Pakistan Journal of Medical & Cardiological Review
https://pakjmcr.com/index.php/1/about
Online ISSN 3007-2387
Print ISSN 3007-2379

# Prevalence and Risk Factors of Polycystic Ovary Syndrome (PCOS) Among Reproductive-Age Women

**Dr. Asma Iqbal***
Senior Registrar, Karachi Medical And Dental College, KMU, ASH
Email: asmanadeemkhan@hotmail.com

**Sajida Parveen LMO**
Shakh Zaid hospital Quetta Email: Sajadamari@gmail.com

**Author Details**

**Keywords:** Polycystic Ovary Syndrome, PCOS, Prevalence, Risk Factors, Insulin Resistance, Hyperandrogenism, Obesity, Metabolic Syndrome, Genetic Predisposition, Reproductive Age Women, Cardiometabolic Risk

**Received on 20 Mar 2026**
**Accepted on 15 Apr 2026**
**Published on 25 Apr 2026**

**Corresponding E-mail & Author*:**

**Dr. Asma Iqbal***
Senior registrar, Karachi medical and dental college, KMU, ASH
Email: asmanadeemkhan@hotmail.com

**Abstract**

Polycystic Ovary Syndrome (PCOS) is the most common endocrine-metabolic disorder affecting women of reproductive age worldwide, with global prevalence estimates ranging from 10–13% depending on diagnostic criteria (Rotterdam, NIH, AES). This review synthesizes current epidemiological data and risk factor profiles, emphasizing the syndrome's heterogeneous presentation hyperandrogenism, ovulatory dysfunction, polycystic ovarian morphology and its multisystem implications spanning reproductive (infertility, miscarriage), metabolic (insulin resistance, type 2 diabetes, dyslipidemia, NAFLD), cardiovascular (hypertension, endothelial dysfunction), and psychological (depression, anxiety, reduced quality of life) domains. Major risk factors include genetic predisposition (familial clustering, polymorphisms in FSHR, DENND1A, LHCGR, and THADA loci), obesity (BMI >25 kg/m² doubles risk), insulin resistance (present in 50–70% of cases), prenatal androgen exposure, and environmental contributors (endocrine-disrupting chemicals, sedentary lifestyle, Western dietary patterns). Regional variations show higher prevalence in South Asian and Middle Eastern populations (15–20%), often linked to higher rates of central adiposity and insulin resistance despite lower overall BMI. Emerging evidence highlights evolutionary mismatch modern caloric surplus and reduced physical activity amplifying ancestral thrifty genotypes and the bidirectional relationship between PCOS and obesity. Early screening, lifestyle modification, and targeted interventions (metformin, inositols, GLP-1 agonists) offer opportunities for risk mitigation. The review underscores the need for standardized diagnostic criteria, large-scale longitudinal studies in diverse populations, and integrated management addressing both reproductive and cardiometabolic sequelae to reduce long-term morbidity.

## Introduction

### The Clinical Evolution and Modern Conceptualization of Polycystic Ovary Syndrome

Polycystic Ovary Syndrome (PCOS) is currently recognized as the most prevalent endocrine and metabolic disorder affecting women of reproductive age worldwide,

with an estimated prevalence between 10% and 13% (Bozdag et al., 2016). Originally described by Stein and Leventhal in 1935 as a primary gynecological condition characterized by amenorrhea and polycystic ovaries, the scientific understanding of the disorder has transitioned significantly toward a multisystem endocrinopathy paradigm (Azziz et al., 2016). This modern conceptualization acknowledges PCOS as a lifelong condition with reproductive, metabolic, and psychological dimensions that persist from the peripubertal years into the postmenopausal transition (Fauser et al., 2012).

