THE ROLE OF GUT MICROBIOTA ON POLYCYSTIC OVARIAN SYNDROME (PCOS): A NARRATIVE REVIEW

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ABSTRACT

Polycystic ovarian syndrome (PCOS) affects a significant proportion of women of reproductive age, with prevalence estimated to be between 4% and 21%. It is now understood that PCOS is a complex condition influenced by various internal and external factors. Changes in the gut microbiome have been linked to PCOS, and it is believed that gut microbes may play a role in the development of the syndrome. While many studies have suggested a connection between gut microbiota and PCOS, the specific cause-and-effect relationship remains unclear. Within the intestinal tract and feces, there are six primary bacterial species, with Bacteroidetes and Firmicutes being the most important for overall health. Shifts in the abundance of these bacteria have been associated with PCOS. The gut microbiota/bile acid/interleukin-22 (IL-22) axis has been investigated as a potential mechanism underlying the development of PCOS. Certain types of gut microbiota can trigger oxidative stress and inflammatory responses from mononuclear cells in women with PCOS by influencing dietary factors. Probiotics have demonstrated potential benefits for specific parameters related to PCOS, such as BMI, FPG, and lipid profiles. Women with PCOS have been found to consume lower levels of dietary fiber compared to those without the condition. Although several explanations have been proposed for the link between gut microbiota and PCOS, further research is necessary to explore other potential mechanisms.

Keywords: Gut Microbiota; Microbiome; Polycystic Ovarian Syndrome

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INTRODUCTION

The prevalence of polycystic ovarian syndrome (PCOS) among women of reproductive age ranges from 4% to 21%, and it has emerged as the most prevalent endocrine disorder affecting women's reproductive system.[1,2] PCOS diagnosis based on 2003 Rotterdam criteria is comprised of two out of the following three features: hyperandrogenism, oligo-or amenorrhea, or polycystic ovaries.[2] The characteristics of PCOS are persistent anovulation, high androgen levels, polycystic ovarian changes, insulin resistance (IR), and obesity.[1,3]

It is now recognized that PCOS is a multifactorial disorder influenced by significant internal factors, such as gut microbiota, and external environmental factors, like lifestyle choices.[3] It was found that intestinal microorganisms are related to the homeostasis of physiological functions. Gut microbiome alterations are associated with PCOS, and gut microbes may be involved in the pathology of PCOS. PCOS patients with IR experience alterations in the intestinal flora.[1,2] Faecal microbiota transplantation in mice from women with PCOS also developed the phenotype.[4] Previous studies showed that the gut microbiome is affected in PCOS patients.[5,6] Moreover, gut microbiome alterations could specifically affect the reproductive endocrine system. Based on a study result, intestinal microbiota alterations included increased species of Bacteroidetes and a decrease in Firmicutes. At the genus level, *Bacteroidetes* and *Lachnoclostridium* were found to increase.[1] Many studies have indicated a link between gut microbiota and PCOS, but the exact cause-and-effect relationship between them is still uncertain.

MATERIALS AND METHODS

A systematic literature search was performed using PubMed, Web of Science, and Scopus databases for peer-reviewed articles published between January 2015 and December 2025. The search strategy employed both Medical Subject Headings (MeSH) terms and free-text keywords including: "polycystic ovarian syndrome," "PCOS," and "gut microbiota" Reference lists of relevant articles were manually searched to identify additional studies. Studies included if they investigated molecular mechanisms of PCOS, examined gut microbiota function or expression, explored gut microbiota regarding the PCOS, or analyzed gut microbiota in PCOS or related conditions. Both original research articles and review papers were considered, with emphasis on recent high-quality studies providing mechanistic insights.

