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THE ROLE OF GUT MICROBIOTA IN THE PATHOGENESIS AND TREATMENT OF POLYCYSTIC OVARY SYNDROME (PCOS): A REVIEW ARTICLE

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ABSTRACT

Polycystic Ovary Syndrome (PCOS) is a common endocrine disorder that affects around 10–15% of women of reproductive age. It is characterized by a combination of metabolic, hormonal, and reproductive abnormalities, including insulin resistance (IR) and hyperandrogenism. Recent research suggests that gut microbiota dysbiosis is a major contributing factor, influencing insulin metabolism, inflammation, and hormonal regulation. Changes in the microbial community, especially an increase in pro-inflammatory bacteria exacerbate IR and androgen imbalances. Interventions such as probiotics, prebiotics, metformin, and lifestyle changes show potential in addressing these issues. However, more studies are needed to optimize microbiome-based treatments.

Aim of the Study: This article explores the role of gut microbiota dysbiosis in PCOS, with a focus on its impact on inflammation, insulin resistance, hormonal imbalances, and potential microbiome-centered therapeutic approaches.

Materials and Methods: A systematic review of the literature was conducted using PubMed, Google Scholar, and Medline. The main search terms included "PCOS," "gut microbiota," "inflammation," "insulin resistance," "androgens," and "microbial diversity." Studies linking gut dysbiosis to PCOS were selected for inclusion.

Results: Women with PCOS typically exhibit lower microbial diversity, with a higher abundance of pro-inflammatory species, which correlates with increased insulin resistance and elevated androgen levels. Specific bacteria, including *Lactobacillus*, *Bifidobacterium*, and *Candida*, have been identified as markers of dysbiosis.

Therapeutic interventions aimed at restoring microbial balance, such as probiotics, prebiotics, antibiotics, and metformin, show promise in alleviating PCOS symptoms.

Conclusions: Gut dysbiosis plays a crucial role in the pathophysiology of PCOS by exacerbating inflammation, insulin resistance, and hormonal imbalances. Emerging microbiome-targeted therapies could complement current treatments, but further research is essential to refine these approaches and develop standardized clinical protocols.

Keywords: polycystic ovary syndrome (PCOS), gut microbiota, inflammation, insulin resistance, hyperandrogenism.

INTRODUCTION

Polycystic ovary syndrome (PCOS) affects 10-15% of women of reproductive age. Diagnosis is based on the Rotterdam criteria, which require two of three features: (I) oligo- or anovulation, (II) clinical or biochemical signs of hyperandrogenism, and (III) polycystic ovaries seen on ultrasound, after excluding other disorders (59). Insulin resistance (IR) plays a crucial role in the pathophysiology of PCOS, with its expression differing across tissues and PCOS phenotypes. Genetic and epigenetic factors, in conjunction with hyperandrogenism and obesity, exacerbate IR, positioning it as a central element in the pathogenesis of the syndrome (80). PCOS increases the likelihood of developing metabolic issues early in life. Common complications include traditional cardiovascular disease (CVD) risk factors such as obesity, glucose intolerance, type 2 diabetes, abnormal cholesterol levels, and high blood pressure.

Among these, obesity stands out as a prevalent concern frequently raised by individuals with PCOS (25). Additionally, depression is more common in women with PCOS than in the general population, significantly reducing their quality of life. Low self-esteem in these patients is often linked to PCOS-related issues such as obesity, acne, androgenic hair loss, and hirsutism (31). The exact cause of PCOS remains unclear, but it is considered a multifactorial disorder involving genetic, metabolic, endocrine, and environmental factors. Evidence suggests that PCOS may begin in utero in genetically predisposed individuals, manifest during puberty with menstrual irregularities, and persist throughout reproductive years (46). The concept of gut microbiota dysbiosis in PCOS, introduced decades ago, suggests that disturbances in gut bacteria can activate the immune system, increase serum insulin levels, impair the function of insulin receptors, elevate ovarian androgen levels, and disrupt normal follicle maturation (27). Studies suggest that gut microbiota (GM) may contribute to the onset of PCOS by impacting energy absorption, the metabolism of short-chain fatty acids (SCFA), lipopolysaccharides, choline, bile acids, intestinal permeability, and the brain-gut axis (81). The purpose of this review is to examine the link between PCOS and gut microbiota, outlining possible underlying mechanisms and exploring emerging treatment options based on this connection.