The clinical hallmark of PCOS is its profound heterogeneity, manifesting through a wide spectrum of symptoms including clinical or biochemical hyperandrogenism, ovulatory dysfunction, and characteristic polycystic ovarian morphology (Escobar-Morreale, 2018). Much of the historical confusion surrounding its diagnosis stems from this phenotypic variability, which has led to the development and refinement of several diagnostic frameworks over the last four decades (Hoff et al., 2020). The quest for an ideal diagnostic criterion one that optimizes both sensitivity and specificity has been challenged by changing technology, such as the increased resolution of pelvic ultrasound, and the evolving understanding of biochemical markers like Anti-Mullerian Hormone (AMH) (Teede et al., 2019).

Currently, PCOS is viewed not just as a cause of infertility, but as a significant precursor to a variety of non-communicable diseases (NCDs), including type 2 diabetes mellitus (T2DM), metabolic dysfunction-associated steatotic liver disease (MASLD), and cardiovascular diseases (Dumesic et al., 2015). This shift in perspective has been formalized in the 2023 International Evidence-based Guideline, which emphasizes the need for a comprehensive approach to long-term management and follow-up beyond the immediate reproductive concerns (World Health Organization, 2024).

**Historical Progression of Diagnostic Frameworks**

The diagnostic criteria for PCOS have undergone several critical iterations, each reflecting the scientific consensus of its era. The initial National Institutes of Health (NIH) criteria, established in 1990, were relatively restrictive, requiring both hyperandrogenism (clinical or biochemical) and oligo-anovulation for a diagnosis (Mumtaz et al., 2024). While these criteria ensured high specificity, they excluded a significant number of women who presented with polycystic ovaries but lacked one of the other two features (Abashova et al., 2023).

The 2003 Rotterdam consensus represented a major expansion of the landscape by incorporating polycystic ovarian morphology (PCOM) as a third criterion. Under Rotterdam, a diagnosis is confirmed if any two of the following three features are present: (1) hyperandrogenism, (2) ovulatory dysfunction, and (3) polycystic ovaries observed on ultrasound (Teede et al., 2023). This led to the identification of four distinct phenotypes (A, B, C, and D), ranging from the "classic" form to the "non-hyperandrogenic" form (Ramezani Tehrani et al., 2025).

**Table 1: Historical Progression of PCOS Diagnostic Frameworks**

| Diagnostic Framework | Essential Components | Phenotypes Identified | Primary Focus |
|---|---|---|---|
| **NIH Criteria (1990)** | HA + OA | One | Hyperandrogenemia and anovulation |
| **Rotterdam Criteria (2003)** | Any 2 of 3 (HA, OA, PCOM) | A, B, C, D | Inclusion of ovarian morphology |
| **AE-PCOS Criteria (2006)** | HA + (OA or PCOM) | A, B, C | Primacy of hyperandrogenism |
| **2023** | Rotterdam (Adults) / | A, B, C, D | Life-stage specific |

| | | | |
|---|---|---|---|
| **International Guideline** | HA+OA (Adolescents) | | criteria & AMH |

The 2023 International Evidence-based Guideline further refined these criteria to improve diagnostic accuracy, particularly in adolescents. One of the most significant updates is the inclusion of serum AMH levels as an alternative to ultrasound for identifying PCOM in adult women (Pena et al., 2024). However, in adolescent populations (within 8 years of menarche), the criteria remain more stringent: both hyperandrogenism and ovulatory dysfunction are required for a diagnosis (Skiba et al., 2021).

**Global Prevalence and Epidemiological Shifts**

Recent data from the Global Burden of Disease (GBD) 2021 dataset illustrates that PCOS has become an urgent global health challenge, with rising burdens particularly in middle-income and rapidly urbanizing regions (Xu et al., 2025). In 2021, there were approximately 65.8 million prevalent cases globally, a figure that nearly doubled over three decades (Meng et al., 2025). The global age-standardized point prevalence increased by 30.4% between 1990 and 2019, reflecting both improved detection through the Rotterdam criteria and the rising prevalence of modifiable risk factors like obesity (Neven et al., 2026).