DISCUSSION

OVERVIEW OF PCOS

PCOS is the most common female reproductive endocrine disorder affecting reproductive-aged women.[1,2] PCOS has an estimated worldwide prevalence of 5%-15%.[7] PCOS is a complex disorder that is characterized by reproductive, metabolic, and psychological features.[8] PCOS diagnosis based on 2003 Rotterdam criteria is comprised of two out of the following three features: hyperandrogenism, oligo-or amenorrhea, or polycystic ovaries.[2] The characteristics of PCOS are persistent anovulation, high androgen levels, polycystic ovarian changes, insulin resistance (IR), metabolic syndrome, and obesity.[1,3]

Irregular menstrual cycles could be defined as usual in the first-year post-menarche as part of the pubertal transition, <21 or >45 days (1 to <3 years post menarche), <21 or >35 days or <8 cycles per year (3 years post menarche to perimenopause), >90 days for any one cycle (1-year post menarche), and primary amenorrhea by age 15 or > three years post breast development. PCOS should be considered a possible diagnosis when irregular menstrual cycles occur. Even with regular cycles, ovulatory dysfunction can occur, and measuring serum progesterone levels can confirm anovulation.[8] PCOS are closely related to infertility, cardiovascular disease risk, hyperandrogenism, gestational diabetes, impaired glucose tolerance, type 2 diabetes, obstructive sleep apnea, endometrial cancer, and so on in women.[8,9]

PCOS pathophysiology includes hormonal imbalance, inflammation, and insulin signaling pathways. The pathophysiology of PCOS involves intrinsic ovarian dysfunction, which is significantly affected by external factors such as disruptions in the hypothalamic-pituitary-ovarian axis and hyperinsulinemia. Increased pulsatility of gonadotrophin-releasing hormone (GnRH) leads to excessive luteinizing hormone (LH) secretion, impacting both ovarian androgen production and oocyte development. Disrupted feedback between the ovaries, pituitary, and hypothalamus exacerbates gonadotrophin irregularities. Hyperinsulinemia results from both peripheral insulin resistance and abnormal pancreatic beta cell function. PCOS tends to run in families, with various genetic abnormalities contributing to the syndrome's features and the heterogeneity of symptoms. Environmental factors, such as diet and lifestyle, also influence the expression of the syndrome.[10]

ROLE OF GUT MICROBIOTA

The gut microbiota, comprising over 100 trillion microbes, resides in the human gut. Recently, scientists have acknowledged that these intestinal inhabitants establish a

mutually beneficial relationship with the host, offering a variety of advantages. For instance, commensal microbes regulate the immune system, prevent pathogen colonization, and extract nutrients from food. However, gut microbiota dysbiosis has been linked to various diseases, including diabetes, obesity, and cardiovascular disease.[11] Microbiota composition and microbial metabolite production alteration may majorly impact the host metabolism.[12]

There are six main bacterial species in the intestinal tract, which can also be found in feces: Bacteroidetes, Firmicutes, Actinobacteria, Proteobacteria, Fusobacteria, and Verrucomicrobia. Bacteroidetes and Firmicutes comprise approximately 90 % of the bacterial composition in healthy individuals. Proteobacteria, a group of opportunistic bacteria and pro-inflammatory pathogens, account for less than 5 % of the bacterial population. Bacteroidetes and Firmicutes, which are the primary constituents, play a vital role in preserving overall health.[13] These bacteria are beneficial bacteria found predominantly in the human gut.[14]

Women showed higher alpha diversity in their gut microbiome compared to men. Research also indicated that men had a greater prevalence of Bacteroidetes, including *Prevotella* and *Bacteroides thetaiotaomicron*, compared to women.[7] The gut microbiota is a highly diverse and metabolically active community comprising approximately 3.9×10^{13} microbial cells. These microbes perform various beneficial functions for the host, such as fermenting undigested nutrients, synthesizing vitamins, and interacting with the immune system. The lack of these bacteria could have a notable effect on the host, other microbes in the community, and their metabolic products, particularly the short-chain fatty acid (SCFA) butyrate. Butyrate holds great significance for health because of its favorable characteristics and its impact on the immune system.[15]

