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Reviewer Paper

Gut Microbiota Dysbiosis and Insulin Resistance in Polycystic Ovary Syndrome: Pathophysiological Links and Therapeutic Perspectives

[1]Omaima Saad Al-Qaissy, [2]Rabiah Muayad Sabri Shawkat, [3]Marwa Jewi, [4]Ibrahim M Kamal, [5]Baker jamal
Department of Medical Instrumentation Engineering Techniques / Electrical Engineering Technical College / Middle Technical University
[2]Department of cancer, Experimental Treatment,Mustansiriyah University, Iraqi Center for Cancer and Medical Genetic Research, Baghdad, Iraq.
[3]College of basic education, Al-Mustansiriya University, Baghdad, Iraq.
[4]Department of pathology, College of Medicine, University of Baghdad, Iraq.
[5]Rudn University, College of chemistry.

Correspondence email | omaima.saad@mtu.edu.iq
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ABSTRACT

Polycystic ovary syndrome (PCOS) affects approximately 8–13% of women of reproductive age and is one of the most prevalent endocrine and metabolic conditions in women of reproductive age, and is commonly characterized by insulin resistance, hyperandrogenism and reproductive dysfunction. Recent studies have provided increasing evidence regarding the involvement of gut microbiota alterations in the pathogenesis of PCOS, acting via intricate metabolic, endocrine, inflammatory and immune pathways. Cellular changes in the gut associated with these factors such as an increase in intestinal permeability, inflammation due to lipopolysaccharide, changes in the production of short-chain fatty acids, and bile acid metabolism have all been proposed as mechanisms by which gut dysbiosis contributes to the development of insulin resistance. This narrative review aims to provide a synthesis of the available evidence and make a critical appraisal of the link between gut microbiota dysbiosis and insulin resistance in PCOS. A literature search was conducted in PubMed, Scopus, and Google Scholar from August 2025 to January 2026, including studies published between 2015 and 2025, using the keywords such as “PCOS”, “gut microbiota”, “gut microbiome”, “dysbiosis”, and “insulin resistance”. Special focus was given to the pathophysiological mechanisms linking alterations of the gut microbiome to metabolic dysfunction, the role of host–microbiome interactions, and novel therapeutic strategies based on the gut microbiome. The evidence reviewed here showed a close relationship between insulin resistance and gut dysbiosis, and gut dysbiosis may play a role in the metabolic and reproductive symptoms of PCOS. Other microbiome-targeting strategies like probiotics, prebiotics, synbiotics, and metformin have shown promise in enhancing insulin sensitivity, inflammatory markers, hormonal regulation, and metabolic health. Other novel methods, such as postbiotics and FMT are promising, but clinical evidence is limited.

KEYWORDS: *Polycystic ovary syndrome; Gut microbiota; Dysbiosis, Insulin resistance; Probiotics; Hyperandrogenism; Microbiome*

1. INTRODUCTION

Polycystic ovary syndrome (PCOS) is among the most prevalent endocrine disorders in women of reproductive age, with an estimated global prevalence of 8-13% (1,2). Its clinical presentation encompasses hyperandrogenism, oligo-anovulation and ovulatory abnormalities, polycystic ovarian morphology; however, the metabolic dimension of the syndrome—particularly insulin resistance (IR)—is increasingly recognized as a central pathological driver that substantially elevates cardiometabolic risk (3,31). Recent studies have shifted focus toward the hypothesis that such derangements of metabolism can be triggered by gut microbiota dysbiosis through immune, endocrine, and inflammatory pathways (4). The intestinal microbiota is a multifaceted community of microorganisms that regulates host metabolism and immunity. It is known that an endocrine axis that involves the gut microbiota, central nervous system, and insulin exists, which contributes to the critical role of microbiota in the metabolic and hormonal regulation (29) (Figure1). The underlying mechanisms linking gut dysbiosis to the development, progression and metabolic dysfunction of PCOS remain incompletely understood, and further large-scale studies are needed to establish their clinical relevance (5,28). Considering such pathophysiological knowledge, a

holistic perspective on the prevalence and clinical presentation of PCOS is necessary for accurate diagnosis and effective treatment. Recent systematic reviews have demonstrated that modulation of the gut microbiota through probiotics, prebiotics, and synbiotics may improve insulin resistance, androgen excess, and lipid metabolism in women with PCOS (30). Against this background, the present narrative review aims to provide a comprehensive and critically appraised and updated overview of: (i) the pathophysiological relationship between gut microbiota dysbiosis and insulin resistance in PCOS; (ii) the genetic and epigenetic dimensions of host–microbiome interaction; and (iii) the emerging and established therapeutic strategies targeting the gut microbiome in PCOS management.