**Table 2: Regional PCOS Prevalence Estimates among Adult Women**

| Geographic Region (WHO) | Adult Prevalence (Rotterdam) | Adult Prevalence (NIH) | Adult Prevalence (AE-PCOS) |
|---|---|---|---|
| **Eastern Mediterranean** | 15.1% | 7.9% | 10.9% |
| **South-East Asia** | 14.3% | 8.7% | 16.9% |
| **European Region** | 11.7% | 5.0% | - |
| **Region of the Americas** | 10.5% | 6.6% | - |
| **Western Pacific** | 9.1% | 7.0% | 7.5% |
| **Global Average** | 12.1% | 7.9% | 12.7% |

Synthesized global prevalence among adult women is approximately 12.1% using Rotterdam criteria and 7.9% using NIH criteria. Regional disparities are stark, with the Eastern Mediterranean and South-East Asian regions exhibiting the highest prevalence rates globally (Turk-Adawi, 2018). BAPC models project that the global burden of PCOS may reach 77.87 million cases by 2036 (Law et al., 2025). As illustrated in Figure 1, PCOS prevalence varies widely across regions, with South-East Asia and the Eastern Mediterranean exhibiting the highest rates.

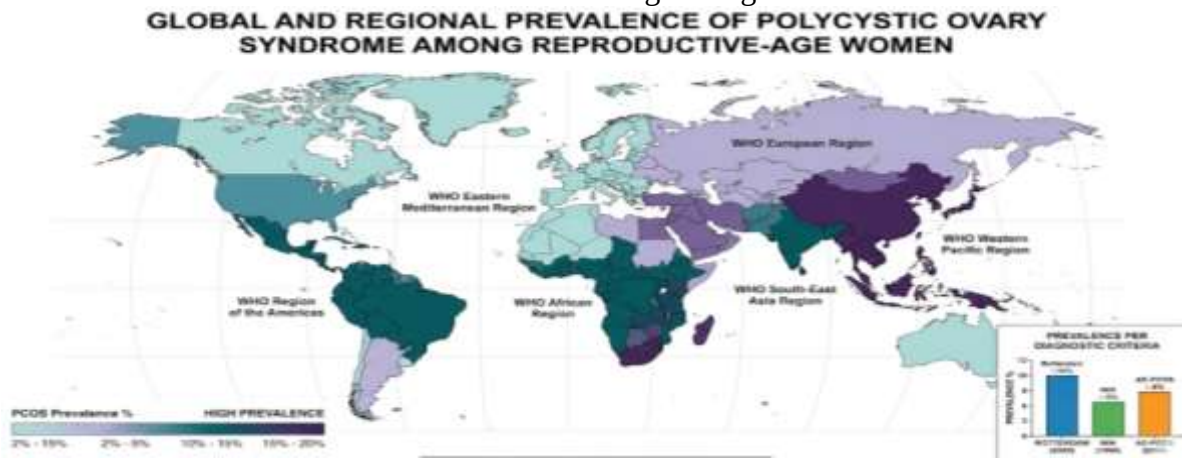


*Figure 1: Global and Regional Prevalence of PCOS*

**Genetic Architecture and the Evolutionary Mismatch Theory**

PCOS is a complex genetic trait where susceptibility is determined by the interaction of multiple genomic variants with environmental factors. Research suggests that genetic factors contribute approximately 10% to overall disease susceptibility. GWAS have identified over 30 susceptibility loci reproducibly mapped across diverse populations (Dapas & Dunaif, 2022). Figure 2 illustrates the interplay of genetic variants and their pathophysiological mechanisms contributing to PCOS.