GUT MICROBIOTA AND METABOLISM

The gut microbiota evolves symbiotic with its host, contributing to various physiological functions, including gut barrier regulation, immune response development and maintenance, and energy metabolism. High microbial abundance and diversity contribute to environmental stability. Disturbances in the normal gut microbiota (dysbiosis) have been linked to the development of conditions such as obesity, insulin resistance or diabetes, autoimmunity, inflammatory bowel disease, and kidney disease.[16,17]

Dietary fiber is one of the numerous factors that can impact the gut microbiota composition. While human digestive enzymes cannot hydrolyze dietary fiber, gut microbes can act on it, producing metabolites such as SCFA. Soluble fiber is typically fermented by the intestinal microbiota into SCFA, mainly acetate, propionate, and butyrate. These SCFAs can be absorbed into the bloodstream, impacting the host's metabolic regulation or serving as substrates for other microbes. The short-chain fatty acids (SCFAs) are carried into the body's circulation and have the potential to directly impact the body's metabolism by attaching to G-protein coupled receptors (GPRs). These receptors are found in various metabolically active tissues and play a role in maintaining glucose levels and regulating lipid metabolism. Furthermore, there are indications that SCFAs could work as inhibitors of histone deacetylases, thus influencing gene expression. SCFAs serve as a crucial energy source for cells in the lining of the gut and contribute to strengthening the gut barrier function. This improved function of the gut barrier reduces the entry of microbes and their byproducts into the blood, reducing immune responses associated with metabolic disorders. Research also suggests that other metabolic byproducts produced by microbes, such as indole and enterolactone, which are the results of microbial processing of dietary tryptophan and lignan, respectively, can influence metabolic regulation. Several studies have shown that increased levels of SCFAs from the gut can enhance insulin sensitivity, control weight, and reduce inflammation, which could potentially lower the risk of developing metabolic diseases.[18]

The decrease in the number and variety of microorganisms is closely associated with disease-related alterations, such as inflammation.[17] Chronic persistent inflammation at a low level plays a vital role as a risk factor in the onset of PCOS, possibly leading to increased levels of androgens and insulin resistance and playing a part in the abnormal alterations observed in PCOS.[19] Patients suffering from PCOS might encounter initial inflammation in the ovaries, potentially affecting the emergence and progression of PCOS by way of the hypothalamic-pituitary-ovary axis.[20]

EVIDENCE LINKING GUT MICROBIOTA AND PCOS

It is now recognized that PCOS is a multifactorial disorder influenced by significant internal factors, such as gut microbiota, and external environmental factors, like lifestyle choices.[3] Variations in diversity of the vaginal microbiome are strongly connected to PCOS. Noteworthy distinctions in the vaginal microbiome were noted between PCOS patients and women in good health, regardless of the presence of Lactobacillus. The alpha diversity of the vaginal microbiome decreased significantly in PCOS patients when

Lactobacillus spp. was dominant. Actinomyces could be utilized as a marker for detecting PCOS. Streptococcus might impact the pathological changes in PCOS by influencing the endocrine environment of the female reproductive system.[20] Alterations in the abundance of bacteria in Bacteroidetes and Firmicutes were found to be correlated with PCOS. In individuals with PCOS, there was a lower presence of Bacteroidetes and a higher presence of Firmicutes.[1,21]

Recent studies have indicated that an excess of androgens in women with PCOS and female rodent models is associated with changes in the gut microbiome. Research has revealed that PCOS is linked to reduced alpha diversity and changes in the abundance of specific bacteria belonging to the Bacteroidaceae, Clostridiaceae, Erysipelotrichidae,Lachnospiraceae,Lactobacillaceae,Porphyromonadaceae,Prevotellaceae,Ruminococcaceae, and S24-7 families, which have previously been associated with metabolic dysregulation.[7] Lu *et al.* discovered that there were noteworthy alterations in the vaginal microbial environment of PCOS patients, with Lactobacillus being the dominant species. This was supported by a decrease in the overall abundance of microbial flora and a declining pattern in bacterial diversity. The initial study identified a substantial reduction in microbiome abundance in PCOS patients who did not have vaginitis, indicating that the microbial population existed prior to the onset of inflammation.[20]

