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THE ROLE OF GUT MICROBIOTA IN THE PATHOGENESIS OF POLYCYSTIC OVARY SYNDROME

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ABSTRACT

Introduction and aim of the study: Polycystic ovary syndrome (PCOS) is a multifactorial endocrine–metabolic condition affecting a significant proportion of women of reproductive age. Recent scientific evidence suggests that disturbances in gut microbiota composition may play an important role in the metabolic, hormonal and inflammatory abnormalities characteristic of PCOS. The aim of this review was to summarize current knowledge on the relationship between gut microbiota and PCOS, with particular emphasis on pathophysiological mechanisms and potential therapeutic implications.
Materials and Methods: This review was conducted through a systematic search of the PubMed database, using keywords related to PCOS, gut microbiota, dysbiosis, insulin resistance and hormonal imbalance. Relevant original research articles were analyzed to identify common mechanisms and clinically meaningful associations.
Results: Findings indicate that gut dysbiosis in PCOS is closely linked to increased intestinal permeability, chronic low-grade inflammation, altered short-chain fatty acid (SCFA) production and disturbances in bile acid and tryptophan metabolism. These changes interact with key endocrine pathways regulating insulin sensitivity, androgen excess and estrogen conversion. Recent studies also highlight the therapeutic potential of dietary interventions, probiotics, prebiotics, synbiotics and bioactive nutrients, which can help restore microbial balance and improve metabolic and hormonal outcomes.
Conclusions: Understanding the role of the gut microbiota in PCOS pathogenesis opens new perspectives for adjunctive therapeutic strategies. Microbiota-targeted interventions may provide meaningful support alongside conventional treatment, helping to address underlying mechanisms rather than only managing symptoms.

KEYWORDS

Polycystic Ovary Syndrome, Gut Microbiota, Dysbiosis, Insulin Resistance, Hormonal Imbalance, Hyperandrogenism

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Introduction

Polycystic ovary syndrome (PCOS) is one of the most prevalent endocrine and metabolic disorders affecting women of reproductive age. The global prevalence of PCOS is estimated to range between 5% and 18%, depending on diagnostic criteria and study population, while in overweight adolescents it may reach as high as 18–26% [2].

PCOS is primarily characterized by excessive androgen secretion, ovulatory dysfunction, and the presence of polycystic ovarian morphology. It is frequently accompanied by abdominal obesity, insulin resistance, impaired glucose metabolism, and dyslipidemia. Excessive consumption of processed foods and simple carbohydrates can lead to fluctuations in blood glucose and insulin levels, disrupting normal endocrine regulation and increasing the risk of developing PCOS [3].

At the pathophysiological level, hormonal dysregulation plays a central role in the development of PCOS and its associated metabolic complications. Alterations have been observed in insulin secretion and sensitivity, growth hormone (GH), ghrelin, liver-expressed antimicrobial peptide 2 (LEAP-2), and gonadotropin-releasing hormone (GnRH) activity, as well as in the luteinizing hormone (LH) to follicle-stimulating hormone (FSH) ratio and in androgen and estrogen concentrations. These disturbances are strongly associated with metabolic abnormalities such as insulin resistance, obesity, type 2 diabetes, menstrual irregularities, and infertility. Elevated insulin levels, decreased GH, increased ghrelin, and leptin resistance contribute to obesity and glucose intolerance, whereas reduced GH and estrogen levels, combined with elevated LEAP-2, LH, androgens, and LH/FSH ratio, are linked to impaired fertility in women with PCOS [1].

These complex hormonal and metabolic imbalances indicate that PCOS is not solely an endocrine disorder but a multifactorial condition involving numerous physiological systems. Recent research highlights the potential role of the gut microbiota in modulating metabolic and hormonal pathways related to PCOS. Understanding the bidirectional relationship between gut dysbiosis and the endocrine–metabolic features of PCOS may provide new insights into its pathogenesis and support the development of novel therapeutic strategies.

Methodology

This review was conducted systematically to identify and analyse relevant scientific literature on the role of gut microbiota in the pathogenesis of polycystic ovary syndrome (PCOS), with particular emphasis on hormonal, metabolic and immunological mechanisms and what benefits may result from modifying it through diet and other interventions.The PubMed database was searched. Articles were searched using the following words: „Polycystic Ovary Syndrome'' „Gut Microbiota'' „Dysbiosis" „Insulin Resistance" „Hormonal Imbalance" „Hyperandrogenism".

