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Dietary Patterns and Nutritional Influences in Polycystic Ovary Syndrome: Implications for Metabolic and Reproductive Health

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Abstract

Polycystic Ovary Syndrome (PCOS) is a multifactorial endocrine–metabolic disorder characterized by hyperandrogenism, ovulatory dysfunction and metabolic abnormalities, including insulin resistance and low-grade chronic inflammation. Although genetic predisposition contributes to its pathogenesis, accumulating evidence highlights the pivotal role of environmental and lifestyle factors, particularly dietary patterns in modulating disease onset, severity and progression. Nutrition functions not only as an energy source but also as a biological regulator influencing insulin signaling, oxidative stress, mitochondrial dynamics, adipokine secretion, gut microbiota composition and hormonal homeostasis. These interconnected metabolic pathways directly affect ovarian steroidogenesis, follicular development, menstrual regularity and fertility outcomes. Current research increasingly emphasizes overall dietary patterns rather than isolated nutrients, recognizing the complex interplay between macronutrient composition, glycemic load, inflammatory potential and metabolic flexibility. This review critically evaluates contemporary evidence regarding the influence of carbohydrate quality, dietary fats, protein intake, micronutrient status, inflammatory dietary indices, gut–endocrine interactions and meal timing on PCOS pathophysiology. Clinical implications of nutritional interventions, including low-glycemic index diets, anti-inflammatory dietary models and personalized nutrition approaches, are discussed in the context of improving metabolic and reproductive outcomes. Reframing dietary management of PCOS from a solely weight-centric model to a metabolically targeted strategy may enhance therapeutic efficacy. A mechanistic understanding of diet–metabolism–ovary interactions is essential for developing precision-based nutritional interventions to modulate disease over the long term and improve reproductive health.

Keywords: Dietary Patterns, Insulin Resistance, Metabolic Dysfunction, Nutritional Regulation, Polycystic Ovary Syndrome.

Introduction

Polycystic ovarian syndrome (PCOS) is one of the most frequent yet complex endocrine disorders affecting women of reproductive age worldwide. It is estimated to affect a considerable proportion of women during their reproductive years and represents a major cause of reproductive, metabolic and endocrine abnormalities. PCOS was originally described primarily as a reproductive disorder based on the clinical manifestations observed in affected women. These manifestations included irregular menstrual cycles, chronic anovulation (lack of ovulation), hyperandrogenism (excessive androgen production) and the presence of multiple cysts on the ovaries (1). When the disorder was first characterized, the underlying pathology was believed to originate mainly at the level of the ovaries and PCOS was therefore largely considered an ovarian dysfunction. However, scientific understanding of PCOS has evolved considerably over the past few decades. Advances in both metabolic and endocrine research have demonstrated that PCOS cannot be explained solely by ovarian abnormalities and instead represents a systemic disorder involving multiple physiological path-ways (2). This evolving perspective recognizes PCOS as a multifactorial

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condition in which reproductive disturbances coexist with complex metabolic dysfunctions. Women diagnosed with PCOS frequently present with metabolic abnormalities such as insulin resistance, dyslipidemia, increased central or visceral adiposity, endothelial dysfunction, oxidative stress and chronic low-grade inflammation. Importantly, these metabolic disturbances are not isolated phenomena but rather interconnected processes that collectively contribute to the pathophysiology of the disorder. Insulin resistance is considered a central metabolic abnormality that influences endocrine regulation by promoting hyperinsulinemia, which subsequently stimulates ovarian androgen production and disrupts normal follicular development.

These interactions illustrate that PCOS should be viewed as a systemic metabolic disorder with significant physiological implications rather than merely a reproductive condition. As scientific understanding of PCOS continues to expand, lifestyle-related factors have increasingly been recognized as important contributors to the development, expression and progression of the disorder. Environmental influences such as diet, physical activity and body weight regulation can significantly affect metabolic pathways that are closely linked to PCOS pathogenesis (3). Among the various lifestyle factors associated with PCOS, diet represents one of the most significant determinants of metabolic health because it directly influences several biological systems involved in energy regulation and endocrine function. Unlike many genetic risk factors that cannot be modified, dietary behavior is a modifiable environmental factor that can influence metabolic stability and disease progression. Nutrients are not only sources of energy but also act as biological signals that regulate cellular metabolism and endocrine activity. The metabolic effects of nutrients extend to multiple physiological pathways, including glucose–insulin homeostasis, lipid metabolism, inflammatory responses, oxidative stress regulation, mitochondrial activity and the composition of the gut microbiota. These interconnected cellular processes contribute to the maintenance of systemic metabolic balance and play an important role in regulating endocrine pathways that influence ovarian function and reproductive health (4).

Through these mechanisms, dietary intake can affect insulin sensitivity, inflammatory signaling, hormonal regulation and metabolic flexibility, all of which are closely associated with PCOS pathophysiology. Furthermore, the biological effects of nutrition extend beyond simple caloric intake; the quality, composition and timing of nutrient consumption may significantly influence metabolic responses. For example, diets high in refined carbohydrates or saturated fats may exacerbate metabolic disturbances, whereas diets rich in fiber, unsaturated fats and antioxidant compounds may improve metabolic regulation. These dietary influences demonstrate that nutrition plays an active role in shaping metabolic and endocrine homeostasis. Therefore, dietary patterns should be considered critical components in the management and prevention of metabolic dysfunction associated with PCOS. Another important characteristic of PCOS is the considerable heterogeneity observed in its clinical presentation and metabolic features. Women affected by PCOS may display a wide range of symptoms and metabolic profiles, which complicates both diagnosis and treatment strategies (5). Some individuals with PCOS maintain a normal body weight yet still exhibit insulin resistance, hyperandrogenism, or metabolic dysregulation, whereas others may present with obesity, dyslipidemia and chronic inflammatory states. This variability indicates that PCOS cannot be treated using a single uniform therapeutic approach and highlights the need for individualized management strategies tailored to each patient's metabolic profile. The heterogeneity of PCOS also reflects the complex interactions between genetic predisposition, hormonal regulation and environmental influences such as dietary habits. Consequently, understanding the relationship between nutrition and metabolic dysfunction is essential for developing effective therapeutic strategies. Investigating how dietary patterns influence metabolic pathways may provide valuable insights into the mechanisms responsible for disease progression and symptom severity. Nutritional interventions have the potential to address the underlying metabolic abnormalities associated with PCOS by improving

insulin sensitivity, reducing inflammation and restoring hormonal balance.

These improvements may ultimately contribute to better reproductive outcomes and overall metabolic health. Therefore, diet should not be viewed merely as a peripheral lifestyle factor but rather as an important regulator of biological function that can influence both metabolic and reproductive systems. Recognizing the central role of dietary patterns in PCOS management may help guide the development of targeted nutritional strategies aimed at reducing metabolic dysfunction and improving long-term health outcomes in women affected by this complex disorder (6, 7).

Metabolic Foundations of PCOS

PCOS is fundamentally rooted in metabolic dysfunction, with insulin resistance serving as a central pathological feature. Compensatory hyperinsulinemia amplifies ovarian androgen production, contributing to hyperandrogenism and disrupted follicular development. Chronic low-grade inflammation, oxidative stress and adipose tissue dysfunction further impair insulin signaling and endocrine balance (8). These interconnected metabolic disturbances form the biological basis linking nutrition, metabolic instability and reproductive dysfunction in PCOS.