GUT MICROBIOME

The human gut microbiota consists of about 10¹³ to 10¹⁴ microorganisms (14). The most common are bacteria, especially anaerobes. The intestinal microbiome also includes viruses, protozoa, archaea, and fungi (55). *Bacteroidetes* and *Firmicutes* are the two most represented bacterial types in the human gut microbiome. *Actinobacteria*, *Fusobacteria*, and *Verrucomicrobia* are present but in a relatively minor presence (14). Human gut microbiota creates a symbiotic relationship with the host and is essential for the proper functioning of the human body, including the endocrine system (6). There are reports indicating the relationship between the personal microbiota of the body and the production of sex hormones. There is a difference between the gut microbiome of women and men (16). The differences between sexes are supposed to be mostly linked to the relationship between gut microbiome and estrogen levels as well as testosterone levels. It has been found that healthy women are more likely to have a higher abundance of *Bacteroidetes*, a lower abundance of *Firmicutes*, the *Ruminococcaceae* family and increased diversity. Healthy men with proper testosterone levels are found to have a higher abundance of *Ruminococcus*, *Acinetobacter*, and an increased microbial diversity. Raised testosterone levels in women were positively correlated with *Escherichia* and *Shigella* spp. The levels of *Ruminococcus* spp. were correlated negatively with high testosterone levels in women. Women with PCOS have been found to have a different gut microbiome from healthy women (8). In recent studies changes in gut microbiome were observed between premenopausal and postmenopausal women. Estrogen deficiency associated with menopause is found to cause gut microbial alterations (16). Furthermore higher estrogen levels were linked to gut microbial diversity (60). Premenopausal women were found to have an increase of the microbial steroid downregulation, which was found to be positively associated with plasma progesterone levels (45). In women using hormonal contraceptives reduction in gut microbial diversity was found. It is due to the fact of serum estradiol and progesterone reduction in these patients (40).

GUT MICROBIOME AND PCOS

Patients with PCOS exhibit decreased gut microbial diversity and damage to the intestinal mucosal barrier when compared to healthy individuals. This foundational imbalance may set the stage for further

metabolic and immune complications in PCOS (40).

In cases of PCOS, microbial diversity tends to diminish, often characterized by fewer beneficial bacteria such as Lactobacilli and Bifidobacteria. Meanwhile, an increase in detrimental bacteria, like *Escherichia* and *Shigella*, is frequently observed (3). A different study found a decrease in *Lachnospira* and *Prevotella*, along with an increased presence of *Bacteroides*, *Parabacteroides*, *Lactobacillus*, *Fusobacterium*, and *Escherichia/Shigella* in PCOS patients. These microbial alterations suggest an imbalance leaning towards pro-inflammatory bacteria, disrupting the typical harmony with anti-inflammatory species (37). Higher concentrations of bacterial groups, including Proteobacteria, Gammaproteobacteria, Enterobacteriaceae, Erysipelotrichaceae, Planococaceae, Gemmales, and Bacillales, were identified in women with PCOS. Increased Proteobacteria has been linked to type 2 diabetes, metabolic syndrome, and inflammatory bowel disease, whereas individuals with non-alcoholic fatty liver disease (NAFLD) showed an increase in Gammaproteobacteria (43). Additionally, in PCOS patients, the fungal composition of the gut microbiome is altered. Specific fungal markers, such as *Candida*, *Malassezia*, *Kazachstania*, *Microascus*, *Coniochaeta*, *Xepicula*, *Paraphoma*, *Pyrenochaetopsis*, *Cephalophora*, *Epicoccum*, and *Sclerophora*, are commonly observed. Furthermore, in PCOS patients with a BMI of 24 or above, even more distinct fungal genera appear as notable indicators (78).