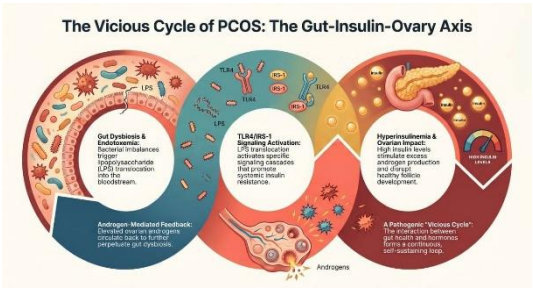

Figure 1. The Gut Microbiota–Insulin Resistance–Ovarian Dysfunction Axis in PCOS

Methodology of the Review

This narrative review was done by searching the PubMed, Scopus and Google Scholar databases from August 2025 to January 2026. The articles were identified by searching combinations of the keywords “polycystic ovary syndrome”, “PCOS”, “gut microbiota”, “gut microbiome”, “dysbiosis” and “insulin resistance” combined with Boolean operators (AND/OR) published from the year 2015 to 2025. The time frame 2015–2025 was chosen to reflect emerging technologies in the sequencing of the gut microbiome during that period, especially 16S rRNA amplicon sequencing and shotgun metagenomics, which have significantly improved the resolution and reproducibility of gut microbiome profiling. Additionally, the time period covered the latest revisions to the diagnostic criteria for PCOS, and the increased amount of clinical evidence relating gut dysbiosis with metabolic dysfunction ensured the relevance and currency of the literature reviewed. Studies were included if they were original research papers, clinical studies, or review papers that had the following focus: relationship between gut microbiota dysbiosis and insulin resistance in PCOS and relationship between insulin resistance and PCOS. Articles that were not published in English, conference abstracts, editorials and studies not directly related to the topic of the review were omitted. Studies were considered for inclusion in the review if the title, abstract and full-text were relevant to the aims of the review. Formal quality assessment tool not applied – this work was a narrative review.

Insulin Resistance in PCOS

PCOS is strongly associated with insulin resistance, which promotes compensatory hyperinsulinemia, increased ovarian androgen production and altered follicular development (7). Insulin resistance is a central component of PCOS pathophysiology and may occur independent of obesity (8). Hyperinsulinemia also contributes to the metabolic syndrome, raising the chances of type 2 diabetes and cardiovascular disease (9). Insulin resistance is a pathological disorder whereby the insulin target tissues including the muscle, liver and adipose tissue exhibit a reduced response to normal insulin levels. This is followed by compensatory hyperinsulinemia until pancreatic β-cell function becomes insufficient to meet

metabolic demands, ultimately contributing to the development of type 2 diabetes mellitus (10). IR is directly connected with metabolic syndrome and a plethora of pathological conditions such as cardiovascular diseases, obesity, fatty liver, and PCOS. In PCOS, IR and compensatory hyperinsulinemia are present in approximately 70% of patients, contributing to androgen excess, anovulation, impairing follicular maturation and metabolic dysfunction (8). Dietary interventions, exercise and pharmaceutical agents are used to enhance insulin sensitivity, which can have a profoundly positive effect in improving the clinical symptoms and endocrine pathophysiology of PCOS (11).

Understanding insulin resistance is essential for elucidating the pathogenesis of PCOS, as it represents a key metabolic abnormality contributing to both the reproductive and metabolic manifestations of the syndrome. The different markers help in diagnosing and assessing IR such as homeostasis model assessment of insulin resistance (HOMA-IR), quantitative insulin-sensitivity check index (QUICKI), fasting glucose-to-insulin ratio, the glucose/insulin (G/I) ratio, and the McAuley index (12,13).

Insulin resistance is one of the key issues of the polycystic ovary syndrome (PCOS) management, which is addressed with pharmacological interventions. Metformin remains the first-line pharmacological therapy and has demonstrated consistent efficacy in improving insulin sensitivity, lowering hyperinsulinemia, and alleviating both metabolic and reproductive issues in those with the condition (14,16). Some groups of patients have responded positively to thiazolidinedione, such as pioglitazone, though their use is limited due to adverse effects (17). The latter agents can be helpful in the treatment of metabolic dysfunction related to polycystic ovary syndrome (PCOS). In turn, the introduction of inositol derivatives and GLP-1 receptor agonists to the treatment plan was recommended by recent studies, especially when patients manifested obesity or glucose intolerance (18,32).

Pharmacological treatment is often combined with lifestyle interventions, including dietary modification and physical activity, to improve metabolic and reproductive outcomes in women with PCOS (19).