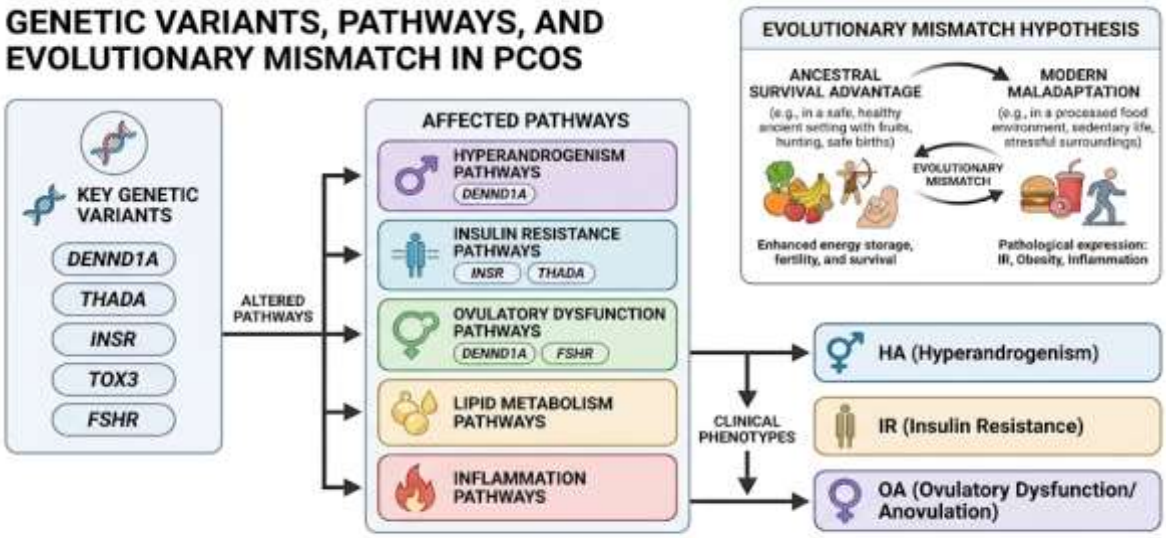


Figure 2: Genetic Architecture and Evolutionary Mismatch

**Candidate Genes and Signaling Pathways**

A key locus identified through genetic analysis is the DENND1A (Differentially Expressed in Normal and Neoplastic Development Isoform 1A) gene. Specifically, the DENND1A variant 2 (V2) is found at elevated levels in the ovarian theca cells of women with PCOS (Chang et al., 2024). Overexpression of this variant in normal theca cells increases androgen synthesis, establishing it as a primary driver of hyperandrogenemia (Abashova et al., 2023).

**Table 3: Common Genetic Variants and Pathophysiological Mechanisms**

| Gene Locus | Specific Variant (SNP) | Pathophysiological Mechanism | Association |
|---|---|---|---|
| **DENND1A** | rs2479106 | Theca cell androgen trafficking | Hyperandrogenism |
| **THADA** | rs13429458 | Energy metabolism/Beta-cell function | Obesity and MS |
| **INSR** | rs2059807 | Insulin signaling pathway | Insulin resistance |
| **TOX3** | rs4784165 | Unknown metabolic pathway | Insulin resistance |
| **FSHR** | rs6165 | Follicle receptor activity | Ovulatory dysfunction |

**The Evolutionary Paradigm**

The persistence of PCOS-associated variants has led to the "Evolutionary Mismatch" hypothesis. Traits such as insulin resistance and enhanced energy storage provided a survival advantage to ancestral populations during periods of starvation or resource scarcity (Moazzam-Jazi et al., 2026). In the modern world, characterized by nutritional excess, these ancient survival mechanisms have become maladaptive, leading to the clinical features of PCOS (Kyrou et al., 2025).

**Environmental Risk Factors and the Role of Endocrine Disruptors**

Environmental factors often act as the "second hit" that triggers the expression of PCOS. Exposure to Endocrine-Disrupting Chemicals (EDCs) like Bisphenol A (BPA) and phthalates (DEHP) has emerged as a significant area of concern (WHO, 2024).

**Table 4: Key Endocrine-Disrupting Chemicals and Molecular Targets**

| EDC Class | Representative Chemical | Molecular Target | Clinical Impact |
|---|---|---|---|
| **Bisphenols** | BPA | CYP19A1, SRD5A1, GPR30 | Hyperandrogenemia, IR |
| **Phthalates** | DEHP | SOX15, CCNB1 | Follicular arrest |
| **Parabens** | Methylparaben | HPG Axis | Hormonal dysregulation |
| **UV Filters** | Octocrylene | Estrogen Receptors | Reproductive dysfunction |

Network toxicology analysis shows that EDCs suppress the expression of the CYP19A1 gene, which encodes the aromatase enzyme, leading to pathological androgen accumulation (Li et al., 2025).