One study found a significant increase in *Bacteroides vulgatus* in the gut microbiota of individuals with PCOS, alongside decreased levels of glycodeoxycholic acid and tauroursodeoxycholic acid (54). The gut microbiome affects host metabolism by interacting with signaling pathways. In patients with PCOS, alterations in the microbiome can impact bile acid metabolism (62). This suppresses interleukin 22 secretion from intestinal innate lymphoid cells (ILC3), which plays a role in the onset of ovarian dysfunction and metabolic issues in PCOS (82). Women diagnosed with PCOS often experience the rise in opportunistic pathogens, especially those associated with the *C. perfringens* group and *Staphylococcus* species, may play a role in exacerbating chronic inflammation (33).

In the gut microbiota, endotoxin-induced activation of macrophages stimulates the production of TNF- α and IL-6, leading to chronic low-grade inflammation, which contributes to insulin resistance and the progression of PCOS (75). The gut-brain axis may contribute to PCOS pathology, with lower levels of gut-brain peptides - such as ghrelin, cholecystokinin, and peptide YY found in affected patients. Altered gut microbiota is believed to influence insulin resistance and hyperandrogenism through its effects on these peptides. Additionally, the microbiota interacts with hormones like estrogens, androgens, and insulin, which may play a key role in the hormonal imbalance seen in PCOS (44). Among women with PCOS, a decrease in gut microbiota α diversity appears connected to elevated hyperandrogenism, total testosterone, and hirsutism levels (69). Gut microbiota analysis in PCOS patients revealed that those with higher testosterone levels had increased *Prevotella*, *Blautia*, *Dialister*, *Ruminococcus torques* group, and *UCG-002*, while *Alistipes*, *Dysosmobacter*, *Phocaeicola*, and *Faecalibacterium* were reduced. Eight specific genera effectively differentiated high- and low-testosterone groups in PCOS (77). An unhealthy diet contributes to greater gut mucosal permeability, permitting more lipopolysaccharides (LPS) from Gram-negative bacteria to leak into the bloodstream. This triggers immune system activation, impairs insulin receptor function, and elevates insulin levels (70). Once in the bloodstream, LPS bind to the CD14-Toll-like receptor complex on immune cell membranes, triggering signaling pathways that increase pro-inflammatory cytokines and lead to chronic inflammation (18). Increased fasting insulin levels stimulate the pituitary gland to secrete more luteinizing hormone (LH), which boosts androgen production by enhancing the activity of the enzyme cytochrome P450C17 α in theca cells. Elevated androgen levels contribute to symptoms such as excessive hair growth, acne, and hair thinning, while higher local androgen levels in the ovaries can lead to premature follicle atresia and ovulatory dysfunction (41). In individuals with PCOS who are overweight or obese, Ruminococcaceae is found to be more prevalent. This bacterial group may be related to clinical symptoms associated with excessive androgens (10).

One study identified notable changes in the gut microbiota of women with PCOS, regardless of obesity, when compared to non-obese controls. The microbial imbalance in PCOS closely mirrored the dysbiosis commonly associated with obesity (42). In an additional study, it was found that diet has a significant impact on gut microbial diversity. The lowest diversity and noticeable shifts in microbial composition were observed in mice on diets with the highest levels of protein, carbohydrates, or fats (58). Moreover, it was observed that women with PCOS have disruptions not only in their gut microbiota but also in their vaginal microbiome. This research compared the lower genital microbiome profiles of women with PCOS to those of healthy controls, revealing that women with PCOS had significantly reduced levels of *Lactobacillus* species in both the cervical and vaginal areas compared to healthy individuals (71). Addressing gut dysbiosis may be key to managing PCOS symptoms, as microbial imbalances contribute to inflammation, insulin resistance, and hormonal disruptions.