Gut Microbiota and Dysbiosis

The intestinal microbiota plays a role in energy homeostasis, immune and intestinal barrier regulation (20). Dysbiosis can be described as the loss of microbial diversity and changes in the composition of the bacterial community resulting in increased intestinal permeability and metabolic endotoxemia (21). Dysbiosis-driven translocation of lipopolysaccharide (LPS) leads to systemic inflammation via TLR4 activation, triggering pro-inflammatory cytokine release (TNF-α, IL-6) that disrupts the IRS-1/PI3K/Akt insulin signaling cascade, thereby promoting insulin resistance in PCOS (22).

Changes in bacterial composition, including a decrease in beneficial organisms such as Lactobacillus and Bacteroides, have been steadily observed in women with PCOS (23,24). Such microbiome remodeling is associated with hyperandrogenism, impaired glucose metabolism, and pro-inflammatory condition (25). The relationship between gut microbiota and androgen excess in PCOS appears to be bidirectional. Elevated androgen levels have been shown to directly alter gut microbial composition, further reducing the abundance of beneficial taxa such as Lactobacillus and Bacteroides, while promoting the growth of pro-inflammatory species (23,24). On the other hand, microbiome remodeling associated with PCOS, especially the decrease in SCFA-producing bacteria, could cause dysfunction of the intestinal barrier and increase systemic inflammation, which could further promote ovarian androgen secretion by activation of inflammatory signaling pathways (25). This two-way interaction means that dysbiosis and hyperandrogenism feed into each other and collectively drive the metabolic and reproductive decline observed in PCOS, making this a vicious cycle.

Table 1. Gut Microbial Taxa and Their Mechanistic Links to Metabolic and Endocrine Outcomes in PCOS

Microbial Taxa / Factor	Direction in PCOS	Mechanism	Metabolic / Endocrine Outcome
Lactobacillus spp.	↓ **Decreased**	Reduced lactic acid production; impaired intestinal barrier integrity	↑ Insulin resistance, ↑ androgens, ↑ pro-inflammatory state
Bacteroides spp.	↓ **Decreased**	Reduced SCFA production; altered bile acid metabolism	Impaired glucose metabolism; pro-inflammatory condition
LPS-producing bacteria	↑ **Increased**	TLR4 activation → TNF-α & IL-6 release → disrupts IRS-1/PI3K/Akt signalling cascade	Systemic inflammation; ↑ insulin resistance in PCOS
SCFA-producing bacteria	↓ **Decreased**	↓ GPR41/GPR43 activation; ↑ intestinal permeability; ↓ butyrate, propionate, acetate	↓ Insulin sensitivity; metabolic and endocrine dysfunction

Abbreviations: LPS, lipopolysaccharide; SCFA, short-chain fatty acid; TLR4, Toll-like receptor 4; TNF-α, tumour necrosis factor-alpha; IL-6, interleukin-6; IRS-1, insulin receptor substrate-1; PI3K, phosphoinositide 3-kinase; Akt, protein kinase B; GPR, G-protein coupled receptor; PCOS, polycystic ovary syndrome.

The genetic susceptibility and the gut microbiota interaction in PCOS.

Polycystic ovary syndrome (PCOS) is a well-recognized multifactorial endocrine disorder with significant genetic component. Family studies and twin analysis have established a high degree of heritability and genetic susceptibility has been shown to play an important role in the development of the disease (10,13). In genes associated with insulin signaling (e.g., INSR), androgen biosynthesis (e.g., CYP17A1), gonadotropin regulation (e.g., LHCGR), and inflammatory pathways, several polymorphisms have been described which may lead to an affected individual's predisposition to hyperinsulinemia, hyperandrogenism, and metabolic dysfunction (18). Ongoing work also indicates that host genetics can affect the composition of gut microorganisms and the integrity of the intestinal barrier, which has significant implications in regulating immunity and metabolic health (12). In contrast, the influence of the gut microbiota on gene expression can occur epigenetically and is bidirectional in terms of interactions between genetic background and microbial environment (33). Dysbiosis could increase insulin resistance and chronic low-grade inflammation, which in genetically predisposed individuals, may worsen the development of endocrine and metabolic dysfunction associated with PCOS (16,17). Thus, including genetic susceptibility in the gut microbiota–insulin resistance axis may offer a more holistic understanding of the complex pathophysiology of PCOS.

Therapeutic Approaches

Dietary modifications play an important role in restoring gut microbial balance. High-fiber and prebiotic diets promote the production of SCFAs — particularly butyrate, propionate, and acetate — which activate GPR41/GPR43 receptors, reduce intestinal permeability, and enhance insulin sensitivity (9,26). Probiotic and synbiotic supplementation has been associated with improvements in insulin resistance, inflammatory indicators, and hormonal parameters of PCOS women (27,5). Metformin remains the first-line pharmacological treatment for PCOS because of its insulin-sensitizing properties and potential effects on gut microbiota composition (11,15).

New microbiome-oriented interventions, such as fecal microbiota transplantation and postbiotics, are promising but need additional clinical testing (28).