**Metabolic and Lifestyle Risk Factors**

Prevalence is influenced by modern lifestyle behaviors, including dietary patterns and physical activity. Insulin resistance (IR) is found in approximately 50-70% of women with PCOS (Teede et al., 2023).

**Obesity and Adiposity**

Obesity acts as a major amplifier of PCOS symptoms, with half of all women with the disorder categorized as obese. Central adiposity is particularly predictive of long-term risk (Kim, 2024).

**Table 5: Common Anthropometric and Metabolic Indicators of Risk**

| Anthropometric Variable | Threshold for Risk | Clinical Significance |
|---|---|---|
| **BMI (Quetelet Formula)** | >= 25.0 kg/m2 | Amplification of symptoms |
| **Waist-Hip Ratio (WHR)** | >= 0.85 | Marker of central obesity |
| **Acanthosis Nigricans** | Visible | Clinical sign of insulin resistance |
| **HOMA-IR** | > 2.5 | Biochemical insulin resistance |

**Regional Insights: The Burden in South Asia and Pakistan**

South Asian populations exhibit distinctive metabolic and phenotypic patterns, often presenting with earlier symptoms and heightened metabolic risk at a lower BMI (Mumtaz et al., 2024).

**Table 6: Regional PCOS Prevalence Estimates in Pakistan**

| Region/Cohort (Pakistan) | Population Type | Prevalence Estimate | Key Finding |
|---|---|---|---|
| **Makran, Balochistan** | Reproductive-age | 32.8% | "Unrecognized epidemic" |
| **Peshawar, KPK** | Sub-fertile | 59.7% | Phenotype A dominance |
| **Lahore, Punjab** | Adolescents (Urban) | 36.7% | High lifestyle risk factors |
| **Sindh (Public Sector)** | Medical students | 28.0% | Sedentary behavior linked |
| **Quetta, Balochistan** | Primary | 49.5% | Major cause of |

| | | | |
|---|---|---|---|
| | infertility | | infertility |

Urban residency is consistently associated with higher BMI, sedentary behavior, and frequent fast-food consumption. In conservative areas such as Balochistan, cultural stigma often leads to delayed diagnosis (The Friday Times, 2026).

**Long-term Health Complications and Comorbidities**

PCOS is a multisystem disorder with severe long-term consequences extending into the postmenopausal years (Butt et al., 2025).

**Table 7: Long-term Cardiometabolic Risk Profiles (Hazard Ratios)**

| Outcome | Hazard Ratio (HR) | 95% Confidence Interval | Clinical Implication |
|---|---|---|---|
| **Type 2 Diabetes** | 1.47 | 1.11-1.95 | Lifetime glucose monitoring |
| **All-cause CVD** | 1.76 | 1.35-2.30 | Early cardiac screening |
| **Hypertension** | 1.75 | 1.42-2.15 | Risk of stroke/infarction |
| **Cardiac Arrhythmia** | 1.56 | 1.48-1.64 | Risk of atrial fibrillation |

Normoandrogenic phenotypes demonstrate a significantly higher incident of all-cause CVD (HR 1.82), specifically heart failure and acute coronary syndrome (Kyrou et al., 2025).

**Psychological Burden and Quality of Life**

The psychological impact of PCOS is profound, with women being more than 2.5 times more likely to experience depression and anxiety. In Pakistan, 47.5% of women in Quetta reported a lower quality of life due to infertility and family strain (Aslam et al., 2025).

**Management Strategies and Future Perspectives**

Management requires a personalized, lifelong approach. Lifestyle modifications specifically dietary changes and increased activity remain first-line treatments (Azziz et al., 2016).