Understanding these changes in the microbiota offers insight into PCOS pathology and its broader impact on health.

RELATED INTERVENTIONS IN PCOS

Although the relationship between gut microbiota and its pathogenesis in PCOS has found, its therapeutic role is still under clinical investigation (9). Treatment strategies can include the use of probiotics, prebiotics, antibiotics, metformin, polyphenols, Fecal Microbiota Transplantation (FMT), IL-22 and lifestyle intervention (8, 67).

PROBIOTICS

According to the World Health Organization (WHO) probiotics are “live microorganisms that, when administered in adequate amounts, confer a health benefit on the host” (17). Probiotics are found in fermented foods. Evidence suggests that probiotics could improve PCOS treatment by weight decrease, beneficial effects on glycaemia, serum insulin levels, triglycerides (TG) and very-low-density lipoprotein (VLDL) cholesterol (8). Probiotics have also been found to reduce malondialdehyde (MDA) and Free Androgen Index (FAI) levels as well as increase Sex Hormone Binding Globulin (SHBG) and nitric oxide (NO) levels (61).

PREBIOTICS

Prebiotics defined by 6th Meeting of the International Scientific Association of Probiotics and Prebiotics (ISAPP) are “selectively fermented ingredients that result in specific changes in the composition and/or activity of the gastrointestinal microbiota, thus conferring benefit(s) upon host health” (15). Prebiotics have been found to have positive effects on growth of *Bifidobacterium* and *Lactobacillus*, which in return increase HDL levels and decrease serum TG, total cholesterol, and LDL cholesterol and fasting plasma glucose levels. Nevertheless, further studies are required to clarify and compare the effectiveness of different probiotic and prebiotic strains and dosages, determine the optimal treatment duration, and assess the health benefits of probiotics, prebiotics on clinical outcomes in PCOS (8).

ANTIBIOTICS

Antibiotics are medications designed to combat bacterial infections by either stopping bacterial growth or killing bacteria directly (73). They function by targeting essential bacterial processes, such as cell wall synthesis or protein production (30). Despite their vital role in modern medicine, excessive or improper use of antibiotics can lead to antibiotic resistance, where bacteria adapt to survive treatment (35). This growing concern underscores the need for careful antibiotic use. Additionally, antibiotics can alter the gut microbiota, potentially disrupting the balance of beneficial bacteria (12), which may have implications for conditions like metabolic disorders, including PCOS (70). Recent research has explored the link between antibiotics and Polycystic Ovary Syndrome (PCOS), focusing on how antibiotic use may affect the gut microbiome. Studies show that individuals with PCOS often have a distinct gut microbiota, with an abundance of bacteria that could contribute to symptoms like elevated testosterone levels, a key factor in excess hair growth, acne, and menstrual irregularities (54). Research suggests that antibiotics, by reducing these specific bacteria, may lower testosterone levels and improve metabolic markers such as insulin sensitivity and ovarian function (66). While promising results have been seen in animal models, clinical evidence in humans remains limited. Further studies are needed to evaluate the effectiveness of antibiotics in PCOS treatment and whether alternatives like probiotics or dietary changes might offer better long-term solutions (28).

FECAL MICROBIOTA TRANSPLANTATION (FMT)

FMT is a method of transplantation of feces from healthy donors to the intestinal tract of patients (22). The primary goal of FMT is to restore a balanced, diverse microbiome in individuals with gut dysbiosis or microbial imbalances (79). Studies found that transplanting the fecal microbiota of healthy mice along with *Lactobacillus* improved the gut microbiota composition and helped restore the hormonal cycle in letrozole-induced PCOS rats.

Letrozole, an aromatase inhibitor, is commonly used to induce a PCOS-like condition in animal models, especially in rats. This model mimics many features of human PCOS, including hormonal imbalances and insulin resistance (29). The PCOS rats that were exposed to a healthy gut microbiome exhibited improvements in hormone levels, glucose and lipid metabolism. Specifically, there was a reduction in testosterone, luteinizing hormone (LH), fasting blood glucose, and insulin levels, alongside improved insulin sensitivity and higher estradiol and estrone levels and as a result normalization of ovarian function (8, 67, 19).