Patients with PCOS showed a noticeable link between the composition of their microbiota and their FSH levels. The presence of numerous anaerobic and facultative anaerobic bacteria displayed a significant inverse relationship with FSH, while Lactobacillus demonstrated a significant positive correlation with FSH levels. PCOS patients exhibited elevated levels of anti-Müllerian hormone, LH, and estrogen in their bloodstream, resulting in a feedback mechanism that decreases FSH levels. Decreased FSH levels are linked to irregular follicular growth and lack of ovulation in these patients.[9] Lu *et al.*'s research discovered that a reduction in FSH showed a strong connection with elevated levels of vaginal opportunistic pathogens. Conversely, a plentiful presence of Lactobacillus seemed to support the maintenance of normal FSH levels.[20]

The changes in the abundance of vaginal flora in PCOS patients could be partly attributed to environmental factors. Streptococcus, which is a common opportunistic pathogen causing suppurative inflammation, is frequently found in feces and the nasopharynx. Group B Streptococcus, a species of this genus, is a significant pathogen in perinatal infections among women. Previous research on vaginal flora has demonstrated a significant positive relationship between the prevalence of Streptococcus, age, and estradiol levels, indicating a potential association with ovarian dysfunction. Lu *et al.* discovered a negative correlation between Streptococcus and FSH, supporting the idea that Streptococcus may impact sex hormones and ovarian function. This underscores the crucial role of the vaginal microbiome in preserving a healthy female reproductive system.[20,22,23]

POTENTIAL MECHANISMS OF INTERACTION

The relationship between the gut microbiota/bile acid/interleukin-22 (IL-22) axis and PCOS has been studied as a potential mechanism. The transfer of fecal microbiota from PCOS-affected women to mice has adverse effects on ovarian functions, insulin resistance, bile acid metabolism, IL-22 secretion, and fertility. IL-22 plays a role in ameliorating the PCOS phenotype.[4,24] Certain types of gut microbiota can cause oxidative stress and inflammation in mononuclear cells of women with PCOS by affecting dietary factors. Oxidative stress and inflammation can also weaken the gut barrier, increasing harmful bacteria in the bloodstream. This activation of the immune system affects the function of insulin receptors, leading to higher insulin levels, which, in turn, can result in elevated androgen levels and abnormal follicle growth. Furthermore, the gut bacteria may contribute to PCOS by influencing energy absorption.[25–28]

Soluble fiber is typically fermented by the intestinal microbiota into SCFAs, primarily acetate, propionate, and butyrate. These SCFAs can be absorbed into the bloodstream, impacting the host's metabolic regulation or serving as substrates for other microbes. The SCFA enter the systemic circulation and can impact the body's metabolism by attaching to G-protein-coupled receptors (GPRs). These receptors are found in various tissues, active in metabolism, and regulate the balance of glucose and lipid metabolism. Additionally, SCFAs serve as an essential energy source for intestinal epithelial cells and help strengthen the gut barrier. Enhanced integrity of the gut barrier limits the entry of microorganisms and microbial compounds into the blood. This leads to a decrease in immune reactions linked to metabolic disorders or vaginitis in individuals with PCOS. SCFA promotes gut well-being, boosts gut barrier function, and lowers the movement of bacterial toxins through the intestinal lining, which can trigger inflammation and insulin resistance.[11] Some studies have shown that increased levels of gut-derived SCFAs can improve insulin sensitivity, aid in weight regulation, and

reduce inflammation, potentially lowering the risk of developing metabolic diseases.[18]

