



A Brief Review on Polycystic Ovarian Syndrome (PCOS) and Related Toxicity

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ABSTRACT

Polycystic ovarian syndrome (PCOS) is a common hormonal disorder in women of reproductive age, characterized by ovarian dysfunction and chronic anovulation. It commonly afflicts 6 and 10% of women of reproductive age. The exact cause of PCOS is not fully understood. Diagnosis is based on three features: oligomenorrhea, hyperandrogenism, and polycystic ovaries. Hyperandrogenism may indicate reproductive and metabolic complications. Obesity can worsen symptoms, which vary across ethnic groups. PCOS may also be difficult to diagnose around menarche and menopause. Its development involves ovarian dysfunction, insulin resistance, and abnormal gonadotropin regulation. PCOS causes multiple health problems which display similarities to metabolic syndrome. Women with PCOS frequently have lipid abnormalities which result in higher triglyceride and low-density lipoprotein (LDL) levels and lower high-density lipoprotein (HDL) levels. The diagnostic criteria require standard physical examination results which include obesity and acanthosis nigricans and acne together with laboratory measurements of hyperandrogenism that show increased luteinizing hormone and decreased sex hormone-binding globulin and elevated free testosterone and androstenedione and dehydroepiandrosterone sulfate levels to confirm the diagnosis. An ultrasound of the ovaries may reveal the many cysts. The oral contraceptive medication decreases androgen and LH levels to treat hirsutism and acne while establishing regular menstruation cycles. To achieve considerable improvements in hirsutism symptoms patients require antiandrogens in most situations. Metformin belongs to a new group of drugs known as insulin sensitizers which treat PCOS. They enhance the unfavorable metabolic profile of PCOS patients by increasing insulin sensitivity and lowering insulin level.

Keywords: Polycystic ovarian Syndrome, hyperandrogenism, metabolic syndrome, insulin sensitizers, oral contraceptives.

INTRODUCTION

Polycystic ovary syndrome (PCOS) creates various health problems which specifically affect female endocrine reproductive health. Females aged 18 to 44 are affected. A chronic hormonal imbalance leads to complications which include multiple cysts and irregular menstruation thus resulting in infertility for females. The research has identified multiple genes which scientists believe will contribute to the development of PCOS⁶.

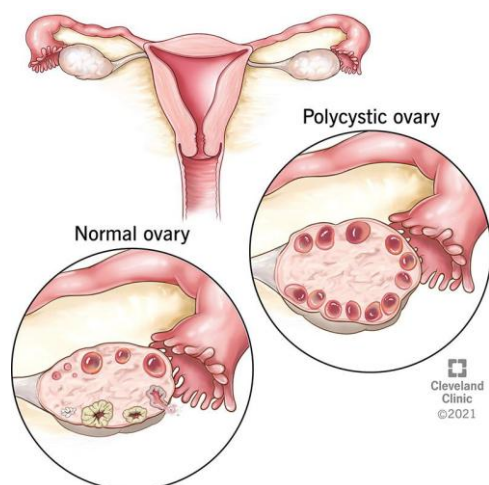


Figure 1: Polycystic ovary in comparison with normal ovary⁹

The condition produces ovarian antral follicle cysts because it results in female hormone levels becoming unbalanced. A cyst is an egg-containing water-filled sac that should have been released for potential fertilization. The transformation

of the egg into a "functional cyst" stops ovulation from occurring. The menstrual cycle disruption caused by blocked ovulation leads to the condition known as amenorrhea. The condition which results in multiple ovarian follicles developing cysts occurs through a hormonal imbalance known as PCOS⁷. Postmenopausal women with PCOS show increased risk for developing metabolic and cardiovascular diseases which persists even after their hyper androgenic symptoms diminish at menopause⁸.

