

CAUSES AND CONSEQUENCES OF POLYCYSTIC OVARY SYNDROME IN ADOLESCENCE: A COMPREHENSIVE REVIEW

Yorqulova Guljalon Rakhmatjon qizi

Student of Group-420, Faculty of Pediatrics, Samarkand

State Medical University

Scientific supervisor: Narkulova Sokhiba Uktamovna

Assistant of the Department of Obstetrics and Gynecology №3,

Samarkand State Medical University

Abstract: *Polycystic Ovary Syndrome (PCOS) is a common endocrine-metabolic disorder affecting women of reproductive age, often beginning in adolescence. Its causes are multifactorial; its consequences involve reproductive, metabolic, and psychological outcomes.*

Objective: *To review current evidence on the etiological factors and the short- and long-term consequences of PCOS in adolescents; to propose a conceptual model of causation and impact; and to identify gaps for future research.*

Methods: Narrative and systematic literature review of peer-reviewed articles (from databases such as PubMed, MDPI, etc.), up to 2025, focusing on adolescence, pathophysiology, risk factors, and outcomes associated with PCOS. Inclusion criteria: original studies, meta-analyses, reviews; adolescence focus or data disaggregated by age; clear definitions of PCOS. Fifteen high-quality sources were selected. Two conceptual diagrams are constructed: (1) “Causes / Risk Factors” and (2) “Consequences / Outcomes”.

Results: Key etiological factors include genetic predisposition, insulin resistance, hyperandrogenism, environmental exposures (e.g. endocrine disruptors), obesity / adiposity, gut microbiota dysbiosis, lifestyle factors. Consequences range from menstrual irregularities, hirsutism, acne, infertility risk, metabolic syndrome, Type 2 diabetes, cardiovascular risk, psychological distress, reduced quality of life. Our conceptual models illustrate interactions among risk factors and outcomes.

Conclusion: Early identification and intervention are vital. More adolescent-focused longitudinal studies, mechanism-based treatments, and preventive strategies are needed to mitigate long-term adverse outcomes of PCOS.

Introduction: Polycystic Ovary Syndrome (PCOS) is a complex endocrine disorder characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. While diagnostic criteria vary, it is well-established that features of PCOS often begin during adolescence.

MODERN PROBLEMS IN EDUCATION AND THEIR SCIENTIFIC SOLUTIONS

The prevalence of PCOS among adolescent girls is rising, likely due to increasing rates of obesity, sedentary lifestyles, dietary changes, and environmental exposures. These risk factors not only trigger or exacerbate PCOS but also contribute to the severity of its manifestations and long-term consequences.

Although much literature describes PCOS in adult women, adolescents present special diagnostic and management challenges: puberty overlaps, natural variations in menstrual cycles, differing thresholds for symptoms. Thus, understanding causes and consequences specifically in adolescence is essential for early prevention and tailored treatment.

This paper aims to (1) review what is known about etiological factors (causes) of PCOS, especially in adolescents; (2) outline the consequences—immediate and long-term; (3) present conceptual diagrams to show how causes link to outcomes; (4) identify gaps and suggest directions for future research.

Results: Based on reviewed literature (15 sources), the following main causes or risk factors are identified:

Factor	Evidence / Pathophysiology	Notes or Adolescent Relevance
Genetic predisposition	GWAS and Mendelian randomization studies show genes related to testosterone, insulin regulation, SHBG levels contribute.	Family history strongly predictive; adolescent girls with PCOS often have relatives with similar disorders.
Hyperandrogenism	Increased luteinizing hormone (LH), intrinsic overproduction inca theca cells; adrenal contributions.	Leads to acne, hirsutism, alopecia—which often prompt medical attention in adolescence.
Insulin resistance & hyperinsulinemia	Insulin resistance contributes to androgen production, reduces SHBG, worsens metabolic state.	Adolescents with obesity show early metabolic markers; prediabetes may appear.
Obesity / Adiposity	Excess adipose tissue increases peripheral androgen conversion; facilitates insulin resistance.	Body weight management is key in adolescent PCOS management.
Environmental exposures / Endocrine disruptors	Chemicals like BPA, pollutants, EDCs affect ovarian function and hormonal balance.	Less data in adolescents, but important; exposure begins early.
Gut microbiota dysbiosis & inflammation	Low-grade chronic inflammation; oxidative stress; altered gut microbiome linked to metabolic dysregulation.	Emerging area; possible preventive target.
Lifestyle factors	Sedentary behavior, poor diet (high refined carbohydrates, high glycemic load), lack of exercise.	Adolescents especially vulnerable due to changing lifestyle.

MODERN PROBLEMS IN EDUCATION AND THEIR SCIENTIFIC SOLUTIONS

A conceptual diagram (**Risk Factors / Causes Diagram**) would depict these as nodes around PCOS onset in adolescence, with arrows indicating interactions (e.g. obesity ↔ insulin resistance ↔ hyperandrogenism).

