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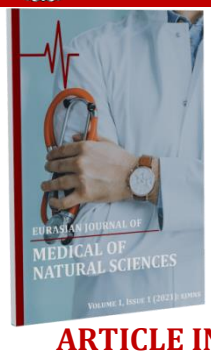


# POLYCYSTIC OVARY SYNDROME: MODERN ASPECTS OF PATHOGENESIS, DIAGNOSIS, AND THERAPY

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*Polycystic ovary syndrome; pathogenesis; diagnosis; treatment; infertility; reproductive endocrinology; personalized medicine.*

**ABSTRACT**

***Background:*** *Polycystic ovary syndrome (PCOS) is among the most prevalent endocrine disorders in women of reproductive age, with a global prevalence ranging from 8% to 13%, depending on diagnostic criteria. It is a multifactorial condition associated with hormonal imbalance, insulin resistance, chronic inflammation, and genetic predisposition.*

***Objective:*** *To summarize current evidence on the pathogenesis, diagnostic approaches, and treatment strategies for PCOS, with an emphasis on personalized medicine and emerging therapeutic directions.*

***Methods:*** *A systematic literature review was conducted in accordance with PRISMA guidelines. Sources included PubMed, Google Scholar, and eLibrary. Inclusion criteria encompassed peer-reviewed studies focused on reproductive endocrinology in women aged 18 to 45 years, using established diagnostic criteria (NIH, Rotterdam, AE-PCOS). Studies of low methodological quality, small sample size, or without full-text availability were excluded. Fifty high-quality publications were selected for final analysis.*

***Results:*** *PCOS is recognized as a systemic disorder necessitating a multidisciplinary and individualized treatment approach. Conventional management includes pharmacological therapies such as combined oral contraceptives, insulin sensitizers, and ovulation induction agents. Lifestyle modifications and assisted reproductive technologies (ART) significantly enhance reproductive outcomes. Emerging evidence suggests a critical role for genetic variants, gut microbiota alterations, and novel biomarkers in advancing diagnostic accuracy and tailoring treatment.*

## INTRODUCTION

Polycystic ovary syndrome ranks as one of the most common endocrine disorders affecting women of reproductive age, with global prevalence estimates ranging from 8% to

13%, depending on the diagnostic criteria used [1–2]. This complex condition is characterized by disruptions in the menstrual cycle, elevated androgen levels, and the presence of multiple ovarian cysts, and it stands as a leading cause of anovulatory infertility [3–4]. Beyond its effects on reproductive function, polycystic ovary syndrome (PCOS) is a multifaceted systemic disorder often linked to metabolic imbalances such as insulin resistance, obesity, type 2 diabetes mellitus, and a greater risk of cardiovascular disease [5–6]. Moreover, women with PCOS are more prone to developing depression and anxiety, highlighting the condition's complex and multidisciplinary character [7–8]. Epidemiological studies show that PCOS prevalence varies based on ethnic background, diagnostic standards, and geographical location. According to the World Health Organization (WHO), approximately 70% of women with PCOS remain undiagnosed and untreated [9-10]. This is largely due to the high variability in clinical manifestations and the lack of a universally accepted diagnostic algorithm. Furthermore, PCOS has a significant impact on female reproductive health. It is one of the primary causes of anovulatory infertility, with evidence suggesting that up to 80% of chronic anovulation cases are attributed to this syndrome [11-12].

From an economic perspective, PCOS also poses a substantial burden on healthcare systems, as its diagnosis and management require considerable financial resources. This is especially significant due to the common coexistence of comorbid conditions like diabetes and cardiovascular disorders often observed in individuals with the syndrome [13–14]. The objective of this review is to analyze current data on the pathogenesis, diagnosis, and treatment of polycystic ovary syndrome while also evaluating emerging therapeutic approaches that may improve patient outcomes.

## MATERIALS AND METHODS

To compile this systematic review, an extensive search of electronic databases—including PubMed, Google Scholar, and eLibrary—was performed. The systematic review followed the PRISMA framework to uphold clarity and precision in the research methodology.

### A. Inclusion Criteria

- Publications in English and Russian that present original research, meta-analyses, or systematic reviews.
- Studies related to reproductive endocrinology, focusing on key aspects of PCOS pathogenesis, diagnosis, and therapy.
- Inclusion of adult women (18–45 years old) in the study population.
- Use of validated diagnostic criteria (NIH, Rotterdam criteria, AE-PCOS) and objective treatment assessment methods.
- Articles published no later than January 2024.