Clinical manifestations and systemic complications of polycystic ovary syndrome (PCOS) include psychological symptoms, endocrine disturbances, reproductive dysfunction, metabolic abnormalities and associated comorbidities such as cardiovascular disease, type 2 diabetes, chronic kidney disease and metabolic fatty liver disease, as illustrated in Figure 1.

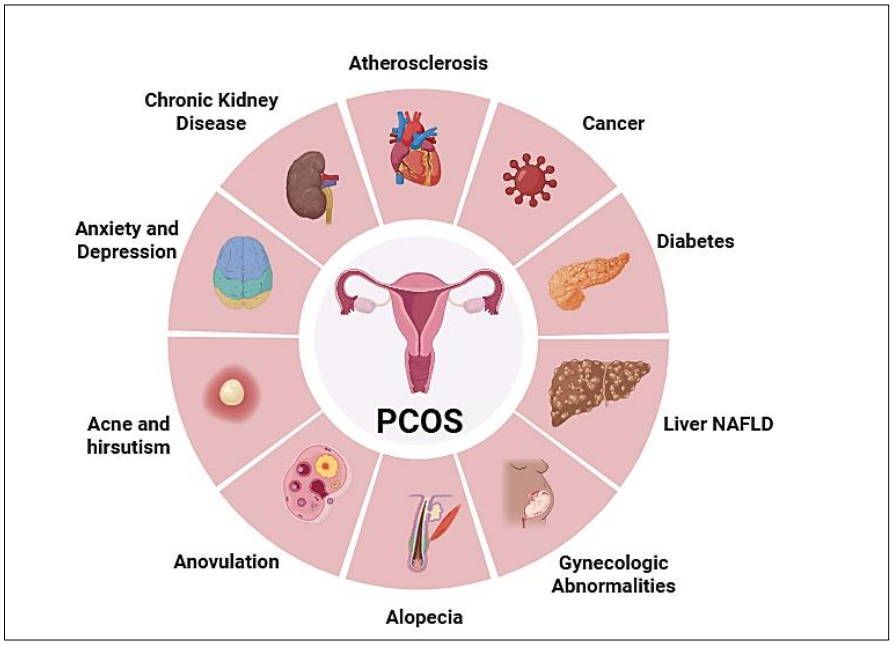

Figure 1: Disease Associated with PCOS and its Clinical Manifestations

Table 1: Mechanistic Links between Dietary Components and PCOS Pathophysiology

Dietary Component	Mechanism	Metabolic Effect	Reproductive Consequence	References
High Glycemic Load (refined carbohydrates)	Rapid glucose absorption → exaggerated insulin release → impaired IRS-1/PI3K signaling	Chronic hyperinsulinemia and insulin resistance	↑ Theca cell androgen synthesis; ↓ SHBG → ↑ bioavailable testosterone; anovulation	(8)
Saturated and Trans Fats	Activation of NF-κB and pro-inflammatory cytokines (↑ TNF-α, IL-6)	Chronic low-grade inflammation; impaired insulin signaling	Follicular arrest; altered steroidogenesis	(9)
Low Dietary Fiber	↓ SCFA production by gut microbiota; ↑ gut permeability (endotoxemia)	Gut dysbiosis; increased insulin resistance	Impaired ovulation; endocrine imbalance	(10, 11)
Antioxidant Deficiency	↑ ROS; ↓ antioxidant defense (SOD, GPx)	Oxidative stress; mitochondrial dysfunction	Reduced oocyte quality; follicle damage	(12, 13)

Excess Energy Intake	Adipocyte hypertrophy; adipokine imbalance (↓ adiponectin, ↑ leptin)	Adipose dysfunction; systemic insulin resistance	Exacerbated hyperinsulinemia and androgen excess	(14, 15)
Low Omega-3 Intake	Reduced anti-inflammatory eicosanoids; altered ω-6/ω-3 ratio	↑ Inflammatory signaling; impaired lipid metabolism	Worsened endocrine dysregulation; metabolic dysfunction	(16, 17)
Micronutrient Deficiency (Vit D, Zn, Mg)	Altered insulin receptor phosphorylation; disrupted steroidogenesis	Reduced insulin sensitivity; increased inflammation	Menstrual irregularity; steroid imbalance	(18)
Ultra-Processed Foods	High AGEs; altered microbiota composition	↑ Oxidative stress; systemic inflammation	Follicular dysfunction; potential reduced ovarian reserve	(19)

Note: Upward arrows (↑) indicate an increase, while downward arrows (↓) indicate a decrease in the respective biological processes

Insulin resistance refers to reduced responsiveness of body tissues to insulin, resulting in impaired glucose uptake and compensatory hyperinsulinemia. Dyslipidemia indicates abnormal lipid metabolism characterized by elevated triglycerides and low-density lipoprotein (LDL) cholesterol, along with decreased high-density lipoprotein (HDL) cholesterol. Hepatic steatosis and steatohepatitis represent excessive lipid accumulation and inflammation in the liver that may progress to fibrosis and cirrhosis as discussed in Table 1.

Insulin Resistance: A Central Metabolic Disturbance

Insulin resistance (IR) is a recognized key metabolic dysfunction that contributes to the underlying pathophysiology of PCOS. Unlike most metabolic disorders, where IR primarily develops due to obesity, women with PCOS are often observed to have IR phenotypes that occur independently of their body weight. At the cellular level, IR represents an inability of peripheral tissues (most importantly, skeletal muscle, liver and fat tissue) to respond to insulin, thereby disrupting glucose uptake and transport (19). Subsequently, disruptions in IR lead to impaired insulin activity and decreased ability to clear glucose from the bloodstream as well as compensatory adaptation(s) to metabolic disruption. To maintain homeostasis of blood glucose levels, pancreatic islet beta cells secrete increased amounts of insulin, causing a state of persistent or chronic hyperinsulinemia. Chronic hyperinsulinemia results not only from compensative IR but also serves as an endocrine disruptor (20).

Insulin is a type of hormone that has many different physiological functions. In addition to regulating blood sugar levels, insulin plays a role in regulating hormone production in the ovaries. It does this by having a direct effect on the theca cells in the ovary (the cells that help make testosterone from androgens). When there are higher levels of insulin, it stimulates the production of androgens by increasing the production of enzymes related to steroid production and also enhances the action of luteinizing hormone (LH) in producing androgens (20). At the same time, when there are high levels of insulin, insulin decreases the liver's ability to make sex hormone-binding globulin (SHBG). SHBG is a protein that binds to androgens and prevents them from being used. Thus, the decrease in SHBG increases the amount of free testosterone that circulates in the bloodstream. This combination of increased free testosterone and increased androgen production is a major contributing factor to hyperandrogenism, arrested follicle development and lack of ovulation that are characteristic features of polycystic ovary syndrome (PCOS). Insulin resistance is not only a metabolic abnormality, but it is constantly changing in response to external factors, especially diet (21). Diets that are high in glycemic index cause fast rises in blood sugar levels and exaggerated insulin secretion. These repeated fluctuations in metabolism can lead to progressively lower levels of insulin sensitivity, creating a cycle of high insulin levels and hormonal dysfunction in women with PCOS. In addition, insulin resistance acts as both a metabolic upset and as a hormone amplifier in women with PCOS. This demonstrates how nutrition, metabolism and reproductive function are integrated through insulin (22).