Results

1.Gut Microbiota and Endocrine–Metabolic Function

The intestinal microbiota plays an important role in maintaining the body's homeostasis, influencing the intestinal environment as well as the functioning of distant organs and numerous metabolic processes. Increasingly, it is seen as a separate organ with endocrine properties. Recent research findings indicate that the microorganisms inhabiting the gut are actively involved in shaping and maintaining the normal physiological processes of the host. The composition and diversity of the microbiota is influenced by a number of internal and external factors, including diet, genetic predisposition and hormonal background. Commensal gut bacteria have the ability to produce and secrete hormones, allowing them to influence the metabolism, immunity and behaviour of the host. The microbiome participates in the regulation of the female endocrine system, influencing levels of oestrogens, androgens and insulin, among others. They may be involved in various stages of female reproduction, and disruptions in their composition may contribute to abnormal functioning of the endocrine axis of the female reproductive system. There is also growing evidence of a potential link between specific species of gut bacteria and diseases of the female reproductive system, such as polycystic ovary syndrome (PCOS), endometriosis or bacterial vaginitis (BV). Research suggests that improving the composition and quality of the gut microbiota may benefit reproductive outcomes in women [4].

2. Hyperandrogenism and Gut Microbiota

Hyperandrogenism, defined as excessive androgen levels in women, is a typical clinical feature of patients with polycystic ovary syndrome. Its typical symptoms include excessive male pattern hair on the face, chest, abdomen, acne, seborrhoea, male pattern baldness, menstrual disorders and infertility. Studies show its relationship with the gut microbiota.

Comparative studies have shown that the composition of the gut microbiota in women with polycystic ovary syndrome (PCOS) varies according to testosterone levels. Patients with higher androgen levels (PCOS-HT) show greater abnormalities in the structure of the microbiome than women with lower testosterone levels (PCOS-LT) or controls. In this group, a decrease in the abundance of bacteria from the genera Bifidobacterium, Faecalibacterium and Eubacterium, among others, was found, while Prevotella and Ruminococcus increased. The results suggest that the composition of the microbiota may be a potential biomarker to not only identify patients with PCOS, but also to differentiate its subtypes[5].

Elevated testosterone levels, correlate with increased activity of inflammatory processes. It has been shown that lipopolysaccharide (LPS), a pro-inflammatory cytokine of bacterial origin, can stimulate the release of inflammatory mediators such as interleukin 6 (IL-6). Testosterone, on the other hand, can potentiate IL-6 production in fat cells, enhancing the inflammatory response. [6]

Furthermore, the gut microflora may encode specific enzymes, such as 17-hydroxysteroid dehydrogenase and 3-β-hydroxysteroid dehydrogenase, which are involved in the regulation of testosterone levels. This highlights the role of the gut microflora as a key mediator in the intervention of PCOS.

Testosterone levels significantly affect the composition of the gut microbiota in patients with PCOS, leading to distinct microbial profiles that can potentially inform diagnostic and therapeutic strategies. The interplay between testosterone, gut microflora and inflammatory pathways highlights the complexity of PCOS pathogenesis.

3. Estrogen-Microbiota Axis

A growing body of research indicates that oestrogens and the gut microbiota are involved in a dynamic, bidirectional interaction that plays a key role in the pathophysiology of polycystic ovary syndrome (PCOS). The gut microbiome participates in oestrogen metabolism mainly through the activity of bacterial β-glucuronidase, which enables the conversion of conjugated forms of hormones into their biologically active counterparts, which can re-enter the bloodstream [13,23]. At the same time, fluctuations in sex hormone levels,

such as oestradiol and progesterone, affect microbial diversity and the proportions between the main types of gut bacteria, as confirmed by both population studies and animal models [13,25]. In this context, the concept of the estrobolome, i.e. the set of bacterial genes responsible for oestrogen metabolism, emerges as one of the key elements regulating hormonal balance in women [14,22].

Intestinal dysbiosis, especially that associated with reduced microbial diversity, can lead to decreased β-glucuronidase activity and thus to reduced estrogen deconjugation and decreased levels of circulating active forms of the hormone [22,23]. This disrupts the normal activation of ERα and ERβ oestrogen receptors, which in turn can affect metabolic function, adipogenesis regulation, and reproductive processes such as follicular maturation and ovulation [13].

In PCOS, gut microbiota disorders also exacerbate chronic, low-grade inflammation, which is a key element in the pathophysiology of the disease. Dysbiosis leads to increased intestinal barrier permeability, allowing lipopolysaccharides (LPS) to enter the bloodstream. LPS activates the immune system via Toll-like receptors (TLR4), triggering the production of pro-inflammatory cytokines such as TNF-α, IL-6 and IL-1β [10,11]. These cytokines disrupt insulin signalling in peripheral tissues, contributing to insulin resistance, exacerbating hyperandrogenism and disrupting lipid metabolism [10,11,24]. Activation of NF-κB and JNK pathways further increases oxidative stress and worsens insulin sensitivity, which may exacerbate metabolic and reproductive symptoms in PCOS [10,24].