### B. Exclusion Criteria

- Articles with low levels of evidence, including expert opinions without empirical data and uncontrolled studies.
- Non-peer-reviewed publications (e.g., preprints without subsequent journal publication).
- Studies with small sample sizes (<30 patients) or insufficient statistical power.

- Research not directly related to reproductive endocrinology, such as studies on PCOS in adolescents, men, or animal models.
- Duplicate publications and articles not available in full-text format.

**C. Quality Assessment**

To ensure the quality and reliability of the selected studies, the following methodological tools were employed:

1. GRADE (Grading of Recommendations, Assessment, Development, and Evaluations) – to assess the strength of the evidence.
2. CASP (Critical Appraisal Skills Programme) – to evaluate study reliability and methodological quality.
3. ROBINS-I (Risk Of Bias In Non-Randomized Studies of Interventions) – to analyze bias risk in non-randomized studies.
4. Cochrane Risk of Bias Tool – to identify potential sources of bias in randomized controlled trials (RCTs).

The initial search retrieved:

- 85 publications from PubMed,
- 73 articles from eLibrary,
- 102 publications from Google Scholar.

After removing duplicates and excluding studies that did not meet the inclusion criteria, 50 high-quality studies were selected for final analysis.

**PATHOGENESIS OF PCOS**

Among women of childbearing age, PCOS is frequently identified as a leading hormonal imbalance, with its prevalence worldwide reported to be between 6% and 18%, based on varying diagnostic standards. Although prevalence varies by ethnicity—reaching up to 20% in South Asian populations—up to 70% of women with PCOS remain undiagnosed, underscoring the need for better screening and standardized diagnostic approaches.

PCOS is a multifactorial disorder shaped by genetic susceptibility, endocrine/metabolic dysregulation, and environmental factors. Genetic predisposition is evident in family-based studies showing higher PCOS risk among first-degree relatives. Polymorphisms in genes such as FSHR (reduced FSH sensitivity), INS (insulin resistance), CYP11A1 (altered androgen synthesis), and LHCGR (heightened LH responsiveness) have been linked to PCOS, although no single gene fully predicts its onset [15-16].

One of the central hormonal disturbances in PCOS is hyperandrogenism, which involves increased levels of androgens such as testosterone, androstenedione, and DHEA-S. This imbalance interferes with the ovulatory process and leads to clinical symptoms like excessive hair growth (hirsutism) and acne [17]. Additional hormonal imbalances include dysregulated hypothalamic-pituitary-ovarian (HPO) axis function, often presenting as an increased LH/FSH ratio, and excessive adrenal androgen secretion [18-19].

Insulin resistance is observed in about 50–70% of individuals with PCOS and is a significant factor in worsening hyperandrogenism by promoting excessive ovarian androgen synthesis and decreasing levels of sex hormone-binding globulin (SHBG). This metabolic

imbalance is also associated with obesity, glucose intolerance, type 2 diabetes, and an elevated risk of cardiovascular complications [20–21]. Lifestyle factors—high-calorie diets, physical inactivity, and stress—can exacerbate both endocrine and metabolic abnormalities [22]. Moreover, potential environmental factors, including exposure to endocrine-disrupting chemicals, may influence receptor function and worsen PCOS symptoms [23-24].

Recent findings highlight the role of chronic inflammation and oxidative stress in PCOS, noting elevated inflammatory markers (IL-6, TNF-α, CRP) and increased production of reactive oxygen species (ROS). These processes disrupt folliculogenesis, perpetuate metabolic dysfunction, and heighten cardiovascular risks [25-26].

In summary, PCOS pathogenesis involves a complex interplay of genetic predisposition, hormonal dysregulation, insulin resistance, and chronic inflammation, ultimately impacting both reproductive and metabolic health. Continued research on these interlinked mechanisms is vital for refining diagnostic criteria and developing more individualized therapeutic strategies.

Polycystic ovary syndrome (PCOS) is diagnosed based on clinical, biochemical, and ultrasound findings.

Due to its heterogeneity, several criteria have been developed:

1. **NIH Criteria (1990)**

For a diagnosis of PCOS to be confirmed, the following conditions must be met: the presence of oligo- or anovulation, typically manifested through irregular or absent menstrual cycles; clinical or biochemical signs of hyperandrogenism, such as hirsutism, acne, or elevated androgen levels; and the exclusion of other endocrine disorders, including hyperprolactinemia, congenital adrenal hyperplasia, and Cushing's syndrome. A notable limitation of this diagnostic approach is the lack of ultrasound assessment, which may result in underdiagnosis of the syndrome [27–29].