Epidemiology:

1. Worldwide research shows that approximately 4% to 13% of women between the ages of 15 and 49 years old experience PCOS. The World Health Organization estimates show that up to 70% of cases remain undetected according to their prevalence estimates which range between 6% and 13%¹⁰.
2. The general findings typically present the most prevalent rates ranging between 4 to 8%¹¹.
3. The study shows that the US population has a PCOS prevalence which ranges from 6.5% to 8% according to European research and health experts estimate the condition affects 4% to 12% of the population¹².
4. Research using ultrasound shows that between 21% and 22% of women selected randomly exhibit polycystic ovaries which does not result in complete diagnostic criteria fulfilment for PCOS¹³
5. Research studies from India show that their results show widespread disease rates which range between 3.7%



and 22.5% because of both urban and rural populations and different diagnostic methods used to measure the disease. The prevalence rates of adolescence in urban areas reach 22.5% according to Rotterdam standards while rural areas show a prevalence rate of approximately 6%¹⁴

Etiology:

1. Increased androgens: Menstrual periods become irregular when ovaries are unable to release eggs due to elevated testosterone that is androgens.
2. Genetic: The pathophysiology of PCOS involves a large number of genes.
3. Insulin resistance: High blood glucose levels can be a result of insulin resistance. Ovaries release more male hormones when insulin levels are elevated, which exacerbates other PCOS symptoms. A significant contributor to insulin resistance is obesity.
4. Inflammation: The bodies of women with PCOS are more prone to inflammation. Elevated androgen levels can result from increased inflammation¹⁵.

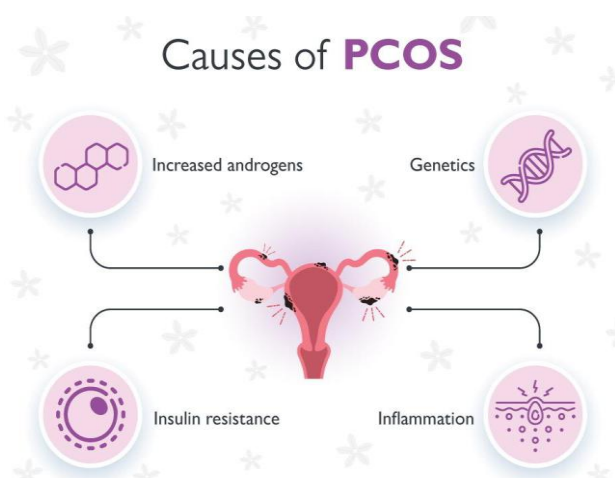


Figure 2: Causes of PCOS¹⁵

Pathophysiological mechanisms

● Resistance to Insulin and Hyperinsulinemia:

Approximately 70% of women with PCOS have insulin resistance, regardless of their level of obesity¹⁶.

Elevated insulin levels, or hyperinsulinemia, worsen the ovaries' ability to produce androgen, mainly by making ovarian theca cells more sensitive to stimulation from luteinizing hormone (LH), which increases androgen synthesis^{16,17,21}.

Additionally, insulin increases free androgen levels by reducing the liver's production of sex hormone-binding globulin (SHBG)²¹.

● Hyperandrogenism

The overexpression of steroidogenic enzymes, particularly cytochrome P450c17, by theca cells in the ovaries results in

an excess of androgen synthesis (testosterone, androstenedione)^{16,17,21}.

Increased testosterone interferes with healthy follicular growth, resulting in anovulation and halted follicle growth^{19,21}.

● Hypothalamic-Pituitary-Ovarian (HPO) Axis Dysfunction

Increased LH and comparatively lower follicle-stimulating hormone (FSH) secretion are the results of altered gonadotropin-releasing hormone (GnRH) secretion^{16,19,21}

Low FSH degrades granulosa cell activity, decreasing aromatization of androgens to estrogens and impeding proper follicle maturation and ovulation, whereas elevated LH encourages theca cells to create more androgens^{19,21}

● Ovarian Dysfunction

Multiple immature antral follicles occupy the ovary, giving it the distinctive polycystic appearance^{19,21}.

Granulosa cells may luteinize too soon as a result of too much testosterone and insulin, which would further hinder ovulation¹⁶.

● Chronic Low-Grade Inflammation

Irrespective of obesity, women with PCOS frequently exhibit elevated inflammatory markers, such as C-reactive protein, indicating a role for chronic inflammation in the condition¹⁶.

● Adipose Tissue and Estrogen Production

Peripheral conversion of androgens to estrogens (mostly estrone) in adipose tissue is elevated in obese women with PCOS, which leads to hormonal imbalance and unopposed endometrial estrogenic stimulation^{16,12}

● Other Contributing Factors

A greater quantity of halted follicles in the ovaries is reflected in elevated anti-Müllerian hormone (AMH) levels¹⁹.