Hyperinsulinemia and Endocrine Amplification

Hyperinsulinemia role in PCOS is not limited to its role as a compensatory metabolic factor, but also functions as a powerful endocrine modulating hormone, which significantly dictates ovarian

physiology and the balance of reproductive hormones (i.e., oestrogen and androgens). High circulating insulin levels are also synergistic with luteinizing hormone (LH), thereby increasing the production of androgens in the theca cells of the ovary. This interaction represents a significant point of convergence between metabolic and reproductive control systems (23). In addition, insulin enhances LH-stimulated steroidogenic signaling by upregulating the activities of multiple steroidogenic enzymes and therefore increasing the production of androgens. This is an important mechanism by which hyperinsulinemia is directly associated with the excess production of androgens and therefore contributes to the disruptions in normal follicular development and ovulation associated with PCOS (24). Excess androgen production can have widespread physiological effects on reproductive physiology. For example, elevated circulating levels of androgens may interfere with follicular maturation and speed up the onset of follicular arrest (not ovulating). In addition, hyperandrogenism alters HPO axis regulatory mechanisms, thus perpetuating the hormonal imbalances that characterize PCOS (25).

Hormonal irregularities that arise from metabolic abnormalities can cause reproductive problems because of the hormone-based feedback system. As such, hyperinsulinemia is the main connection between metabolic dysfunction and issues found in the ovaries. One critical aspect of insulin-mediated endocrine amplification is that it is extremely responsive to dietary changes; thus, creating nutritional patterns to stabilize the glucose-insulin axes may help reduce the hyperinsulinemia-induced excess of androgens (26). Additionally, adopting a low-glycemic approach to diet can help reduce the amount of insulin the body is producing, which will subsequently reduce the stimulatory effects of insulin on steroid production from the ovaries. An understanding of how this all works occurs regarding dietary intervention to create a better approach for regulating metabolism and hormones. Dietary composition plays an important role in influencing the hormonal systems in PCOS, so hyperinsulinemia should be thought of as an endocrine disruptor that would be affected by the effects of metabolism or nutrition and therefore, eating patterns could have significant therapeutic implications (27).

Chronic Low-Grade Inflammation

PCOS is classified as having a low-grade chronic inflammatory and metabolic stress response, rather than an acute immune response. In fact, the chronic inflammatory response associated with PCOS is not clinically presented as an overt inflammatory process, but it is still considered a biologically significant inflammatory process. Higher levels of circulating inflammatory cytokines are believed to alter the basal metabolic process, as these cytokines can measurably interfere with normal insulin action through disturbing the insulin signaling pathway (28). Specifically, inflammatory cytokines can: (a) decrease the responsiveness of insulin receptors; (b) reduce the functioning of glucose transporter proteins; and (c) contribute to overall reduction in the metabolic efficiency of the metabolism. In addition, elevated levels of inflammatory cytokines promote body fat accumulation and facilitate the development of insulin resistance by providing additional signals for metabolic dysfunction and inflammation. Mechanisms related to the chronic inflammatory process in PCOS are broad, including, but not limited to, problems with adipose tissue function; oxidative damage; and excessive metabolic overload (29).

The cytokines found produced by adipocytes and immune cells (also known as macrophages) in the adipose tissue microenvironment can have an impact on regulating metabolism throughout the body. Even without being overweight or having excessive adipose, the presence of an abnormal adipose tissue metabolism can stimulate inflammation. This implies that all women with PCOS, whether overweight or not, will have physiological problems due to a metabolic disorder because of this evidence of inflammation and metabolic dysfunction related to abnormal adipose tissue function (30). In addition, the diet plays a major role in regulating inflammatory signals (i.e., whether your dietary habits promote or decrease production of inflammatory cytokines). Diets high in refined (simple) carbohydrates, processed foods, or saturated fats increase production of proinflammatory cytokines. Conversely, diets emphasizing consumption of unprocessed foods, dietary fiber, or unsaturated fats produce an anti-inflammatory response. Thus, nutrient quality influences the overall burden of inflammation in the body. In women with PCOS,

this diet-induced inflammation can contribute to metabolic instability through worsening insulin resistance and disorders of hormonal regulation. Therefore, when we understand that inflammation is a metabolically mediated phenomenon, there is considerable merit for utilizing dietary approaches that specifically target the inflammatory process (31). Thus, low-grade chronic inflammation serves as the critical mechanistic pathway linking nutritional behaviors to metabolic and reproductive dysfunctions in women diagnosed with PCOS.

Oxidative Stress and Cellular Dysfunction

Oxidative pressure is an important factor in PCOS pathophysiology but often goes unnoticed. It is a result of an unbalanced amount of ROS produced and the body's ability to counteract this through antioxidants. While ROS are naturally created through normal cellular metabolism, an excessive amount of ROS and/or high levels of oxidative stress can damage the integrity of cells and prevent them from functioning properly. In women with PCOS, higher oxidative stress levels are linked to increased insulin resistance, endothelial dysfunction and disruptions within the ovaries (32). Oxidative stress causes damage to the insulin receptor and impacts how well the body uses glucose. In addition, oxidative stress has a role in reproductive health as too much oxidative stress can cause problems in and around follicles in the ovaries, resulting in poor oocyte (egg) quality and/or impaired maturation of the oocyte. Lastly, oxidative damage contributes to a cascade of events that create inflammatory processes (causing further damage to the body) and metabolic abnormalities. All these factors work together and are directly related to each other: oxidative stress, inflammation and insulin resistance (33).

The amount of food we eat has a big impact on how well our bodies can fight off the harmful effects of free radicals (ROS- reactive species). Foods that don't have enough antioxidants decrease our ability to fight off ROS and, therefore, our bodies become more likely to undergo cell death due to damage by free radicals. In contrast, foods that have lots of fruits, vegetables and other foods that contain polyphenols increase our ability to fight against ROS (34). Additionally, fat content in our diets also affects oxidative stress; namely, saturated fat promotes the generation of ROS, whereas unsaturated fats help to protect us from oxidative stress. The primary way that food affects oxidative stress is through the availability of antioxidants in our diets, suggesting that nutrition provides a mechanism to help maintain the integrity of cells. The modulation of oxidative stress through nutrition is consistent with improving metabolic and reproductive outcomes for women with PCOS (35).

Adipose Tissue Dysfunction

The development of adipose tissue is now seen as an active endocrine organ that has many different functions besides just storing fat. Adipose tissue influences the metabolic functions of an individual throughout the body via the secretion of adipokines, cytokines and other bioactive substances that play key roles in such functions as how sensitive someone is to insulin, how inflammatory the body is, how hungry someone is, etc. Dysfunction of adipose tissue is one of the major underlying causes of both the metabolic and endocrine abnormalities that are present in PCOS (36). Changes in the biological make-up of adipose tissue are present in PCOS, even when significant obesity does not exist, demonstrating some of the underlying metabolic changes associated with PCOS.PCOS-related adipose tissue dysfunction is characterized by an altered structural and functional state. This includes abnormal adipocytes (hypertrophy), the ability to store lipids, dysregulation of adipokine secretion and excessive inflammatory activity. These changes create a state of metabolic inflexibility in which adipocytes have a limited ability to respond appropriately to nutrient levels or hormone signals (37). Central to the development of this metabolic inflexibility is the dysregulation of adipokine secretion. Adipokines such as adiponectin, leptin, resistin and visfatin regulate insulin signalling, glucose metabolism and inflammation. Decreased levels of adiponectin and the alteration of leptin signalling are increasingly reported in women with PCOS, leading to reduced insulin sensitivity and metabolic instability (38).