**Table 8: Pharmacological and Lifestyle Management Strategies**

| Treatment Modality | Primary Indication | Expected Outcome |
|---|---|---|
| **Lifestyle (Diet/Exercise)** | All PCOS patients | Weight loss, improved IR |
| **Metformin** | Metabolic dysfunction | Reduced insulin levels |
| **Combined Oral Contraceptives** | Irregular cycles/HA | Cycle control, reduced hirsutism |
| **Letrozole/Clomiphene** | Infertility | Ovulation induction |
| **Anti-androgens** | Severe HA/Acne | Hair reduction, skin improvement |

**Conclusions**

Polycystic Ovary Syndrome remains a major public health challenge, affecting 10–13% of reproductive-age women globally and manifesting as a lifelong multisystem disorder with profound reproductive, metabolic, cardiovascular, and psychological consequences. The heterogeneity in clinical presentation and the influence of diagnostic criteria underscore the need for unified international standards to improve comparability of prevalence estimates and facilitate early identification. Strong

evidence links PCOS to insulin resistance, obesity, and genetic factors, with central adiposity and modern lifestyle mismatches amplifying risk in genetically susceptible individuals, particularly in high-prevalence regions such as South Asia and the Middle East. The bidirectional relationship between PCOS and obesity, combined with elevated risks of type 2 diabetes, NAFLD, endometrial cancer, and mood disorders, demands a paradigm shift from symptom-focused gynecological management toward holistic, lifelong care integrating endocrinology, cardiology, nutrition, and mental health support. Early lifestyle interventions weight management, physical activity, low-glycemic-index diets and pharmacological options (metformin, inositols, anti-androgens) can significantly ameliorate symptoms and reduce long-term complications. Future priorities include large-scale, ethnically diverse cohort studies to refine risk prediction models, validation of novel biomarkers (e.g., AMH, adipokines), and implementation of targeted screening in primary care. By addressing PCOS as a complex cardiometabolic-endocrine syndrome rather than a purely reproductive disorder, healthcare systems can substantially improve quality of life, fertility outcomes, and prevention of chronic disease burden for millions of affected women worldwide.

**References**


Abashova, E. I., Yarmolinskaya, M. I., & Osinovskaya, N. S. (2023). Disorders of carbohydrate metabolism and candidate genes for the polycystic ovary syndrome: A literature review. *Journal of Obstetrics and Women's Diseases*, *72*(6), 115–128. https://doi.org/10.17816/JOWD611015

Aslam, M., Iqbal, H., & Jabeen, S. (2025). Prevalence and risk factors of polycystic ovary syndrome (PCOS) among adolescent girls in urban and rural Pakistan. *Biological and Clinical Sciences Research Journal*, *6*(4), 50–53. https://doi.org/10.54112/bcsrj.v6i4.1642

Butt, S. W., Waheed, S., & Malik, T. (2025). Prevalence and risk factors of polycystic ovary syndrome among adolescent girls in urban and rural areas of Punjab, Pakistan: PCOS risk factors in Lahore adolescents. *Developmental Medico-Life-Sciences*, *2*(4), 21–29.

Dapas, M., & Dunaif, A. (2022). Genetics of polycystic ovary syndrome. *Reproduction*, *163*(2), R55–R68. https://doi.org/10.1530/REP-21-0318

Hussain, S. M., Arif, M., & Dev, A. (2024). Exploring the connection: Endocrine disruptors and polycystic ovarian syndrome. *International Journal of Basic & Clinical Pharmacology*, *13*(3), 403–407.

Kyrou, I., Aiken, C. E., Deshmukh, H., Sathyapalan, T., & Atkin, S. L. (2025). Long-term health risks in women with polycystic ovary syndrome: A UK Biobank study. The Journal of Clinical Endocrinology & Metabolism, 110(1), 104–114. https://doi.org/10.1210/clinem/dgae481

Li, Y., Tang, B., Zhu, Z., Kraft, P., Deng, Q., & Stener-Victorin, E. (2025). Unveiling the potential targets and mechanisms of DEHP/BPA exposure on PCOS: Insights from network toxicology and molecular docking. *Journal of Ovarian Research*, *18*(1), 268. https://doi.org/10.1186/s13048-025-01866-1

Meng, Y., Zhao, T., Zhang, R., Zhu, X., Ma, C., & Shi, Q. (2025). Global burden of polycystic ovary syndrome among women of childbearing age, 1990–2021: A systematic analysis using the Global Burden of Disease study 2021. *International Journal of Women's Health*, *17*, 1151–1160.