Table 2. Microbiome-Targeted Interventions in PCOS: Mechanisms, Outcomes, and Evidence Levels

Intervention	Mechanism of Action	Metabolic / Endocrine Outcomes	Evidence Level
Metformin	Insulin sensitizer; modulates gut microbiota composition, increases Akkermansia abundance	↓ Insulin resistance, ↓ hyperinsulinemia, ↓ androgens, ↑ menstrual regularity	**Strong**
Probiotics	Restore microbial diversity; reduce intestinal permeability and systemic inflammation	↓ HOMA-IR, ↓ inflammatory markers, ↑ insulin sensitivity	**Moderate**
Prebiotics / High-fiber diet	Promote SCFA production (butyrate, propionate, acetate); activate GPR41/GPR43	↑ Insulin sensitivity, ↓ intestinal permeability, ↓ metabolic endotoxemia	**Moderate**
Synbiotics	Combined probiotic + prebiotic effect; synergistic modulation of gut microbiota	↓ Insulin resistance, ↓ inflammatory indicators, ↑ hormonal parameters	**Moderate**
Inositol derivatives (Myo- & D-chiro-inositol)	Insulin second-messenger activity; improves ovarian insulin signalling	↓ Insulin resistance, ↓ androgens, ↑ ovulation rate	**Emerging**
GLP-1 Receptor Agonists	Incretin effect; gut microbiota modulation; reduce appetite and body weight	↓ Glucose intolerance, ↓ BMI, ↑ metabolic outcomes in obese PCOS	**Emerging**
Fecal Microbiota Transplantation (FMT)	Direct transfer of healthy donor microbiome to restore eubiosis	Potential ↓ insulin resistance and inflammation; requires clinical validation	**Investigational**
Postbiotics	Bioactive compounds from microbial metabolism (SCFAs, bacteriocins, peptides)	Potential anti-inflammatory and insulin-sensitizing effects; under investigation	**Investigational**

Evidence levels: Strong = consistent RCT evidence; Moderate = limited/heterogeneous RCTs or meta-analyses; Emerging = early-phase trials or observational data; Investigational = preclinical or case-series only, requires further clinical validation. Abbreviations: SCFA, short-chain fatty acid; GPR, G-protein coupled receptor; HOMA-IR, homeostatic model assessment of insulin resistance; FMT, fecal microbiota transplantation; BMI, body mass index; PCOS, polycystic ovary syndrome.

DISCUSSION AND CONCLUSION

Dysbiosis of gut microbiota appears to be closely linked to insulin resistance and metabolic disturbances observed in polycystic ovary syndrome (PCOS), though definitive causal relationships remain to be established through longitudinal research. A change in microbial composition is associated with the increased intestinal permeability, systemic inflammation, and hormone imbalance, thus worsening the severity of the disease. Dietary, probiotic, and pharmacological interventions targeting the gut microbiome may offer promising therapeutic options to improve metabolic and reproductive outcomes, though most remain investigational and require further clinical validation. It should be noted that the majority of studies reviewed here are limited by small sample sizes, heterogeneous microbiome sequencing methods, and key confounders such as BMI, dietary habits, and metformin use, which may independently influence gut microbiota composition. These limitations preclude definitive causal

conclusions and underscore the need for standardized, large-scale longitudinal studies. Further studies should focus on identifying microbial biomarkers and optimizing targeted microbiome-based therapy.

Recommendations

- All criteria and definitions of PCOS should be standardized for better cross-study comparability along with definition of insulin-resistance (HOMA-IR cut-off definition).
- Perform big-scale longitudinal cohort studies to determine the link between dysbiosis of the gut and the insulin resistance, where the latter is regarded as a consequence of the former.
- Include confounding parameters of interest in the design and analysis of studies including body mass index/adiposity, habitual diet, physical activity, antibiotic exposure, probiotic use and metformin treatment.
- Use appropriate and standardized microbiome profiling techniques and reporting criteria, such as sampling, selection of sequencing platforms, and bioinformatics pipelines, to enhance reproducibility.
- Evaluate microbiome-directed therapy in adequately powered randomized controlled trials, based on combined outcomes, such as HOMA-IR, oral glucose tolerance test indices, inflammatory outcomes and androgen outcomes.
- Investigate factors linking dysbiosis with insulin resistance such as intestinal permeability, metabolic endotoxemia, chronic inflammation and microbiota-derived metabolites.
- Identify microbial signature and potential microbial biomarkers by multi-omics (metagenomics and metabolomics) solutions, which can be used for risk stratification.
- Create individual treatment strategies according to the degree of obesity, hyperandrogenism, initial microbiome and lifestyle factors.
- Future studies may benefit from incorporating artificial intelligence and machine learning to improve disease prediction and personalized therapeutic strategies.

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