Currently, there are no clinical data on the use of FMT for treating PCOS, aside from animal studies. Therefore, further research is needed to better evaluate this approach for PCOS treatment in humans (63).

METFORMIN

Metformin is a commonly prescribed treatment for type 2 diabetes (53). Its most important effects include: reduction of gluconeogenesis in the liver, increased secretion of GLP-1, decreased glucose absorption in the intestine, improved utilization of glucose in peripheral tissues and anti-inflammatory effect which contributes to the improvement of the aforementioned benefits. Metformin is also widely used in the management of PCOS (33,57). The reported effects of metformin in the management of PCOS include restoring ovulation, promoting weight loss, lowering circulating androgen levels, and reducing the risks of miscarriage and gestational diabetes mellitus (GDM). It has been also found that metformin therapy in in vitro fertilization (IVF) enhances pregnancy outcomes. Additionally, metformin has been shown to aid in weight reduction, which can further alleviate symptoms of PCOS (34). Recent studies have increasingly recognized the impact of metformin on gut microbiota. Research has shown that metformin can alter the gut microbiota in patients with diabetes. After the treatment the microbiota of treated patients was transplanted into hyperglycemic mice, leading to improved glucose tolerance, suggesting that metformin's therapeutic effects may be mediated through changes in the gut microbiome (20).

Another research has shown that metformin increased the abundance of *Akkermansia*, *Bacteroides*, *Butyricimonas*, and *Parabacteroides* in mice on a high-fat diet. It also reduced IL-1 β and IL-6 levels in fat, which was linked to changes in bacterial populations. Additionally, fecal microbiota from metformin-treated mice and *Akkermansia muciniphila* extracellular vesicles improved the mice's body weight and lipid profiles (36).

INTERLEUKIN-22

Interleukin-22 (IL-22) is an immune-modulatory cytokine that plays a crucial role in maintaining mucosal barrier integrity and regulating immune responses. IL-22 is produced by several immune cells, including T-helper cells and innate lymphoid cells (ILCs), and is known to promote the production of antimicrobial peptides, which help maintain intestinal barrier function (65). Scientists transplanted gut microbiota from women with PCOS into mice, which led to insulin resistance, disrupted oestrous cycles, and cyst-like follicles, mimicking human PCOS symptoms. Administering *B. vulgatus* had similar effects, suggesting its role in PCOS pathogenesis. The study found that *B. vulgatus* alters bile acid metabolism, reducing bile acids that stimulate IL-22-producing group 3 innate lymphoid cells (ILC3s) in the gut microbiota. Mice transplanted with *B. vulgatus* showed fewer ILC3s and lower IL-22 levels. These changes, along with insulin resistance and ovarian abnormalities, were reversed by IL-22 or the bile acid glycodeoxycholic acid. The findings suggest that targeting the bile acid–IL-22 signaling axis could be a potential treatment for PCOS (54, 32).

Polyphenols are secondary metabolites found in plants, primarily serving to protect against ultraviolet radiation and pathogen attacks. In recent years, there has been growing interest in the health benefits of dietary plant polyphenols, particularly for their antioxidant properties (50). Dietary interventions, especially bioactive compounds such as polyphenols, are promising for modulating the gut microbiota (72).

ANTHOCYANINS

Anthocyanins, found in foods like grapes, can improve ovarian function in PCOS by modulating steroid hormone expression and regulating the gut microbiota (47). Anthocyanins promote beneficial bacteria like *Bifidobacterium*, *Lactobacillus* and *Enterococcus*, improving gut health and barrier function. Therefore, anthocyanins may help improve gut microbiota imbalance by lowering serum androgen levels, although this needs further experimental validation (82).