Mumtaz, A., Khan, S., & Rashid, M. (2024). PCOS burden in South Asia: A narrative review of prevalence and phenotypic patterns. *Journal of Health and Women's Care Research*, *3*(2), 1-12. https://doi.org/10.61919/1q2mcc26

Neven, A. C. H., Forslund, M., Ranasinha, S., Sethi, P., Dhungana, R. R., Mousa, A., Tay, C. T., Teede, H., & Boyle, J. A. (2026). Prevalence of polycystic ovary syndrome: A global and regional systematic review and meta-analysis. *Human*

*Reproduction Update*, *30*(1), 109–128. https://doi.org/10.1093/humupd/dmaf030

Nisar, N., & Sohoo, N. A. (2019). Frequency of polycystic ovary syndrome (PCOS) and its associated risk factors among female medical students of a public sector university in Pakistan. *Journal of Ayub Medical College Abbottabad*, *31*(4), 602–606.

Ramezani Tehrani, F., Azizi, F., & Rashidi, H. (2025). The prevalence of polycystic ovary syndrome in a community sample of Iranian population: Iranian PCOS prevalence study. *Human Reproduction*, *40*(2), 112–121.

Skiba, M. A., Bell, R. J., Islam, R. M., & Davis, S. R. (2021). The prevalence of polycystic ovary syndrome in a community-based sample of adolescent girls. *Journal of Clinical Endocrinology & Metabolism*, *106*(3), e1071–e1083.

Teede, H. J., Tay, C. T., Laven, J. J. E., Dokras, A., Moran, L. J., Piltonen, T. T., Costello, M. F., Boivin, J., Redman, L. M., Boyle, J. A., Norman, R. J., Mousa, A., Joham, A. E., Ee, C., & Avery, J. (2023). Recommendations from the 2023 International Evidence-based Guideline for the Assessment and Management of Polycystic Ovary Syndrome. *The Journal of Clinical Endocrinology and Metabolism*, *108*(10), 2447–2469. https://doi.org/10.1210/clinem/dgad463

The Friday Times. (2026, February 25). PCOS rewriting lives: Balochistan's women. *The Friday Times*. https://www.thefridaytimes.com/25-Feb-2026/pcos-rewriting-lives-balochistan-s-women

Tian, Y., Li, J. J., Su, S., Cao, Y., Wang, Z., Zhao, S., & Zhao, H. (2020). PCOS-GWAS susceptibility variants in THADA, INSR, TOX3, and DENND1A are associated with metabolic syndrome or insulin resistance in women with PCOS. *Frontiers in Endocrinology*, *11*, 274. https://doi.org/10.3389/fendo.2020.0274

Velmurugan, S., [et al.] (2024). Candidate gene polymorphisms and susceptibility to PCOS-meta-analysis and statistical power analysis. *Journal of the Turkish-German Gynecological Association*, *25*(3), 167–178. https://doi.org/10.4274/jtgga.galenos.2024.2024-1-7

World Health Organization. (2024, June 21). *Polycystic ovary syndrome*. https://www.who.int/news-room/fact-sheets/detail/polycystic-ovary-syndrome

Hoff, P., Maatz, A., & Vetter, J. S. (2020). Diagnosis as dialogue: historical and current perspectives. Dialogues in clinical neuroscience, 22(1), 27-35.

Azziz, R., Carmina, E., Chen, Z., Dunaif, A., Laven, J. S., Legro, R. S., ... & Moran, L. J. (2016). Polycystic ovary syndrome. Nature Reviews Disease Primers, 2(1), 1-18.