2. **Rotterdam Criteria (2003)**

The diagnostic criteria for PCOS have been expanded to include polycystic ovarian morphology identified via ultrasound. The diagnosis of PCOS is established when at least two of the following criteria are met: irregular or absent ovulation (oligo- or anovulation), clinical signs or laboratory confirmation of elevated androgen levels, and ultrasound features indicative of polycystic ovaries—defined by the presence of 12 or more follicles measuring 2–9 mm in diameter or an ovarian volume exceeding 10 $cm^3$. This broader definition may result in higher prevalence rates, as it allows for the diagnosis of PCOS even in cases lacking overt signs of hyperandrogenism [29–30].

**3.AE-PCOS Society Criteria (2006)**

This diagnostic approach emphasizes hyperandrogenism as the central feature of PCOS. To establish the diagnosis, clinical or biochemical evidence of androgen excess is mandatory, accompanied by either oligo- or anovulation or the presence of polycystic ovarian morphology. These more stringent criteria aim to clearly differentiate PCOS from other conditions that share overlapping characteristics [31–32].

**4.Phenotypic Variations**

PCOS is categorized into four distinct phenotypes (A–D) based on the combination of clinical features, each associated with specific reproductive and metabolic profiles [33–34].

- **Phenotype A (Classic Form):** Characterized by oligo- or anovulation, elevated androgen levels, and polycystic ovarian morphology; this type is linked to the most pronounced metabolic disturbances.
- **Phenotype B (Anovulatory Hyperandrogenic):** Shares similar metabolic risks with phenotype A but lacks sonographic evidence of polycystic ovaries.
- **Phenotype C (Ovulatory with Hyperandrogenism):** Ovulatory function is retained despite androgen excess, and it is typically associated with a lower likelihood of infertility.
- **Phenotype D (Non-Hyperandrogenic):** Often diagnosed during adolescence, this variant presents the mildest symptoms and lacks biochemical or clinical signs of androgen excess.

**Key Clinical Manifestations and Associated Risks**

Polycystic ovary syndrome (PCOS) exerts a multifaceted impact on reproductive, metabolic, and psychological health. It commonly leads to menstrual irregularities and ovulatory disturbances, including oligomenorrhea, amenorrhea, and anovulation. Elevated androgen levels contribute to clinical manifestations such as excessive hair growth (hirsutism), acne, and androgenic alopecia [33]. Metabolic complications are commonly seen in PCOS, with obesity impacting 40–70% of affected individuals, insulin resistance occurring in 50–70%, and a heightened likelihood of developing type 2 diabetes and lipid metabolism disorders [34]. On the reproductive front, women with PCOS frequently encounter issues such as infertility, repeated miscarriages, and an increased risk of gestational diabetes [22]. In addition, the condition is frequently associated with psychological concerns, including anxiety, depression, and diminished overall quality of life [35].

**Diagnostic Workup**

The diagnostic approach to PCOS involves a combination of clinical assessment, laboratory testing, imaging, and exclusion of alternative conditions. Clinical evaluation focuses on identifying menstrual disturbances, signs of hyperandrogenism, obesity, and relevant family history. Hormonal analysis typically includes measurements of total and free testosterone, DHEA-S (to assess adrenal contributions), the LH/FSH ratio, and prolactin levels to rule out hyperprolactinemia. Anti-Müllerian hormone (AMH) is often elevated (≥4.5 ng/mL), indicating a higher number of antral follicles. Metabolic evaluation involves fasting glucose or oral glucose tolerance testing, insulin levels with HOMA-IR calculation, and a lipid panel. Transvaginal ultrasound plays a critical role in reinforcing the diagnosis of PCOS. As defined by the Rotterdam criteria, polycystic ovarian morphology is characterized by either the presence of 12 or more follicles ranging from 2 to 9 mm in diameter or an ovarian volume greater than 10 $cm^3$. However, with advancements in imaging resolution, the 2018 revised guidelines recommend raising the follicle count threshold to 20 or more [36]. It is also essential to exclude other endocrine disorders that can present with symptoms similar to PCOS, such as hyperprolactinemia, congenital adrenal hyperplasia, Cushing's syndrome, and thyroid

dysfunctions. Although the Rotterdam criteria are the most widely applied in clinical settings, the most accurate and effective diagnostic approach for PCOS continues to be a matter of ongoing discussion. Phenotyping helps tailor treatment: classic PCOS (phenotype A) often warrants aggressive metabolic management, while ovulatory PCOS (phenotype C) may have fewer complications. Ongoing research seeks to refine diagnosis through novel biomarkers (e.g., microRNAs) and advanced imaging techniques, potentially improving early detection and personalized management of PCOS.