The development and variability of PCOS are influenced by both genetic and environmental variables, such as prenatal androgen exposure and postnatal obesity²⁰.

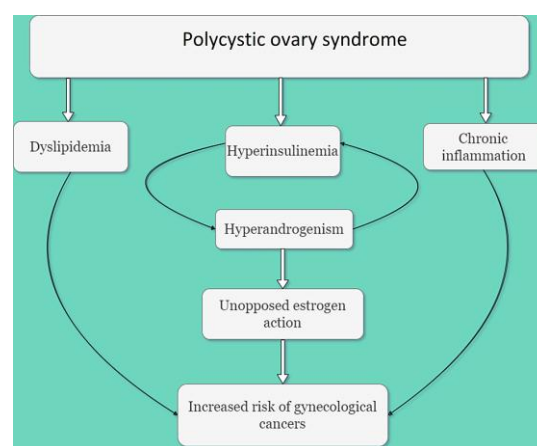


Figure 3: Pathophysiology of PCOS²¹.

Diagnosis

1. Pelvic exam:

Your doctor may examine your reproductive organs for growths, lumps, or other abnormalities during a pelvic exam²².

2. Blood test:

Tests on blood can determine hormone levels. Menstrual issues or androgen excess that mimics PCOS may be ruled out by this testing. Other blood tests, like fasting triglyceride and cholesterol readings, may be performed on you. A glucose tolerance test can gauge how your body reacts to glucose, or sugar²².

3. Ultrasound:

The thickness of your uterine lining and the appearance of your ovaries can both be examined using an ultrasound. Your vagina is exposed to a transducer, which resembles a wand. On a computer screen, the sound waves that the transducer produces are converted into visuals²².

Complications

- Infertility
- Gestational diabetes or pregnancy-induced high blood pressure
- Miscarriage or premature birth
- Non-alcoholic steatohepatitis — a severe liver inflammation caused by fat build-up in the liver
- Metabolic syndrome — a cluster of conditions including high blood pressure, high blood sugar, and unhealthy cholesterol or triglyceride levels that significantly increase your risk of heart and blood vessel (cardiovascular) disease
- Type 2 diabetes or prediabetes
- Sleep apnea
- Depression, anxiety and eating disorders
- Cancer of the uterine lining (endometrial cancer)

PCOS is frequently associated with obesity, which can exacerbate its consequences¹⁸.

Treatment

1. Lifestyle changes:

Insulin levels can be positively impacted by eating a balanced diet and keeping a healthy body weight⁹.

2. Oral contraceptives:

Oral contraceptives commonly known as birth control pills increases the regularity of your periods, lowers the androgen activity results from lowering androgen levels and reduces excessive hair growth and help get rid of acne²³.

3. Insulin sensitising agents:

These drugs increase the body's sensitivity to insulin and maintain more stable blood sugar levels. They reduces hair growth and get rid of acne, boosts your weight loss, results in reduced cholesterol levels, increases the frequency of your periods and slightly lessen PCOS-related infertility²³.

4. Anti-androgens

These drugs stop the body from producing androgens and reduce the action of them. Anti-androgens can help women with PCOS to reduce androgen levels, to diminish hair growth and to remove acne. Although they may lessen symptoms, anti-androgen drugs are not FDA-approved for the treatment of PCOS²³.

5. Acne treatment:

Common treatments for acne include retinoids, antibacterial agents, and antibiotics; the suggested course of action varies according to the severity of the acne. The remedies are available in a range of dosages and formats, including pills, creams, and gels; higher strength choices could need a prescription. Retinoids should not be used by those who are or want to become pregnant since they can result in congenital abnormalities²³.

Toxicity related to PCOS

1. Hormonal disruption

The term "hormonal toxicity" in PCOS mainly refers to the hormonal imbalance and disruption that the illness causes, which is frequently made worse by exposure to endocrine-disrupting drugs (EDCs). Gonadotropin-releasing hormone (GnRH) pulse production is accelerated in PCOS due to a well-known neuroendocrine malfunction that results in incorrect gonadotropin synthesis with increased luteinizing hormone (LH) and low follicle-stimulating hormone (FSH). The symptoms of PCOS are caused by this imbalance, which also results in excessive androgen synthesis by the ovarian theca cells, poor aromatization of androgens to estrogens in granulosa cells, and abnormal follicular development^{24,25}.