RESVERATROL

Resveratrol is a plant-derived distyrene compound with antifungal and antibacterial properties. It is present in a variety of fruits, including grapes (and their juices), oranges, cranberries, currants, and peanut skins (2, 49). Research has shown that resveratrol is a potent antioxidant, as well as possessing antibacterial, anti-obesity, anti-inflammatory, and anticancer effects (56). Other beneficial effects of resveratrol include decreasing sinus follicles, increasing secondary follicles, reducing granulosa cell death, lowering androgen levels and oxidative stress, and increasing insulin sensitivity (5, 38, 39). After microbiota transplantation from donors who had consumed a resveratrol-supplemented diet, the recipients showed an increase in the Shannon index. Their Firmicutes/Bacteroidetes ratio at the phylum level also significantly increased. Additionally, the relative abundances of *Lactobacillus aviarius* and *Lactobacillus salivarius* were higher, while the relative abundance of *Bacillus velezensis* decreased (82, 74).

CATECHINS

Catechins are polyphenolic compounds, specifically flavanols, which belong to the flavonoid family and are found in various plants. Major dietary sources of these flavanols include green tea, wine, and cocoa-based products (1). The catechin derivatives found in green tea include epicatechin (EC), epigallocatechin (EGC), epicatechin gallate (ECG), and epigallocatechin gallate (EGCG). Among these, EGCG exhibits the most significant anti-inflammatory and anticancer potential (11). They are known for their anti-infective properties and have been studied for their antimicrobial effects, especially in relation to gastrointestinal diseases, including colon cancer (82). However, catechins are volatile and prone to degradation and metabolism, particularly when they interact with hydroxyl groups on phenolic rings under physiological conditions. Even when administered intravenously, catechins are partially degraded before reaching their target tissues. They are primarily metabolized in the liver, small intestine, and colon (82, 4). Research has shown that catechins can reduce inflammation and matrix degradation. For instance, a study by Hong et al. demonstrated that catechins can significantly decrease the expression of p-NF- κ B p65 and inflammatory proteins (IL-1 β , IL-6, and TNF- α) in uterine tissues, suggesting that oolong tea catechins can inhibit uterine inflammation through the suppression of p-STAT3 signaling (82, 26). Supplementation with green tea extract has been found to lower serum LH levels and improve insulin resistance in PCOS rats (82, 13). Clinical studies have also reported that catechin supplementation helps normalize hormone levels in women with PCOS, likely due to its antioxidative effects (82, 21).

ISOFLAVONES

Isoflavones, primarily found in soy products, have anti-inflammatory effects and may help manage PCOS. They can modulate the gut microbiota, with studies showing that isoflavones increase the diversity of gut bacteria in women with PCOS. Additionally, they influence the production of equol, a compound that is beneficial for managing PCOS symptoms (82). The interaction between gut microbiota and polyphenols in PCOS is complex, and further research is needed to understand how polyphenols influence gut microbiota composition and metabolite production. This knowledge could lead to new therapeutic options for managing PCOS (82).

LIFESTYLE INTERVENTION

Management of PCOS has primarily focused on medical treatments, but lifestyle changes, including dietary adjustments, exercise, and reducing psychosocial stressors, are recommended as part of therapy, especially for individuals with obesity. Obesity negatively impacts both metabolic and hormonal balance in PCOS, potentially reducing the effectiveness of treatments (24). Short-term weight loss effectively improves insulin sensitivity and restores fertility, but maintaining it and assessing long-term benefits remain challenging. Current approaches should prioritize lasting results through diet and exercise (48). Diet plays a key role in regulating the gut microbiome, as changes in macronutrient composition influence the balance of microbial populations (58). A well-planned diet should include appropriate calorie levels, a balanced distribution of macronutrients, and low-glycemic index foods. Vitamin D and inositol supplementation are particularly effective in addressing symptoms of polycystic ovary syndrome. They enhance carbohydrate metabolism, reduce insulin resistance, promote weight loss, and support menstrual regularity and ovulation, improving fertility outcomes (64). Evidence for a specific diet for PCOS is limited, as adjustments to protein, carbohydrate, or fat content generally produce similar results in managing symptoms (7). Dietary fiber intake serves as a useful indicator of a healthy whole-food diet. Adequate fiber supports better insulin sensitivity, lowers blood glucose and inflammation, and reduces androgen and LPS levels, factors linked to PCOS development (51). Sleep disturbances reduce energy expenditure, promoting fat accumulation and worsening insulin resistance, which can aggravate PCOS symptoms. Additionally, poor sleep may undermine the effectiveness of lifestyle changes after diagnosis. Improving sleep could support women with PCOS in adopting and maintaining healthier habits (68). In experiments on mice, sleep deprivation disrupted gut tryptophan-kynurenine metabolism, increased permeability, triggered systemic inflammation, and weakened the blood-brain barrier. It also altered hippocampal metabolism, linking it to anxiety and depression (23).