Improving the composition of the gut microbiota through a diet rich in fibre, prebiotics, probiotics and lifestyle changes has been shown to reduce inflammatory markers (IL-6, TNF-α), increase β-glucuronidase activity and restore hormonal balance [1,6,7]. Thus, microbiota modulation can not only affect oestrogen metabolism, but also limit pro-inflammatory metabolic pathways that exacerbate hormonal and metabolic disorders in PCOS, making it a promising therapeutic target.

4. Insulin Resistance and Gut Microbiota

Insulin resistance (IR) is one of the most common metabolic abnormalities in women with polycystic ovary syndrome (PCOS) and plays a central role in its pathophysiology. Recent research has shown that intestinal microbiota composition and function are closely linked to glucose metabolism, inflammation, and insulin sensitivity [8]. Women with PCOS often display gut dysbiosis characterized by decreased microbial diversity and altered proportions of Firmicutes and Bacteroidetes, which may disrupt intestinal barrier integrity and promote systemic inflammation [9,10].

One of the major mechanisms connecting gut dysbiosis with impaired insulin action is increased intestinal permeability. Translocation of lipopolysaccharides (LPS) from Gram-negative bacteria into systemic circulation triggers chronic low-grade inflammation via cytokines such as TNF-α and IL-6, which interfere with insulin receptor signaling and glucose uptake [11]. Dysbiosis also influences the availability of short-chain fatty acids (SCFAs), especially butyrate, essential for maintaining epithelial barrier integrity and exerting anti-inflammatory effects [12]. Reduced SCFA production weakens gut barrier function, enhances metabolic endotoxemia, and contributes to worsening insulin sensitivity.

Beyond SCFAs, several other metabolites produced by the gut microbiota play a crucial role in glucose and hormonal regulation. Altered production of secondary bile acids, shaped by the microbial community, affects receptors such as FXR and TGR5 which are key regulators of lipid and glucose metabolism. Studies using PCOS models have shown that disturbances in bile acid composition contribute to insulin resistance and ovarian dysfunction [7]. Additionally, dysbiosis disrupts tryptophan metabolism, reducing levels of indole derivatives that activate the AhR receptor, which is involved in preserving mucosal immunity and stimulating GLP-1 secretion [20,21]. Lower GLP-1 levels further impair insulin sensitivity and metabolic balance. Together, these metabolic alterations, including decreased SCFAs, modified bile acids, and reduced indole metabolites, exacerbate systemic inflammation and worsen IR in PCOS [20].

Animal and human studies demonstrate that modifying gut microbiota composition can meaningfully improve metabolic outcomes. Supplementation with specific probiotics, particularly Bifidobacterium and Lactobacillus strains, has been associated with increased insulin sensitivity, reduced fasting insulin levels, and lower HOMA-IR scores [8]. Interventions aimed at restoring microbial balance such as diets high in fermentable fiber, prebiotics, probiotics, and targeted lifestyle modifications represent a promising therapeutic strategy to alleviate insulin resistance and metabolic dysfunction in PCOS.

5. Dietary and Microbiota-Targeted Therapies

In recent years, dietary and microbiological interventions that can modulate the composition and activity of the gut microbiota have been gaining importance in the context of polycystic ovary syndrome (PCOS). Women with PCOS have reduced species diversity of gut bacteria, an increased Firmicutes/Bacteroidetes ratio and increased inflammation, which may promote the development of insulin resistance and hormonal disorders. A balanced diet rich in nutrients that support healthy gut microflora can reverse these changes and contribute to improved metabolic and hormonal function [15].

Consuming fibre-rich foods such as vegetables, fruit, whole grains and legumes promotes the growth of beneficial bacteria of the Lactobacillus and Bifidobacterium genera. These microorganisms produce short-chain fatty acids (SCFAs) such as butyrate, propionate and acetate, which strengthen the intestinal barrier, reduce inflammation and increase tissue sensitivity to insulin. In contrast, a diet rich in saturated fats and simple sugars can lead to dysbiosis, increased intestinal permeability and translocation of lipopolysaccharides (LPS) into the bloodstream, exacerbating the chronic inflammation characteristic of PCOS [16].