The obesity-related aspect of fat tissue disorder is very important. Immune cells and fat cells work together in the fat tissue microenvironment and produce immune chemicals that affect the way the entire body regulates its metabolism. In women with PCOS, the fat tissue has often been described

as having a pro-inflammatory mode of pro-inflammation due to an increase in macrophages entering the fat tissue along with an increase in the metabolism of immune chemicals. These immune chemicals then block the insulin receptor signal, making the person more resistant to insulin and causing high levels of insulin (39). Since it has already been proven that when insulin levels are high, it creates too many male hormones in the ovaries of women, fat tissue disorder also plays a part in reproductive health problems. The way we eat has a strong effect on how our fat tissue works. When someone eats lots of empty calories consistently, it causes stress to fat cells, fat to leave the fat cell and happens to create inflammation. On the other hand, if someone eats in a way that will help the body's metabolism, it allows for fat cells to function normally and produce the right number of hormones and less inflammation (40). Because of the way diet can both regulate fat tissue function as well as be regulated by fat tissue function, diet should be focused on when developing a health strategy for those who are overweight and/or have diabetes. Dietary methods should also not primarily focus on weight loss but rather on how diet can modify fat tissue function at the molecular and cellular level, thereby affecting how the body produces energy and produces hormones through the insulin receptor signal pathway (41).

Dietary Patterns in PCOS

Dietary patterns play a crucial role in the management of PCOS by influencing insulin sensitivity, inflammation and hormonal balance. Diets with low glycemic index carbohydrates help stabilize blood glucose and reduce hyperinsulinemia, a key driver of androgen excess. Western dietary patterns rich in refined sugars and saturated fats may worsen metabolic dysfunction and systemic inflammation. In contrast, Mediterranean-style diets emphasising whole grains, fruits, vegetables, legumes and healthy fats show beneficial effects on metabolic and reproductive outcomes in Figure 2. Therefore, overall dietary quality rather than calorie restriction alone is central to improving PCOS-related disturbances.

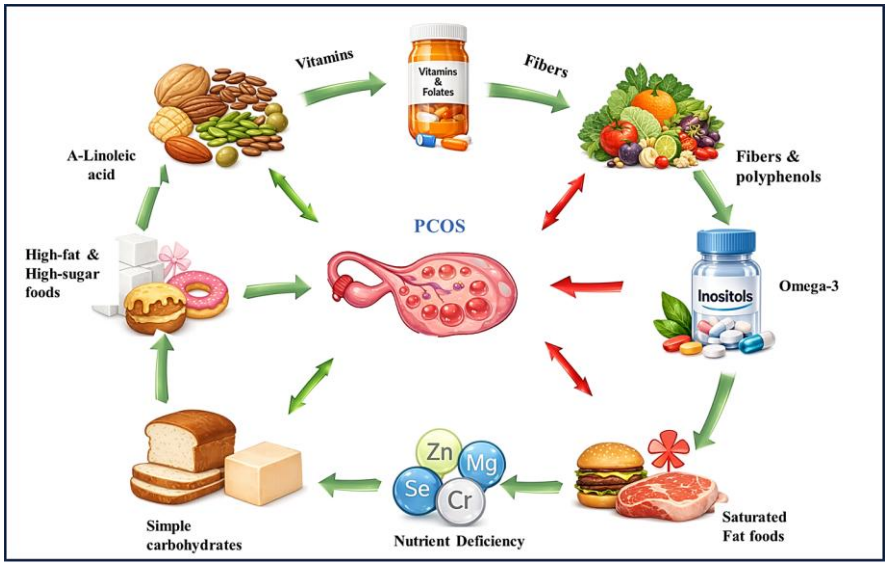

Figure 2: Dietary Components Influencing PCOS Clinical Features

Figure 2 shows the dietary components influencing PCOS clinical features where increased intake of vitamins, fibre, polyphenols, inositols, omega-3 fatty acids, complex carbohydrates and plant-based foods rich in α-linolenic acid may improve PCOS outcomes, whereas high consumption of saturated fats and refined carbohydrates may worsen metabolic and reproductive disturbances (41).

Glycemic Characteristics of Diet

PCOS has had a lot of research completed on it regarding nutritional aspects of carbohydrate quality because glucose control is so important to metabolic health. The glycemic quality of food refers to how a food affects the level of glucose in your blood after you eat it and whether that food influences how much insulin will be released from the pancreas. Because PCOS is characterized by high levels of insulin in the circulation due to

insulin resistance, there are even greater physiological ramifications of consuming a diet high in glycemic load foods (42). After eating high glycemic load foods, there are greater fluctuations in postprandial (after eating) glucose levels, leading to increased insulin production from the pancreas and thus more stress placed on the body. Repeatedly exposing the body to high levels of insulin results in an unstable metabolic state through many different mechanisms. High levels of insulin will cause the liver to store fat, increase the amount of fat stored in adipose tissue and disrupt insulin signaling pathways within the cell. In addition, when insulin is present, it will also act as a co-gonadotropin and promote the formation of androgens in the ovaries, further contributing to the dysregulation of the endocrine system. Therefore, a diet high in glycemic load may contribute to the metabolic and reproductive aspects of PCOS, illustrating that glycemic quality is a physiological mechanism contributing to PCOS rather than a peripheral component of nutrition (43).

Stabilizing glucose-insulin dynamics by reducing glycemic variability reduces the impact of hyperinsulinemia on the endocrine system. Additionally, eating foods with a low glycemic index allows for a slower absorption of glucose, which results in less insulin release in response to increases in blood sugar levels, thereby improving metabolic flexibility. Furthermore, the glycemic quality of the food consumed may have a greater impact on metabolism than just caloric restriction alone. Therefore, dietary practices that address only the energy content of the food consumed and ignore the glycemic quality will likely not yield optimal outcomes regarding metabolism (44). This highlights the need to evaluate the characteristics of a diet rather than solely the energy content of the diet. The glycemic stability of the food consumed likely has an influence on the reproductive system as well. Furthermore, excess androgens from insulin may lead to follicular arrest, anovulation and menstrual irregularities. Therefore, nutritional strategies that promote glycemic control may indirectly improve reproductive outcomes by preventing the amplification of the effects of the insulin hormone through the endocrine system. Thus, the glycemic component of food should be a primary target for treatment for women with PCOS (45).

Western Dietary Patterns

Western-style diets usually consist mainly of refined carbohydrates, ultra-processed foods, saturated fats and not enough dietary fibers and micronutrients (whole foods). These eating habits have a strong relationship with metabolic disturbances such as insulin resistance, dyslipidemia, systemic inflammation, oxidative stress and weight gain (adiposity). When it comes to PCOS, Western eating patterns can serve as significant environmental factors that may increase existing metabolic issues related to the disorder. Eating foods that are high glycemic (sugar) can create large spikes in blood glucose causing an increase in insulin secretion and a compensatory rise in insulin levels (hyperinsulinemia). In addition, high intakes of saturated fat create inflammatory mediators and negatively affects lipids, leading to metabolic instability (46).