Hormonal toxicity in PCOS can be exacerbated by endocrine-disrupting substances such bisphenols, triclosan, and others that imitate or obstruct normal hormone functioning. These substances may exacerbate PCOS-related ovarian dysfunction and infertility by interfering with steroidogenesis, mitochondrial activity, and hormone metabolism in ovarian cells. For instance, exposure to triclosan has been demonstrated to impair ovarian cells' mitochondrial function and change the levels of progesterone and estradiol, which can have detrimental effects on the generation of steroid hormones and ovarian function²⁴.

2. Insulin resistance

In polycystic ovarian syndrome (PCOS), insulin resistance is characterized by a compromised cellular response to insulin, which results in hyperinsulinemia that worsens hyperandrogenism and metabolic problems. The term



"toxicity" frequently refers to detrimental effects from advanced glycation end products (AGEs) or nutrient overload^{26/27/28}.

3. Inflammation and oxidative stress

Inflammation and oxidative stress cause long-term imbalances that harm cells, worsen insulin resistance, and hinder reproductive function in polycystic ovarian syndrome (PCOS)^{29/30}.

4. Liver overload

Through altered hepatokines and inflammation, hepatic dysfunction contributes to PCOS, and genetic overlaps promote dyslipidaemia and insulin resistance. In certain situations, risks are independent of obesity and include the development of cirrhosis, fibrosis, and cardiovascular disease. Beyond enzymes, screening with ultrasonography or CK-18 is recommended, particularly for patients with hyperinsulinemia^{31/32/33}.

5. Gut dysbiosis

Increased intestinal permeability caused by gut dysbiosis in PCOS allows lipopolysaccharides (LPS) from gram-negative bacteria to enter the bloodstream, resulting in endotoxemia, persistent low-grade inflammation, insulin resistance, and hyperandrogenism^{34/35}.

CONCLUSION

Polycystic ovarian syndrome (PCOS) is known as a complex endocrine and metabolic condition which has major effects on the reproductive, metabolic, and psychological systems. Despite being common among women of reproductive age, its precise cause is still unknown and involves a number of factors, including genetic susceptibility, hormonal dysregulation, insulin resistance, chronic inflammation, and environmental factors such as endocrine-disrupting toxins. In addition to impairing menstrual health and fertility, PCOS raises the long-term risks of endometrial cancer, liver dysfunction, cardio metabolic illnesses, and mental health issues. The connection between PCOS and related toxicity, including oxidative stress, insulin resistance, hormonal toxicity, and gut dysbiosis, emphasizes the significance of early detection and all-encompassing treatment. While pharmacological methods like oral contraceptives, insulin sensitizers, and anti-androgens offer symptom-specific relief, lifestyle changes continue to be the mainstay of treatment. Targeted treatment of related toxicities, preventive measures, and tailored medicine must be the main emphasis of future therapeutic methods. Improved diagnosis, better treatment outcomes, and an all-around higher quality of life for those with PCOS will result from a deeper comprehension of the disease's pathophysiology, environmental triggers, and molecular mechanisms.