**V.** Management of polycystic ovary syndrome (PCOS) focuses on correcting hyperandrogenism, menstrual irregularities, infertility, and metabolic dysfunctions. The choice of therapy depends on age, reproductive goals, and the severity of metabolic and hormonal disturbances [37].

1. **Pharmacological Treatment**

Hormonal treatment options include combined oral contraceptives (COCs) and anti-androgens. COCs are considered the first-line therapy for women not aiming to conceive, as they help normalize menstrual cycles and reduce androgen levels [38]. The most effective combinations typically involve ethinylestradiol paired with progestins that have anti-androgenic properties, such as cyproterone acetate or drospirenone. In cases of pronounced hirsutism, additional use of anti-androgen agents like spironolactone, flutamide, or finasteride may be warranted. However, concurrent administration of COCs is essential to avoid the risk of fetal masculinization in case of pregnancy. Insulin-sensitizing agents such as metformin and inositols play a key role in managing PCOS-related metabolic disturbances. Metformin, usually administered in daily doses ranging from 500 to 2000 mg, enhances insulin sensitivity, helps decrease circulating androgen levels, and may aid in restoring regular ovulatory cycles. For patients who are unable to tolerate metformin, inositols—specifically myo-inositol and D-chiro-inositol—serve as an alternative, also contributing to reductions in insulin and androgen levels.

Ovulation induction therapies are essential for treating anovulatory infertility. Clomiphene citrate is typically the first-line pharmacologic option, showing effectiveness in approximately 70–80% of cases. Letrozole, an aromatase inhibitor, is another viable choice, often demonstrating superior outcomes in women with higher body mass index and carrying a reduced likelihood of multiple gestations. In cases where oral agents are unsuccessful, treatment may escalate to gonadotropin therapy or surgical interventions such as laparoscopic ovarian drilling.

**Non-Pharmacological Approaches**

Lifestyle interventions, including dietary adjustments and physical activity, are fundamental in the management of PCOS. Achieving a weight loss of just 5–10% can lead to the resumption of ovulation in up to 70% of obese women affected by the syndrome. Consistent exercise further contributes by enhancing insulin sensitivity and decreasing androgen levels.

In cases of severe obesity, metabolic (bariatric) surgery may be considered for individuals with a body mass index (BMI) exceeding 35. Such surgical interventions have been shown to improve reproductive outcomes, reduce hyperandrogenic symptoms, and lower associated

metabolic risks [39]. Following surgery, careful monitoring of vitamin and micronutrient levels is essential to prevent deficiencies.

**Reproductive Technologies**

If initial ovulation induction treatments like clomiphene citrate or letrozole do not yield successful outcomes, therapeutic strategies may escalate to include gonadotropin administration or assisted reproductive techniques such as in vitro fertilization (IVF). To reduce the likelihood of ovarian hyperstimulation syndrome (OHSS), mild stimulation protocols are typically recommended in these cases [40]. The management of polycystic ovary syndrome (PCOS) is based on several key principles. For women not aiming to conceive, combined oral contraceptives (COCs) remain the primary therapeutic option, helping regulate menstrual cycles and reduce androgen levels. To address insulin resistance, commonly associated with PCOS, agents such as metformin or inositols are widely used. When fertility is a concern, clomiphene citrate and letrozole serve as first-line medications for ovulation induction. Lifestyle modifications, particularly weight reduction, are essential for improving both endocrine and metabolic outcomes. In cases where standard treatments fail to induce ovulation, assisted reproductive technologies like in vitro fertilization (IVF) may be considered.A multifactorial and personalized approach, targeting hormonal, metabolic, and reproductive aspects, is essential for successful PCOS management.

**FUTURE DIRECTIONS**

Despite advances, optimal PCOS management and pathophysiological understanding continue to evolve. Current research highlights the following areas:

1. **Novel PCOS Biomarkers**

Anti-Müllerian hormone (AMH) is currently utilized in clinical practice, though its full potential continues to be investigated. MicroRNAs (miRNAs) are emerging as important regulators of genes linked to androgen excess and metabolic dysfunction. Adipokines—such as leptin, adiponectin, and visfatin—serve as indicators of obesity and insulin resistance. Additionally, exosomes containing specific proteins and RNA molecules hold promise for enhancing early diagnosis and improving risk assessment strategies in PCOS patients [41].