Prolonged consumption of a western-style diet creates metabolic stress in several ways. High blood sugar and fat cause complications with how insulin works, lowers the metabolic flexibility of the body and allows for fat to accumulate in areas that are not intended (like the liver). These changes lead to increased oxidative stress and inflammation that further impair how insulin works. The cycle of insulin resistance and too much insulin led to negative endocrine effects associated with PCOS. Changes in the gut bacteria significantly impact how Western diets lead to metabolic problems with lower diversity and more inflammation-causing bacteria (47). These changes can cause systemic inflammation (through what is known as metabolic endotoxemia) and increase the risk of developing insulin resistance. In general, the effects of a western-style diet go beyond metabolic problems. Inflammation from the diet can negatively impact how the ovaries produce hormones, how steroids are made and how follicles develop. When inflammation persists, it can disrupt hormone levels leading to increased androgens and trouble with ovulation. The increase in cases of metabolic disease corresponds to the continued move towards eating a western diet pattern globally and indicates that the transition to a western diet pattern could contribute to both disease severity and the overall population level of expression of PCOS (48).

Mediterranean Dietary Patterns

The Mediterranean Diet is based on whole foods (minimally processed) like vegetables, fruits, beans/legumes, whole-grain cereals, nuts, seeds and olive oil as the primary source of dietary fat. The Mediterranean Diet doesn't just include individual food choices; rather, it provides a comprehensive dietary pattern with beneficial effects on metabolic and cardiovascular health. The Mediterranean Diet is gaining popularity in relation to PCOS, given its ability to influence several of the pathophysiologic mechanisms associated with PCOS (49).

The Mediterranean diet has strong anti-inflammatory effects so that it can be effective in managing PCOS. Women with PCOS often have chronic low-level inflammation which can lead to insulin resistance and hormonal dysregulation. The plant-based foods that are a large part of Mediterranean diet offer large amounts of bioactive compounds (polyphenols, flavonoids, carotenoids, etc.) that have both antioxidant and anti-inflammatory effects by reducing oxidative stress, suppressing inflammatory mediators and helping to maintain cellular metabolism in Figure 3.

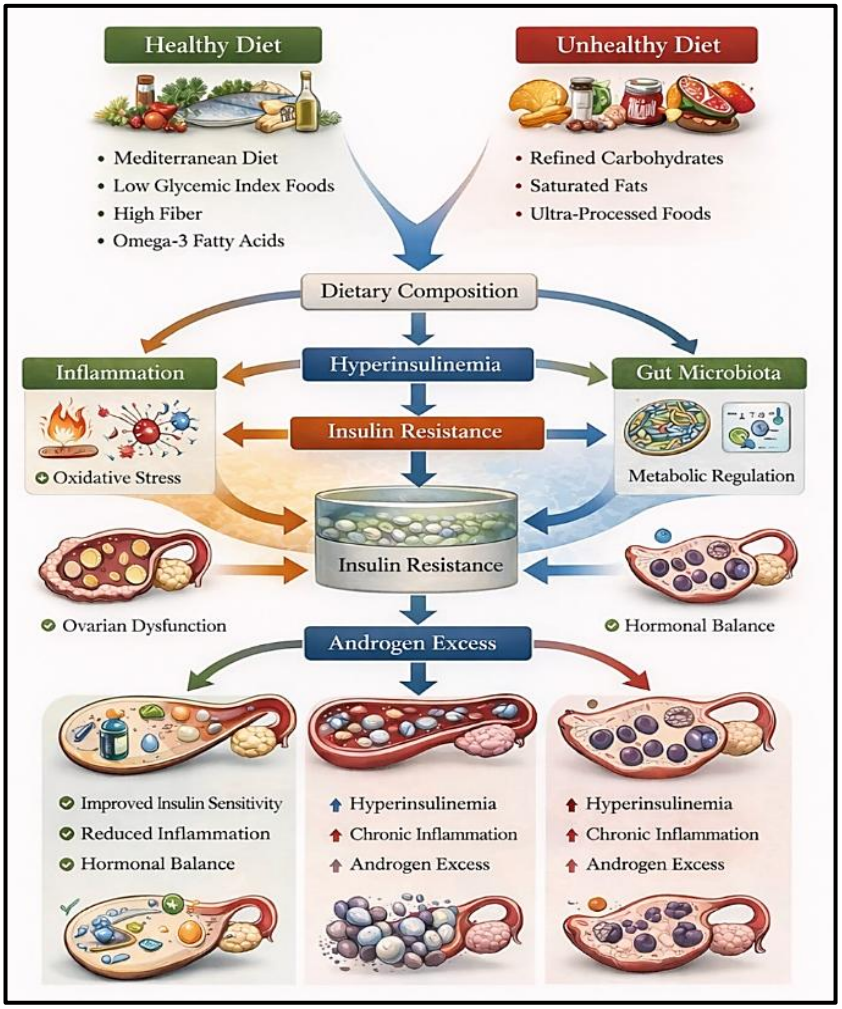

Figure 3: Mechanistic Interaction between Nutrition, Metabolic Dysfunction and Ovarian Physiology in PCOS

The anti-inflammatory effects that are inherent in the Mediterranean diet could also help improve insulin sensitivity and support hormonal balance by reducing inflammation (50). The glycemic profile of Mediterranean foods is also important for metabolic control. The high levels of dietary fiber in whole grains, legumes, fruits and vegetables in Mediterranean diets produce slow absorption rates for glucose, thereby stabilizing post-meal blood sugar levels. A more stable blood sugar level reduces excessive insulin secretion and helps to lower the levels of androgens produced from the ovary due to excess insulin. Increased levels of dietary fibre in the diet also support the

diversity of the gut microbiota and diversity of the gut microbiota appears to be an important factor in overall metabolic health (51, 52).

Although the lipid composition of the Mediterranean diet has also contributed significantly, the principal source of monounsaturated fats in the diet (such as those found in olive oil) improves metabolic flexibility and lipid metabolism. Monounsaturated fats, in contrast to saturated fats that induce inflammation and insulin resistance, provide metabolic protection. In particular, the metabolic response associated with these lipid-mediated phenomena is especially relevant for individuals with polycystic ovary syndrome, given that dyslipidemia and lack of metabolic flexibility is frequent among this population (53). Furthermore, it is important to recognize that the metabolic benefit of the Mediterranean diet may occur without any substantial weight loss. This finding supports the notion that quality of nutrition, as opposed to only a reduction in calories, determines metabolic outcome. Because nutritional approaches that focus on improving metabolic stability, decreasing inflammation and improving insulin sensitivity may offer a more physiologically sound treatment alternative for PCOS management, they warrant further consideration (54).