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REFERENCES

1. Polycystic ovary syndrome: An overview, diagnosis and treatment of pcos. Asian Journal of Pharmacy and Technology [Internet]. 2020;10(4). Available from: <https://www.indianjournals.com/ijor.aspx?target=ijor:ajpt&volume=10&issue=4&article=008>
2. Google Scholar [Internet]. Google.com. 2026. Available from: https://scholar.google.com/scholar?hl=en&as_sdt=0%2C5&q=pcos+pathophysiology&oq=pcos+patho#d=gs_qabs&t=1749009718083&u=%23p%3DSzEx4FNjO_OJ
3. Dumesic DA, Oberfield SE, Stener-Victorin E, Marshall JC, Laven JS, Legro RS. Scientific Statement on the Diagnostic Criteria, Epidemiology, Pathophysiology, and Molecular Genetics of Polycystic Ovary Syndrome. Endocrine Reviews [Internet]. 2015 Oct 1;36(5):487–525. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4591526/>
4. Diamanti-Kandarakis E, Papavassiliou AG, Kandarakis SA, Chrousos GP. Pathophysiology and types of dyslipidemia in PCOS. Trends in Endocrinology & Metabolism. 2007 Sep;18(7):280–5.
5. Harwood K, Vuguin P, DiMartino-Nardi J. Current Approaches to the Diagnosis and Treatment of Polycystic Ovarian Syndrome in Youth. Hormone Research in Paediatrics. 2007;68(5):209–17.
6. Ajmal N, Khan SZ, Shaikh R. Polycystic ovary syndrome (PCOS) and genetic predisposition: A review article. European Journal of Obstetrics & Gynecology and Reproductive Biology: X [Internet]. 2019 Jul;3(100060):100060. Available from: <https://www.sciencedirect.com/science/article/pii/S2590161319300948>
7. Patel S. Polycystic ovary syndrome (PCOS), an inflammatory, systemic, lifestyle endocrinopathy. The Journal of Steroid Biochemistry and Molecular Biology. 2018 Sep;182(0960-0760):27–36.
8. Azziz R, Carmina E, Chen Z, Dunaif A, Laven JSE, Legro RS, et al. Polycystic ovary syndrome. Nature Reviews Disease Primers [Internet]. 2016 Aug 11;2(1). Available from: <https://www.nature.com/articles/nrdp201657>
9. Cleveland Clinic. Polycystic ovary syndrome (PCOS) [Internet]. Cleveland Clinic. Cleveland Clinic; 2023. Available from: <https://my.clevelandclinic.org/health/diseases/8316-polycystic-ovary-syndrome-pcos>
10. World Health Organization. Polycystic ovary syndrome [Internet]. World Health Organization. 2025. Available from: <https://www.who.int/news-room/fact-sheets/detail/polycystic-ovary-syndrome>
11. Sirmans S, Pate K. Epidemiology, diagnosis, and management of polycystic ovary syndrome. Clinical Epidemiology [Internet]. 2013 Dec;6(6):1–13. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3872139/>
12. Scott Lucidi R. Polycystic Ovarian Syndrome: Practice Essentials, Background, Etiology [Internet]. Medscape.com. 2018. Available from: <https://emedicine.medscape.com/article/256806-overview>



