01354-y