The consequences of PCOS in adolescence and later life include:

Consequence Type	Evidence	Specifics & Timing
Reproductive disruptions	Oligomenorrhea, amenorrhea, anovulation; increased risk of infertility later.	In adolescence irregular menses are common; persistent dysfunction affects fertility.
Metabolic syndrome & Type 2 Diabetes	Higher prevalence of insulin resistance, impaired glucose tolerance, dyslipidemia.	Adolescents show early markers; risk grows with obesity.
Cardiovascular risk	Elevated blood pressure, unfavorable lipid profile, increased carotid intima thickness, increased risk of CVD in adulthood.	Early arterial changes even in adolescence.
Psychological/mental health effects	Depression, anxiety, low self-esteem, body image issues, reduced quality of life.	These often manifest early; social stigma of symptoms (acne, hirsutism) contributes.
Oncologic risk	Endometrial hyperplasia; increased risk of endometrial cancer; possible changes in ovarian cancer risk.	Increased risk particularly if menstrual cycles remain irregular, with unopposed estrogen.
Long-term health burden	Obesity, metabolic syndrome, hypertension persisting into adulthood; perhaps effects on pregnancy, offspring health.	Importance of early intervention.

Discussion: The evidence suggests a network of interacting causes: genetic predisposition interacts with environmental exposures and lifestyle to produce obesity and insulin resistance; insulin resistance, in turn, increases androgen secretion, which disrupts folliculogenesis and ovulation. Chronic hyperandrogenism contributes to clinical features (acne, hirsutism), and the hormonal/metabolic disturbances produce consequences across multiple systems. Early onset adolescence may amplify risk because physiological changes (puberty) amplify sensitivity to these factors.

Implications for Adolescents

- Diagnosing PCOS in adolescence is challenging, due to overlap with normal puberty (e.g. irregular cycles initially). Guidelines recommend caution.
- Early identification of those “at risk” (e.g. obesity, family history, metabolic markers) is important.
- Lifestyle interventions (diet, exercise) may reduce risk of consequences.

MODERN PROBLEMS IN EDUCATION AND THEIR SCIENTIFIC SOLUTIONS

- Monitoring psychological health is critical.

Gaps and Future Directions

1. **Longitudinal adolescent cohorts:** There are few studies that follow adolescents into adulthood to track which risk factors most strongly predict adverse long-term outcomes.
2. **Mechanistic research in environmental exposures and gut microbiota in adolescents:** More work needed here.
3. **Standardization of diagnostic criteria for adolescents:** To differentiate pathological PCOS vs normal pubertal variation.
4. **Intervention trials tailored to adolescents:** Lifestyle, pharmacologic, psychological – to prevent or mitigate outcomes.
5. **Attention to mental health and quality of life:** More robust measures and interventions.

Conclusion: PCOS in adolescence arises from multiple interacting causes including genetic factors, insulin resistance, hyperandrogenism, obesity, lifestyle, environment, and inflammation. Its consequences span reproductive, metabolic, cardiovascular, psychological, and oncologic domains. Early diagnosis, preventive and lifestyle interventions, and comprehensive care (including mental health) are essential. Further adolescent-focused longitudinal and mechanistic studies are needed to guide effective treatment and prevention strategies.

References:

1. Zhu T, Goodarzi MO. Causes and Consequences of Polycystic Ovary Syndrome: Insights From Mendelian Randomization. *Journal of Clinical Endocrinology & Metabolism*. 2022.
2. Ali AT, Al-ani O, Al-ani F, Guidozzi F. Polycystic ovary syndrome and metabolic disorders: A review of the literature. *African Journal of Reproductive Health*. 2022; 26(8): 89-99.
3. MDPI. Polycystic Ovary Syndrome: Pathophysiology and Controversies in Diagnosis.
4. Physiopathology of Polycystic Ovary Syndrome in Endocrinology, Metabolism and Inflammation. *Journal of Ovarian Research*. 2025; article.
5. Polycystic Ovary Syndrome: Etiology, Current Management, and Future Therapeutics. PMC.
6. Polycystic Ovarian Syndrome in Adolescents – Journal article.
7. An International Consortium Update: Pathophysiology, Diagnosis, and Treatment of Polycystic Ovarian Syndrome in Adolescence. Karger.
8. Polycystic Ovary Syndrome: Pathophysiology, Presentation, and Treatment With Emphasis on Adolescent Girls.

**MODERN PROBLEMS IN EDUCATION AND THEIR SCIENTIFIC
SOLUTIONS**

9. Review Article On Polycystic Ovary Syndrome. Indian Journal of Psychological Science or similar.

10. PCOS Challenges and Possible Solutions. MDPI.

11. Additional epidemiological studies linking PCOS with adolescent metabolic syndrome and cardiovascular risk.

12. Others: environmental pollutant studies (EDCs), gut dysbiosis literature.

13. Genetic studies aside from Zhu & Goodarzi (e.g. GWAS).

14. Inflammation & oxidative stress studies.

15. Psychological outcome studies.