Macronutrient Influences

Macronutrients affect how our bodies maintain a stable internal environment (also known as metabolic homeostasis) and how our body responds to food. In patients with insulin resistance or abnormalities in hormone production and secretion (such as patients with Polycystic Ovary Syndrome), macronutrients provide energy as well as act as metabolic signals that can regulate hormones, inflammation and other processes within our cells. Optimal health and disease prevention depend on the balance and quality of carbohydrates, fats and proteins consumed and how these macronutrients interact to determine the metabolic response to food (55). The postprandial metabolic response is particularly important in women with PCOS because insulin sensitivity is often impaired in these patients. Specific dietary choices of macronutrients can directly alter glucose-insulin interaction, lipid metabolism, satiety and systemic levels of inflammation (56).

The quality of carbohydrates is an essential factor affecting one's ability to maintain metabolic stability. Refined carbohydrates are considered fast-digesting and absorbing foods, which results in large spikes in one's blood glucose level (glycemic excursions) and excessive amounts of insulin (hyperinsulinemia). Repeated exposure to high blood insulin levels may reduce insulin sensitivity over time and promote endocrine dysfunction caused by hyperinsulinemia. Complex carbohydrates with high fiber content help stabilize insulin secretion/make it easier for insulin to do its job by facilitating the slow absorption of glucose (57). This also applies to the impact of fiber on gut microflora and the regulation of inflammation. The composition of fats can also affect how they impact the body metabolically. Saturated (bad) fats create greater increases in the levels of inflammation, hinder/impaired insulin signaling and negatively impact lipid profiles versus unsaturated (good) fats (monounsaturated) and (polyunsaturated). Unsaturated fats improve lipid metabolism, enhance insulin sensitivity and are anti-inflammatory when consumed. Protein consumption contributes to the management of metabolism by promoting a feeling of fullness (satiation), stabilizing glycaemic response and supporting the preservation of lean mass (58). Adequate/proper amounts of protein consumed can decrease the total caloric intake and regulate blood glucose levels after eating. As such, interactions between macronutrients illustrate that the quality of food consumed and the balance of foods eaten play critical roles in the management of PCOS (59).

Micronutrients and Metabolic Regulation

Micronutrients are important co-factors biologically that control several key regulatory pathways including metabolic pathways, pathways related to enzyme function and pathways related to hormones. In addition to being important for regulating hormone levels in the body, micronutrient deficiencies are also often reported in women with PCOS. The deficiencies of certain vitamins and minerals (e.g., vitamins D, magnesium, zinc and antioxidants) may be associated with disease progression and metabolic stability; their absence can cause insulin resistance, oxidative stress, alterations in

inflammatory processes and hormonal imbalance (60). Micronutrients also play a crucial role in controlling insulin sensitivity, regulating oxidative stress and controlling immune system responses. The role of vitamin D in regulating serum calcium levels (is well-known among healthcare professionals) and in influencing the secretion of insulin, the uptake of glucose, the regulation of immunologic responses are all examples of how vitamin D can affect the body's biochemical milieu (the environment in which biochemistry occurs) (61).

Insulin resistance, systemic inflammation and reproductive dysfunction have all been shown to be associated with vitamin D deficiency. Some vitamins and minerals, especially magnesium, play a role in the metabolism of glucose and activity of insulin receptors, whereas Low magnesium levels also negatively affect insulin sensitivity and can lead to unequal insulin responsiveness for different types of carbohydrates (e.g., glucose, galactose, etc.). Zinc is also involved in antioxidant defence systems, inflammatory control and ovarian function thus, micronutrient deficiencies can contribute to oxidative stress, inflammation and metabolic dysfunction in patients with PCOS through their roles in protecting cells from damage caused by ROS (62). In addition, adequate intake of all micronutrients will aid in maintaining normal mitochondrial function, cellular signaling and hormone regulation. It is important for clinicians and researchers to recognize that the actions of all these micronutrients are often interdependent; therefore, dietary amount rather than supplementation solely should be taken into consideration when evaluating an individual's micronutrient status. These assessments should be made part of the overall metabolic-based dietary interventions (63).

Diet-Induced Inflammation

As a key factor in metabolic health, the inflammatory effects of the diet have begun to become well understood. It has been shown that dietary characteristics can greatly influence chronic low-level inflammation, for example, in polycystic ovarian syndrome (PCOS).

Inflammation is a result of a variety of factors including the production of cytokines by the immune system and the interplay of nutrition and metabolism (64). Foods that have a strong pro-inflammatory effect, such as those containing processed carbohydrates, Trans fats and saturated fats, can produce significant levels of inflammatory mediators and oxidative stress, impairing the body's ability to use insulin and regulate metabolism. In the case of PCOS, chronic inflammation contributes to insulin resistance and alters the function of adipose tissue and the production of ovarian steroid hormones (65).

Pro-inflammatory cytokines disrupt insulin receptor signaling pathways, thereby diminishing the efficiency of metabolism and promoting hyperinsulinemia. On the other hand, inflammatory prevention strategies include eating in a healthful, whole food manner, consuming plenty of fiber, including sources of polyunsaturated fats and incorporating foods that are high in plant-based, antioxidant-rich compounds. Following an anti-inflammatory eating pattern helps to reduce cytokine production, regulate insulin and glucose metabolism and stabilize one's metabolism. The understanding of how one's diet can alter the level of inflammation results in dietary intervention being recognized as a mechanism-based therapeutic approach (66). Consequently, focusing only on achieving a caloric balance may not be as effective for modulating immune-related metabolic processes and, instead, focusing on dietary quality should be an important factor in modifying the immuno-metabolic interactions. Therefore, the way one's diet influences inflammatory processes can potentially be an important mechanism involved in influencing both metabolic and reproductive outcomes for women who have PCOS (67).

Gut Microbiota and Metabolic Interactions

The gut microbiome is becoming recognized as an active and significant regulator of metabolic stability. Its influence is not only confined to gastrointestinal physiology, but rather extends to the regulation of glucose homeostasis, regulation of lipids, regulation of the immune system and the regulation of endocrine function. The gut microbiome continuously interacts with metabolic pathways of the host, thereby interacting with and affecting the balance of energy in the body and inflammatory responses. The diet is among the most potent modifiers of the diversity and composition of the gut microbiota and, therefore, modifies the functional activity of those microbes

(68). In patients with PCOS, there is emerging evidence that dysbiosis of gut microbiota may be contributing to the metabolic and endocrine disruptions that are common in this epidemiologic condition. Gut microbes modify host metabolism through many interconnected and complementary mechanisms. SCFAs, such as acetate, propionate and butyrate are produced by gut microbes and play a critical role in glucose regulation, insulin sensitivity and inflammation (69).

SCFAs (short-chain fatty acids) may be used as metabolic mediators that facilitate the improvement of insulin sensitivity, reinforce the intestinal barrier integrity and regulate the level of immune activation. In contrast, when there is a change in the microflora of the gut (i.e., dysbiosis), these beneficial metabolic interactions may be negatively affected. For example, changes in the gut flora can result in increased gastrointestinal permeability which allows the translocation of LPS into the bloodstream. This condition is termed metabolic endotoxemia and it initiates inflammatory pathways that disrupt insulin receptor signaling and increase insulin resistance (70). The type of diet consumed (dietary pattern) has a major impact on the stability of the gut microflora. Diets high in dietary fiber, prebiotics and colorfully sourced plant-based foods enhance the diversity of the gut microflora and stimulate the production of beneficial metabolites. Conversely, western style diets that are high in refined carbohydrates, have low total fiber intake and contain excessive amounts of saturated fat promote a dysbiotic state within the gut (71). This finding encourages us to think about how to use the gut microflora as an intermediary between the diet and metabolic regulation to develop new therapeutic options. Nutritional approaches that promote microbial stability could modulate the level of inflammation, enhance erythrocyte (RBC) sensitivity to insulin and contribute to the regulation of endocrine homeostasis and thus form a novel frontier of opportunity for management of PCOS (72).