**Genetic Research**

Current research is actively investigating genetic polymorphisms—such as those in CYP11A1, FSHR, and INSR genes—as well as conducting genome-wide association studies (GWAS) to identify novel genes linked to PCOS development [42]. The overarching objective is to create comprehensive genetic panels that would enable personalized therapeutic approaches in the future.

2. **Personalized Therapy**

Pharmacogenetic approaches aim to forecast individual responses to treatments such as metformin or anti-androgens by analyzing genetic variations. Modulation of the gut microbiota through the use of probiotics and prebiotics is being explored as a strategy to enhance insulin sensitivity and mitigate systemic inflammation. Emerging therapies include next-generation anti-androgens, such as selective androgen receptor modulators, which offer targeted

hormonal regulation. Additionally, precision nutrition focuses on tailoring dietary interventions to an individual's unique genetic and metabolic profile for optimized outcomes.

3. **Microbiome and PCOS**

Gut microbiota imbalance, or dysbiosis, observed in PCOS may contribute to heightened insulin levels and chronic inflammation. Therapeutic use of probiotics and prebiotics holds promise in restoring microbial equilibrium, which could lead to improvements in both metabolic parameters and androgen-related symptoms [43].

4. **Artificial Intelligence (AI)**

Machine learning has the potential to improve the interpretation of ultrasound imaging and forecast complications associated with PCOS, thereby supporting more personalized and effective treatment strategies [44]. Overall, integrating emerging biomarkers, genetic insights, microbiome therapy, and AI-driven technologies holds promise for more precise, effective, and personalized management of PCOS.

**DISCUSSION**

Polycystic ovary syndrome (PCOS) remains a complex and heterogeneous condition at the intersection of reproductive endocrinology and metabolic dysfunction, warranting a nuanced and multifactorial approach to its diagnosis and management. The present review synthesizes contemporary insights into its pathogenesis, clinical presentation, and evolving therapeutic landscape, emphasizing the need for personalized and evidence-based interventions [45].

The pathophysiological underpinnings of PCOS are increasingly recognized as multifaceted, involving genetic predisposition, hormonal imbalances, insulin resistance, and systemic inflammation. Recent advances in genomics and epigenetics underscore the involvement of multiple gene polymorphisms, including those in *CYP11A1*, *FSHR*, and *INSR*, which contribute to androgen excess, altered folliculogenesis, and metabolic dysfunction. However, the absence of a unifying genetic marker reflects the polygenic and environmentally modulated nature of PCOS, complicating early detection and uniform diagnosis [46].

The persistent diagnostic ambiguity associated with PCOS stems from the use of divergent classification systems—NIH, Rotterdam, and AE-PCOS—each emphasizing different pathophysiological features. While the Rotterdam criteria remain the most widely utilized, their broader scope may inadvertently inflate prevalence estimates and include phenotypes with distinct metabolic risks. This variability complicates cross-study comparisons, risk stratification, and therapeutic decision-making. The incorporation of refined phenotyping based on biochemical, clinical, and imaging features offers promise in aligning diagnosis with targeted management approaches [47].

Clinically, PCOS presents along a spectrum ranging from classic phenotypes characterized by anovulation and hyperandrogenism to milder forms with predominantly metabolic manifestations. These phenotypic differences are critical, as they predict the degree of metabolic derangement, fertility challenges, and psychological impact. Notably, the substantial burden of comorbid conditions—including obesity, dyslipidemia, insulin resistance, and psychological distress—highlights the systemic nature of the syndrome and underscores the importance of holistic patient care [48].

Therapeutically, current strategies focus on ameliorating hyperandrogenism, restoring ovulatory function, and mitigating metabolic complications. Combined oral contraceptives and insulin-sensitizing agents remain the cornerstone of pharmacologic therapy for non-fertility-seeking patients, whereas ovulation induction agents like clomiphene citrate and letrozole are pivotal in fertility management. However, therapeutic efficacy is highly variable, and resistance to standard treatments remains a challenge, particularly in obese or metabolically compromised phenotypes.