THERAPEUTIC IMPLICATIONS

Angoorani *et al.*'s research demonstrated that supplementation of probiotics, prebiotics, and synbiotics can be effective for PCOS. Specifically, probiotics showed promise in improving certain PCOS-related factors such as BMI, FPG, and lipid profiles. On the other hand, synbiotics were found to be less impactful than probiotics in influencing measurements related to body size, lipid profiles, and glucose regulation. Further research into the efficacy of prebiotics on PCOS-related parameters is necessary. However, a comprehensive meta-analysis suggested that prebiotics have favorable effects on body size, FPG, and C-reactive protein (CRP).[29,30]

The gut microbiota, such as Bifidobacteria and Lactobacillus, are often called "beneficial bacteria" because of their health-promoting characteristics. An increase in Lactobacillus in the gut may lead to the production of SCFA, which can improve gut health, enhance gut barrier function, and decrease the movement of bacterial endotoxins through the gut wall. These endotoxins can cause inflammation and insulin resistance. Studies have demonstrated that probiotic supplementation with *Lactobacillus casei Shirota* (LcS) can prevent insulin resistance induced by high-fat overfeeding in humans. Given the positive impact of Lactobacillus on insulin resistance, which is linked to PCOS, the current research used Lactobacillus transplantation and fecal microbiota transplantation (FMT) to treat PCOS rats. The outcomes indicated that treating PCOS rats with Lactobacillus transplantation and FMT reduced androgen levels in the blood, improved estrous cycles, and normalized ovarian functions. These findings suggest that FMT and Lactobacillus transplantation could effectively treat PCOS in rats.[11]

The first meta-analysis examining the dietary fiber intake in PCOS patients revealed that PCOS women consumed notably less dietary fiber than non-PCOS women, according to Leung et al.[3] One of the vital physiological functions of dietary fiber in humans is its ability to positively influence the microbial ecosystem by directly interacting with gut microbes, thereby boosting the production of essential microbial metabolites.[15,16,31] On the other hand, inadequate consumption of dietary fiber not only leads to a progressive decline in microbial variety and changes microbial metabolism to favor less beneficial substances and break down protective mucin, which can be damaging to the host. Several studies have indicated a notable reduction in gut microbiome diversity in individuals with PCOS. Nevertheless, it is not yet clear whether insufficient dietary fiber contributes to variations in the microbial community.[4,32–34]

Integrating dietary fiber into the diet could influence PCOS by regulating microbial metabolites and short-chain fatty acids (SCFA). These substances are significant microbial products formed in the colon through the breakdown of dietary fiber by intestinal bacteria. They are crucial in controlling host metabolism, the immune system, and cell growth. Reduced fiber intake could decrease the production of these metabolites, particularly SCFA, affecting overall health and well-being. Given that women with PCOS consume less dietary fiber, it is unclear whether their SCFA production is reduced. Additionally, whether increasing SCFA levels through dietary fiber intake or supplementation could benefit PCOS patients requires further investigation.[16,35]

CONCLUSION

PCOS is a multifactorial disorder influenced by significant internal factors, such as gut microbiota and external environmental factors. Many studies have indicated a link between gut microbiota and PCOS, but their exact cause-and-effect relationship is still uncertain. Research has shown that the connection between gut microbiota, bile acid, and interleukin-22 (IL-22) is being studied as a potential mechanism underlying the development of PCOS. Specific types of gut microbiota may trigger oxidative stress and inflammatory responses in the mononuclear cells of women with PCOS by affecting dietary factors. Probiotics have demonstrated promising effects on certain PCOS-related factors such as BMI, FPG, and lipid profiles. However, further research is necessary to explore additional potential mechanisms.

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DECLARATIONS

1. Yosep Sutandar conceptualized the study, designed the review methodology, and supervised the overall writing process. Seftiwan Pratami Djasfar contributed to the structured literature search, data extraction, and drafting of the manuscript. Wilfadri Putra Jonesti was responsible for manuscript editing. Desi Purwaningsih contributed to the literature review on biochemical interactions and critical revision of the final manuscript. All authors have read and approved the final version of the manuscript.
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