13. Solomon CG. THE EPIDEMIOLOGY OF POLYCYSTIC OVARY SYNDROME. *Endocrinology and Metabolism Clinics of North America*. 1999 Jun;28(2):247–63.
14. Ganie MA, Vasudevan V, Wani IA, Baba MS, Arif T, Rashid A. Epidemiology, pathogenesis, genetics & management of polycystic ovary syndrome in India. *Indian J Med Res*. 2019;150(4):333-344. doi:10.4103/ijmr.IJMR_1937_17
15. PCOS (Polycystic Ovary Syndrome): Symptoms, Causes, and Treatment [Internet]. Sprint Medical. 2020. Available from: <https://sprintmedical.in/blog/pcos-polycystic-ovary-syndrome>
16. Rasquin L, Mayrin JV. Polycystic Ovarian Disease [Internet]. Nih.gov. StatPearls Publishing; 2022. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK459251/>
17. Dong J, Rees DA. Polycystic ovary syndrome: Pathophysiology and therapeutic opportunities. *BMJ Medicine* [Internet]. 2023 Oct 1;2(1). Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10583117/>
18. Mayo Clinic. Polycystic Ovary Syndrome (PCOS) - Symptoms and Causes [Internet]. Mayo Clinic. 2022. Available from: <https://www.mayoclinic.org/diseases-conditions/pcos/symptoms-causes/syc-20353439>
19. Carr J, Giannotte B, Moore M. PCOS Part 1: Pathophysiology of PCOS [Internet]. The ObG Project. 2019. Available from: <https://www.obgproject.com/2019/06/12/pcos-part-1-sensitive-care-of-the-pcos-patient/>
20. Rosenfield RL, Ehrmann DA. The Pathogenesis of Polycystic Ovary Syndrome (PCOS): The Hypothesis of PCOS as Functional Ovarian Hyperandrogenism Revisited. *Endocrine Reviews* [Internet]. 2016 Jul 26;37(5):467–520. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5045492/available/T1/?report=objectonly>
21. Cureus.com. 2023 [cited 2026 Feb 20]. Available from: https://assets.cureus.com/uploads/figure/file/415212/lightbox_e7ec5160d2f811edb6a8aff9fd7ba23e-image-1.png
22. Mayo Clinic. Polycystic ovary syndrome (PCOS) - Diagnosis and treatment - Mayo Clinic [Internet]. Mayoclinic.org. 2022. Available from: <https://www.mayoclinic.org/diseases-conditions/pcos/diagnosis-treatment/drc-20353443>
23. What are the treatments for PCOS? [Internet]. <https://www.nichd.nih.gov/>. 2024. Available from: <https://www.nichd.nih.gov/health/topics/pcos/conditioninfo/treatments>
24. Srnovršnik T, Virant-Klun I, Pinter B. Polycystic Ovary Syndrome and Endocrine Disruptors (Bisphenols, Parabens, and Triclosan)—A Systematic Review. *Life* [Internet]. 2023 Jan 1;13(1):138. Available from: <https://www.mdpi.com/2075-1729/13/1/138>
25. Jozkowiak M, Piotrowska-Kempisty H, Kobylarek D, Gorska N, Mozdziak P, Kempisty B, et al. Endocrine Disrupting Chemicals in Polycystic Ovary Syndrome: The Relevant Role of the Theca and Granulosa Cells in the Pathogenesis of the Ovarian Dysfunction. *Cells* [Internet]. 2023 Jan 1;12(1):174. Available from: <https://www.mdpi.com/2073-4409/12/1/174>
26. The Connection Between PCOS and Insulin Resistance [Internet]. www.healthcentral.com. 2022. Available from: <https://www.healthcentral.com/condition/polycystic-ovary-syndrome-pcos/insulin-resistance-pcos>
27. Rojas J, Chávez M, Olivar L, Rojas M, Morillo J, Mejías J, et al. Polycystic Ovary Syndrome, Insulin Resistance, and Obesity: Navigating the Pathophysiologic Labyrinth. *International Journal of Reproductive Medicine* [Internet]. 2014;2014:1–17. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4334071/>
28. Mouanness M, Nava H, Dagher C, Merhi Z. Contribution of Advanced Glycation End Products to PCOS Key Elements: A Narrative Review. *Nutrients* [Internet]. 2022 Jan 1;14(17):3578. Available from: <https://www.mdpi.com/2072-6643/14/17/3578>
29. Velez LM, Seldin M, Motta AB. Inflammation and reproductive function in women with polycystic ovary syndrome†. *Biol Reprod*. 2021;104(6):1205-1217. doi:10.1093/biolre/iaob050
30. Li W, Liu C, Yang Q, Zhou Y, Liu M, Shan H. Oxidative stress and antioxidant imbalance in ovulation disorder in patients with polycystic ovary syndrome. *Frontiers in Nutrition*. 2022 Oct 28;9.
31. Alhermi A, Perks H, Nigi V, Altahoo N, Atkin SL, Butler AE. The Role of the Liver in the Pathophysiology of PCOS: A Literature Review. *Biomolecules* [Internet]. 2025 Feb;15(1):51. Available from: <https://pubmed.ncbi.nlm.nih.gov/39858445/>
32. Chen MJ, Ho HN. Hepatic manifestations of women with polycystic ovary syndrome. *Best Practice & Research Clinical Obstetrics & Gynaecology* [Internet]. 2016 Apr 6;37:119–28. Available from: <https://www.sciencedirect.com/science/article/abs/pii/S1521693416300013>
33. Wilson C. High risk of liver disease in women with polycystic ovary syndrome. *Nature Reviews Endocrinology* [Internet]. 2010 Mar [cited 2026 Feb 20];6(3):122–2. Available from: <https://www.nature.com/articles/nrendo.2009.284>
34. Senthilkumar H, Arumugam M. Gut microbiota: a hidden player in polycystic ovary syndrome. *Journal of Translational Medicine*. 2025 Apr 15;23(1).
35. Guo H, Luo J, Lin H. Exploration of the pathogenesis of polycystic ovary syndrome based on gut microbiota: A review. *Medicine* [Internet]. 2023;102(50):e36075. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10727628/>

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