Chrono Nutrition

Chrono nutrition is an emerging field of study within nutritional science that examines the timing of nutrient intake relative to the body's natural metabolic rhythms. Our bodies have an internal clock system (the circadian clock) that orchestrates our biological functions based on a 24-hour cycle (called the circadian rhythm) and our metabolic processes work in accordance with these cycles at two levels: at the hormonal level (i.e., insulin sensitivity, glucose tolerance) and at the cellular level (i.e., lipid metabolism, energy expenditure). Therefore, the timing at which we consume food can impact both how efficient we are metabolically (the number of calories we burn from food) and how much hormone we are able to produce in response to eating (hormonal response) (73). When we do not align our eating patterns properly (due to irregular eating patterns, late-night eating) with our internal circadian rhythm, this may lead to disrupted metabolic processes. Specifically, during PCOS, the high prevalence of insulin resistance/metabolic instability and associated sleep disturbances makes the study of chrono nutrition particularly salient.

Circadian rhythm disorders might worsen blood sugar fluctuations due to changes in insulin sensitivity and liver glucose control. Eating late at night may lead to much higher amounts of insulin being secreted after eating foods with high sugar content when the body is not burning food as efficiently. The repeated disruption to the circadian pattern of metabolism could create an environment conducive to ongoing excessive amounts of insulin being present, contributing to the metabolic hormone problems seen in women with PCOS. Timing meals in sync with the circadian rhythm, through regular eating patterns and distributing calories earlier in the day, could improve insulin sensitivity and decrease the potential for metabolic instability (74). By considering meal timing as a factor that influences metabolism, nutrition can be more than just looking at the types of nutrients consumed. Chrono nutrition strategies could potentially enhance the overall quality of the diet when combined with changes in dietary quality by stabilizing the relationship between glucose and insulin, minimizing metabolic stress and giving support to the hormonal balance within the body. Clinical data is still accumulating; however, chrono nutrition provides a theoretical basis for resolving metabolic dysfunction in women with PCOS (75).

Behavioural and Psychological Dimensions

Metabolic regulation in the context of PCOS is affected by both psychological and behavioural

factors. Psychological factors such as stress, anxiety and depression can alter how and why women with PCOS eat, thus impacting energy balance as well as endocrine activity. Many women with PCOS are more likely to have depression and anxiety than healthy women, which may impact their eating behaviours due to altered neuroendocrine functioning. More specifically, activation of the hypothalamic-pituitary-adrenal (HPA) axis by stress can lead to an increase in cortisol level, which in turn, can impact appetite and how glucose is metabolized thus, altering how fat is distributed throughout the body (76). The use of emotional eating (consuming palatable, energy-dense food in response to a psychological stressor) has the potential to have adverse effects on metabolic stability by promoting the consumption of high glycaemic index (GI), high-fat diet thus, leading to increased insulin levels and inflammation. The influence of psychological factors on eating behaviours and the relationship between diet and metabolic function suggests that stress may indirectly increase both insulin resistance and hormone dysregulation through maladaptive eating patterns. Additionally, psychological factors may affect adherence to dietary interventions; therefore, the need for integrated strategies for management is critical (77). The behavioural dimension shows that psychological health must be addressed in conjunction with nutritional and metabolic regulation. By utilizing a multi-disciplinary approach to management that utilizes effective stress management techniques, behavioural counselling and nutritional education, it is likely that individuals with PCOS will be able to achieve long-term metabolic regulation (78).

Clinical Evidence

A paradigm shift in dietary intervention strategies is needed due to the mechanistic relationship that exists between the diet, metabolism and endocrine regulation in the context of PCOS in Table 2. Emphasis should not be placed solely on achieving weight loss; while a weight-loss goal still has therapeutic relevance, evidence supports that metabolic control, glycemic control, inflammatory control and hormone regulation are also important therapeutic targets. The quality of the diet and the composition of macronutrients significantly impact both insulin signaling and lipid metabolism as well as positively impacting the function of the endocrine system even when weight loss is not achieved (79). The results of numerous controlled trials and structured dietary changes demonstrate that the Mediterranean-style diet (which includes high levels of monounsaturated fatty acids, omega 3 fatty acids, dietary fiber and polyphenols from plants) significantly affects insulin sensitivity, reduces HOMA-IR, reduces triglycerides and reduces the expression of systemic inflammatory markers. Similarly, low glycemic index dietary patterns will reduce postprandial glucose variation and hyperinsulinemia, which is the primary contributor to excess ovarian androgen production (80). These patterns lead to improvements in circulating total testosterone, increases in sex hormone binding globulin (SHBG), improved menstrual cycles and increased please refer to ovulation rates for individuals with insulin resistance. Diets greater in unsaturated fats and containing antioxidant bioactives also appear to reduce C-reactive protein and oxidative stress levels which may aid in maintaining the integrity of ovarian microenvironments. The above mentions many potential forms of personalized and sustainable nutritional programs to achieve stable glucose and insulin levels as well as control inflammation. By the framing of dietary interventions as a form of metabolic regulatory treatment opposed to simply a lifestyle change; there can be improvement in adherence, patient engagement and long-term clinical results when treating PCOS (81).

In Table 2, dietary interventions such as low-glycemic index and Mediterranean diets have demonstrated beneficial effects on metabolic parameters, including improved insulin sensitivity and reduced fasting glucose levels. These dietary strategies also contribute to reproductive and endocrine improvements, such as reduced androgen levels, restoration of menstrual regularity and improved ovulation. The outcomes summarized in this table highlight the potential role of dietary modifications as supportive therapeutic approaches in the management of PCOS. Macronutrient composition significantly influences metabolic homeostasis and endocrine regulation in women with PCOS. The glycemic characteristics of dietary carbohydrates play an essential role in determining insulin responses and metabolic stability. Diets with a high glycemic index led to rapid postprandial glucose

fluctuations and exaggerated insulin secretion, which may exacerbate insulin resistance and hyperinsulinemia. Clinical studies have demonstrated that low-glycemic index diets improve insulin sensitivity, reduce fasting insulin levels and enhance menstrual regularity in women with PCOS (4, 21, 44).