The emerging paradigm of personalized medicine offers substantial potential to optimize PCOS management. Pharmacogenomics, for example, may soon enable the prediction of individual responses to agents such as metformin or anti-androgens, minimizing trial-and-error prescribing. In parallel, the exploration of gut microbiota dysbiosis in PCOS has opened avenues for microbiome-targeted therapies, with early evidence suggesting favorable metabolic and hormonal modulation following probiotic or prebiotic interventions [49].

Additionally, recent advances in biomarker discovery—including the use of anti-Müllerian hormone (AMH), adipokines, microRNAs, and exosomal content—may enhance diagnostic precision and allow for early risk stratification. These molecular signatures could also facilitate treatment monitoring and response evaluation. Integrating such biomarkers into routine clinical workflows, however, will require rigorous validation in diverse populations.

Artificial intelligence (AI) and machine learning applications in reproductive medicine are also gaining traction. AI-based models can assist in interpreting complex diagnostic data, predicting treatment responses, and customizing stimulation protocols for assisted reproductive technologies (ART), thereby increasing the efficiency and success rates of interventions in women with PCOS.

Nevertheless, significant gaps remain. Longitudinal studies are needed to assess the long-term safety and efficacy of novel treatments, including selective androgen receptor modulators and microbiota-directed therapies. Moreover, greater emphasis must be placed on understanding PCOS in diverse ethnic populations, where phenotypic expression and treatment responses may differ substantially. Furthermore, adolescent and perimenopausal PCOS phenotypes remain under-investigated, representing crucial windows for early intervention and disease modification [49–50].

In conclusion, while strides have been made in unraveling the complexities of PCOS, ongoing research is essential to refine diagnostic algorithms, stratify risk more effectively, and implement individualized care strategies. The convergence of molecular diagnostics, AI technologies, and personalized therapeutics heralds a new era in PCOS management, with the potential to significantly improve reproductive, metabolic, and psychosocial outcomes for affected women.

**CONCLUSION**

Polycystic ovary syndrome (PCOS) is a prevalent endocrine disorder among women of reproductive age, with consequences extending beyond infertility to include metabolic dysfunction and psychological comorbidities. Contemporary evidence underscores its nature as a complex multisystem condition, underpinned by hyperandrogenism, insulin resistance,

chronic inflammation, and genetic factors. Although current diagnostic frameworks rely on clinical and biochemical criteria, emerging biomarkers—such as anti-Müllerian hormone (AMH), microRNAs, and extracellular vesicles—hold promise for enhancing early detection and classification.

Effective PCOS management requires an individualized, multimodal approach integrating pharmacologic, lifestyle, and reproductive interventions. Combined oral contraceptives and antiandrogens remain first-line therapies for menstrual irregularities and hyperandrogenism. Insulin-sensitizing agents, including metformin and inositols, contribute to metabolic improvement and ovulatory restoration. For women seeking conception, ovulation induction with clomiphene citrate or letrozole is recommended, while assisted reproductive technologies (ART), such as controlled ovarian stimulation and in vitro fertilization (IVF), are indicated in resistant cases.

Lifestyle modification is a cornerstone of treatment. Evidence indicates that modest weight loss (5–10%) can significantly improve hormonal and ovulatory function. Nutritional optimization and regular physical activity are essential for managing obesity and mitigating long-term metabolic risks. In select cases of morbid obesity, bariatric surgery may be considered.

Given the heterogeneous and systemic nature of PCOS, interdisciplinary collaboration involving gynecologists, endocrinologists, nutritionists, cardiologists, and mental health professionals is crucial. Such an approach is vital to prevent long-term complications, including type 2 diabetes, cardiovascular disease, and mood disorders.

Future research priorities include the identification of genetic determinants of PCOS, exploration of the gut and vaginal microbiome's role in pathophysiology, and the application of artificial intelligence for improved diagnosis and individualized risk assessment. These directions are anticipated to facilitate precision medicine strategies and optimize reproductive and metabolic outcomes in affected women.

PCOS represents a significant clinical challenge necessitating comprehensive, personalized care. Advances in molecular diagnostics, therapeutic innovation, and interdisciplinary models are key to improving patient outcomes and long-term health trajectories.

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**CONFLICT OF INTEREST**

The authors declare no conflict of interest.

**Conclusion:** PCOS remains a complex reproductive and metabolic disorder. Future treatment paradigms are likely to integrate microbiome-targeted therapies, selective anti-androgens, and AI-based diagnostic models. Advances in genomics and precision medicine hold promise for improving long-term reproductive outcomes in affected women.

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