Beyond fiber and classic probiotics, dietary polyphenols and postbiotics have also been shown to beneficially modulate the gut microbiota and metabolic outcomes in PCOS. Polyphenol-rich foods, such as berries, green tea, cocoa and olive oil, can increase the abundance of beneficial bacteria like *Faecalibacterium prausnitzii* and *Roseburia spp.*, which produce SCFAs and exert anti-inflammatory effects [26,27]. These microbial metabolites not only support intestinal barrier integrity but also regulate glucose and lipid metabolism, improve insulin sensitivity and reduce circulating androgen levels. Furthermore, microbial modification of bile acids by species such as *Bacteroides* and *Clostridium* activates nuclear receptors FXR and TGR5, influencing energy balance, lipid metabolism, and systemic inflammation- processes closely linked to the metabolic and hormonal disturbances observed in PCOS [11,28]. Postbiotics, including SCFA-enriched preparations, further contribute to these effects by enhancing insulin sensitivity, limiting LPS-mediated endotoxemia and modulating inflammatory signaling [29].

Recent clinical and experimental studies further highlight that specific dietary components can directly modulate gut microbiota and improve metabolic outcomes in women with PCOS. A randomized controlled trial demonstrated that supplementation with inulin-type fructans significantly increased Bifidobacterium abundance and enhanced butyrate production, leading to reductions in fasting insulin, HOMA-IR, and inflammatory markers [17,30]. Additionally, omega-3 fatty acids have been shown to improve microbial diversity and lower circulating lipopolysaccharide (LPS) levels, resulting in improved insulin sensitivity and reduced testosterone concentrations [31]. Similarly, resistant starch supplementation promotes the growth of SCFA-producing bacteria, increases GLP-1 secretion, and supports glucose homeostasis, indicating therapeutic potential in metabolic disturbances characteristic of PCOS [32]. These findings demonstrate that beyond general dietary fiber, targeted nutritional components can meaningfully influence microbial metabolic pathways and contribute to improved hormonal and metabolic profiles in PCOS.

A study conducted among overweight women with PCOS showed that six months of synbiotic supplementation, combined with lifestyle modifications (increased physical activity and a low glycaemic index diet), resulted in weight loss and reduced levels of testosterone, LPS and LPS-binding protein (LBP). The improvement in intestinal barrier integrity and reduction in endotoxemia indicate that the gut microbiota may mediate the therapeutic effect of this type of intervention [18].

Similar conclusions were obtained when comparing the efficacy of probiotics and metformin. Women using probiotics showed improved metabolic and hormonal parameters, as well as an increase in the abundance of Akkermansia muciniphila bacteria associated with better glycaemic control and intestinal barrier integrity [19].

The collected data indicate that a diet rich in fibre, with limited processed foods, combined with targeted probiotic or synbiotic supplementation, may be an effective component of PCOS supportive therapy. Modulation of the gut microbiota not only affects metabolism and hormone balance, but also reduces chronic inflammation, thus representing a promising strategy to support the treatment of women with PCOS.

Discussion and Conclusions

Current evidence demonstrates that the gut microbiota plays a central role in the hormonal, metabolic and immunological disturbances characteristic of PCOS. Dysbiosis contributes to increased intestinal permeability, endotoxemia and chronic low-grade inflammation, processes closely linked to insulin resistance and hyperandrogenism [7,10]. Altered microbial composition also affects estrogen and androgen metabolism through decreased β-glucuronidase activity and dysregulation of microbial steroid-transforming enzymes, worsening reproductive and metabolic dysfunctions [13,22].

Furthermore, disturbances in SCFA production, bile acid metabolism and tryptophan-derived indole pathways contribute to impaired GLP-1 secretion, oxidative stress and altered lipid and glucose metabolism, all of which exacerbate PCOS pathophysiology [7,20].

A growing body of clinical evidence supports the therapeutic potential of dietary and microbiota-targeted interventions. Diets rich in fibre, polyphenols and resistant starch, as well as supplementation with probiotics, prebiotics and synbiotics, have been shown to improve insulin sensitivity, reduce inflammatory cytokines and lower circulating androgen levels [30,31,32]. These interventions additionally help restore microbial diversity, enhance SCFA production, stabilise the intestinal barrier and decrease metabolic endotoxemia [11, 29].

Although further high-quality clinical trials are required to standardise optimal therapeutic protocols, including strain-specific probiotic combinations, dietary recommendations and long-term intervention models, the modulation of gut microbiota emerges as a promising and biologically grounded strategy. Incorporating microbiome-targeted approaches alongside conventional treatment could significantly enhance the management of PCOS by addressing its endocrine–metabolic root causes, not merely its clinical manifestations.

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