Exercise is increasingly recognized as a key component in managing PCOS. High-intensity workouts may be most effective in improving cardiorespiratory fitness, body composition, and insulin resistance (52). Physical activity plays a key role in lowering the risk of cardiovascular disease in women with PCOS by enhancing insulin sensitivity and reducing hyperinsulinemia. Additionally, PCOS is often associated with mental health challenges, and exercise can improve psychological well-being, though its impact depends on individual physiological factors (76). Balanced nutrition, regular exercise, and improved sleep are essential for addressing metabolic, reproductive, and psychological challenges. Tailored interventions can significantly enhance overall health and quality of life for women with PCOS.

CONCLUSIONS

1. Gut microbiota plays a key role in the pathogenesis of PCOS: Gut

microbiota dysbiosis is an important factor contributing to the development of PCOS, affecting metabolism, inflammation, and hormonal imbalances in women with the condition.

2. Innovative therapeutic approaches: The use of methods such as probiotics, prebiotics, metformin, fecal microbiota transplantation (FMT), and polyphenols shows significant potential in improving clinical outcomes in PCOS. These interventions can help normalize hormone levels, improve insulin sensitivity, and reduce inflammation.

3. Role of polyphenols in PCOS treatment: Dietary polyphenols, such as anthocyanins, resveratrol, and catechins, have antioxidant and anti-inflammatory properties that help improve gut microbiota function and alleviate PCOS symptoms,

including hormonal imbalances and insulin resistance.

4. Lifestyle's role in PCOS management: Proper nutrition, physical activity, and sleep improvement are essential components of a comprehensive treatment plan for PCOS. These interventions help improve metabolic markers, reduce inflammation, and support hormonal balance.

5. Need for further research: Despite promising results, current approaches require further clinical studies to confirm their effectiveness, safety, and long-term effects. Future research should focus on optimizing dosages, treatment durations, and personalizing therapy based on microbiological and phenotypic characteristics of patients.

6. Prospects of personalized medicine: Approaches focused on personalized medicine, considering microbiota and specific disease characteristics, could significantly improve the effectiveness of PCOS treatment and lead to the development of new, more targeted therapeutic strategies. Overall, the article highlights the importance of a comprehensive and individualized approach to PCOS treatment, taking into account gut microbiota, lifestyle, and dietary interventions, and opens new prospects for developing effective therapeutic methods.

CONFLICTS OF INTEREST

Authors have no conflict of interest to declare.

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AUTHOR CONTRIBUTIONS

Conceptualization: Aleksandra Śledziewska; methodology: Kamila Sieradocha; formal analysis: Magdalena Cyrkler; investigation: Aleksandra Śledziewska; resources: Aleksandra Giba; data curation: Aleksandra Zagajewska; writing - original draft: Aleksandra Giba, Michał Wąsik; writing - review and editing: Kamila Sieradocha, Aleksandra Śledziewska, Aleksandra Reda, Dorota Słupik, Michał Wąsik, Aleksandra Zagajewska; visualization: Kamila Sieradocha; supervision, Aleksandra Śledziewska; project administration: Aleksandra Zagajewska

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