Table 2: Clinical Evidence for Dietary Intervention in PCOS

Study Design	Dietary Intervention	Duration	Metabolic Outcomes	Reproductive/Endocrine Outcomes	References
RCT	Low-Glycemic Index Diet	Variable	Improved HOMA-IR, reduced fasting insulin	Improved menstrual regularity and ovulation	(82)
RCT	Mediterranean/Low-Carbohydrate Diet	~8–12 weeks	↓ Fasting glucose, improved insulin sensitivity	↓ Testosterone restored menstrual cycle	(83)
Observational	Mediterranean Diet Adherence	Cross-sectional	↓ IR, inverse association with inflammation	Lower androgen levels	(84)
Pilot RCT	Mediterranean Diet vs Healthy Eating	12 weeks	Trend toward improved insulin sensitivity	Feasible, hormonal trend improvements	(85)

Notes: Upward arrows (↑) indicate an increase, while downward arrows (↓) indicate a decrease in the respective biological processes

Dietary fat composition also affects inflammatory signaling and metabolic pathways associated with PCOS. Saturated and trans fats promote inflammatory responses and impair insulin signaling pathways, whereas monounsaturated and polyunsaturated fatty acids exhibit anti-inflammatory effects and improve lipid metabolism (13, 46). Omega-3 fatty acids have been shown to reduce inflammatory cytokines and improve metabolic markers in individuals with metabolic disorders. Protein intake contributes to metabolic regulation by improving satiety and stabilizing postprandial glucose responses. Diets with adequate protein content may enhance glycemic control and support lean body mass maintenance. Evidence suggests that balanced macronutrient distribution combined with improved carbohydrate quality may significantly improve metabolic outcomes in women with PCOS (55–57).

Therapeutic Strategies

The translational and therapeutic implications of nutritional regulation in polycystic ovary syndrome (PCOS) highlight diet as a practical and modifiable strategy for improving both metabolic and reproductive outcomes. Evidence from clinical and mechanistic studies indicates that dietary interventions targeting insulin resistance, inflammation and oxidative stress can significantly influence disease progression. Diets emphasizing low-glycemic index carbohydrates help stabilize postprandial glucose levels and reduce hyperinsulinemia, thereby decreasing ovarian androgen production and improving menstrual regularity and ovulatory function. Similarly, Mediterranean-style dietary patterns rich in whole grains, fruits, vegetables, legumes, nuts, olive oil and omega-3 fatty acids exert anti-inflammatory and antioxidant effects that enhance insulin sensitivity and metabolic flexibility.

Increasing dietary fiber intake supports gut microbiota diversity and improves glucose regulation, while limiting refined carbohydrates, ultra-processed foods and saturated fats helps prevent metabolic inflammation and endocrine disruption. Adequate intake of micronutrients such as vitamin D, magnesium and zinc further contributes to improved insulin signaling and hormonal balance. From a translational perspective, these findings suggest that dietary counseling for PCOS patients should focus on improving carbohydrate quality, increasing plant-based foods, incorporating unsaturated fats and maintaining regular meal timing to stabilize circadian metabolic rhythms. Integrating these dietary strategies into clinical management may provide a sustainable therapeutic approach that complements pharmacological treatments and

promotes long-term metabolic and reproductive health in women with PCOS (4, 41).

Limitations

Despite increasing research on dietary interventions in PCOS, several limitations exist within the current body of literature. Many studies involve relatively small sample sizes and short intervention durations, which restrict the ability to evaluate long-term metabolic and reproductive outcomes. Additionally, considerable heterogeneity exists in study design, dietary assessment methods and intervention protocols. Another major limitation is the heterogeneity of PCOS phenotypes. Women with PCOS may exhibit significant variability in metabolic characteristics, including differences in insulin sensitivity, body composition and hormonal profiles. These variations may influence individual responses to dietary interventions. Furthermore, dietary intake in many studies is assessed using self-reported questionnaires, which may introduce measurement bias. Future research should incorporate standardized dietary assessment methods, larger sample sizes and long-term follow-up studies to better understand the sustained effects of nutritional interventions on PCOS pathophysiology (21, 46).

Future Directions

Although nutrition has a clearly defined mechanistic role in PCOS, there are still major gaps in our current understanding of this field. Future research should focus on the following areas: long-term studies of the effects of dietary intervention on PCOS; investigating mechanisms of hormone-diet interactions; how diet affects gut microbiota; how chrono-nutrition may influence PCOS; how to develop precision-nutrition (i.e., tailoring dietary recommendations for each patient) approaches to the treatment of PCOS. Due to the heterogeneous nature of PCOS, research designs must be able to identify phenotype-specific responses to dietary interventions therefore stratified research designs will be required. Tailored precision-nutrition interventions can be designed by using integrated metabolic profiles; genetic predisposition; hormonal profiles and behaviours of each patient. Advances in the following fields will help us develop targeted nutritional approaches: metabolomics; microbiome sciences and digital health technology. Moving from conventional caloric-based nutrition research outcomes to a focus on mechanistic and metabolic regulation will be an important step in further developing and improving treatment of PCOS.

Conclusion

Dietary patterns act as important biological regulators influencing both metabolic and reproductive dysfunction in women with polycystic ovary syndrome (PCOS). Growing evidence indicates that nutritional factors modulate several key physiological pathways, including insulin signaling, inflammatory responses, oxidative stress, adipose tissue metabolism and gut microbiota composition, all of which contribute to the development and progression of PCOS. These interconnected mechanisms emphasize that overall dietary quality and long-term dietary patterns are more critical than the intake of isolated nutrients in managing the disorder. Clinical studies have shown that dietary strategies such as low-glycemic index diets, Mediterranean-style diets and nutrient-dense plant-based dietary patterns can improve insulin sensitivity, reduce hyperinsulinemia, lower androgen levels and help restore hormonal balance, thereby supporting improved ovulatory function and reproductive outcomes. In addition, adequate intake of dietary fiber, antioxidants, omega-3 fatty acids and essential micronutrients may reduce oxidative stress, regulate inflammatory pathways and promote a healthier gut microbiota, further contributing to metabolic and endocrine stability in PCOS. Nevertheless, current evidence is limited by heterogeneity in study populations, variations in dietary interventions and relatively short study durations, which restrict the ability to establish standardized nutritional recommendations. Future research should therefore focus on well-designed long-term clinical trials and personalized nutrition approaches that consider individual metabolic, hormonal and genetic profiles. A deeper mechanistic understanding of the complex interactions between diet, metabolism and ovarian physiology may facilitate the development of targeted nutritional strategies that can effectively improve metabolic health, reproductive function and overall quality of life in women with PCOS.

Abbreviations

GI: Glycemic Index, GL: Glycemic Load, HOMA-IR: Homeostatic Model Assessment of Insulin Resistance, IR: Insulin Resistance, LH: Luteinizing Hormone, MUFA: Monounsaturated Fatty Acids, PCOS: Polycystic Ovary Syndrome, PUFA: Polyunsaturated Fatty Acids, ROS: Reactive Oxygen Species, SHBG: Sex Hormone-Binding Globulin.

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Author Contributions

Mohd Noorjahan Begum: review and editing, writing original draft, Madhan Krishnan: writing original draft, conceptualization, Lalithamma A: writing review and editing, formal analysis, Vandali Jyothi: writing review and editing, formal analysis.

Conflict of Interest

The authors declare no conflicts of interest and assume sole responsibility for both the content and the writing of the article.

Data Availability

This manuscript is a review article, so there is no new data were generated or analyzed during this study; therefore, data availability is not applicable to this manuscript.

Declaration Of Generative AI And AI Assisted Technologies in the Writing Process

This manuscript was written by the authors without the use of generative AI or AI-assisted technologies. All content is original and has been created by the authors themselves.

Ethics Approval

Ethical approval was not required for this study as it is a review article based exclusively on previously published literature and does not involve human participants, animals, or any primary data collection.

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