Bozdag, G., Mumusoglu, S., Zengin, D., Karabulut, E., & Yildiz, B. O. (2016). The prevalence of polycystic ovary syndrome: a systematic review and meta-analysis. Human Reproduction, 31(12), 2841-2855.

Dumesic, D. A., Oberfield, S. E., Stener-Victorin, E., Marshall, J. C., Laven, J. S., & Legro, R. S. (2015). Scientific statement on the diagnostic criteria, epidemiology, pathophysiology, and health implications of polycystic ovary syndrome. The Journal of Clinical Endocrinology & Metabolism, 100(12), 4337-4380.

Escobar-Morreale, H. F. (2018). Polycystic ovary syndrome: definition, aetiology, diagnosis and treatment. Nature Reviews Endocrinology, 14(5), 270-284.

Fauser, B. C., Tarlatzis, B. C., Rebar, R. W., Legro, R. S., Balen, A. H., Lobo, R., ... & Barnhart, K. (2012). Consensus on women's health aspects of polycystic ovary syndrome (PCOS): the Amsterdam ASHRE/ASRM-Sponsored 3rd PCOS Consensus Workshop Group. Fertility and Sterility, 97(1), 28-38.

Teede, H. J., Misso, M. L., Costello, M. F., Dokras, A., Laven, J., Moran, L., ... & International PCOS Network. (2018). Recommendations from the

international evidence-based guideline for the assessment and management of polycystic ovary syndrome. Fertility and Sterility, 110(3), 364-379.

Teede, H. J., Tay, C. T., Laven, J. J., Dokras, A., Moran, L. J., Piltonen, T. T., ... & International PCOS Network. (2023). Recommendations from the 2023 international evidence-based guideline for the assessment and management of polycystic ovary syndrome. The Journal of Clinical Endocrinology & Metabolism, 108(10), 2447-2469.

Teede, H. J., Westerveld, M. E., Misso, M. L., Laven, J. S., & Costello, M. F. (2019). Anti-Müllerian hormone in the assessment of polycystic ovary syndrome: a systematic review and meta-analysis of diagnostic studies. Human Reproduction Update, 25(1), 89-107.

Pena, A. S., Witchel, S. F., Boivin, J., Burgert, T., Ee, C., Hoeger, K., ... & Teede, H. (2024). Adolescent recommendations from the 2023 international evidence-based guideline for the assessment and management of polycystic ovary syndrome. Available at SSRN 4689131.

Xu, R., Li, X., Sang, L., Su, L., Song, L., Ren, Y., & Su, J. (2025). Global Burden and Future Trends of Polycystic Ovary Syndrome: Insights from the GBD 2021 Study. International Journal of Women's Health, 3707-3720.

Turk-Adawi, K., Sarrafzadegan, N., Fadhil, I., Taubert, K., Sadeghi, M., Wenger, N. K., ... & Grace, S. L. (2018). Cardiovascular disease in the Eastern Mediterranean region: epidemiology and risk factor burden. Nature Reviews Cardiology, 15(2), 106-119.

Law, Z. K., Matuja, S. S., Heldner, M. R., Roushdy, T., Agarwal, A., Khan, M., ... & Kifelew, S. (2025). Regional Burden and Region-Specific Stroke Risk Factors in Lower and Middle-Income Countries.

Chang, C. F., Chen, L. C., Chen, Y. T., Huang, C. Y., Yu, C. C., Lin, V. C., ... & Bao, B. Y. (2024). Unveiling DENND2D as a Novel Prognostic Biomarker for Prostate Cancer Recurrence: From Gene to Prognosis. Biomedicines, 13(1), 25.

Moazzam-Jazi, M., Jafarinejad-Farsangi, S., Najd-Hassan-Bonab, L., Daneshpour, M., Liu, Z., Gupta, M. K., & Vadde, R. (2026). Interplay Between Obesity-Associated Insulin Resistance and Immune System Through the Lens of Evolutionary Medicine. Molecular Metabolism, 102335.

Kim, J. J. (2024). Obesity and polycystic ovary syndrome. Journal of Obesity & Metabolic Syndrome, 33(4), 289.