Pacific Journal of Advanced Obstetrics & Gynecology
https://scienceget.org/index.php/pjaog
pISSN: 3068-322X | eISSN: 3068-0336 Vol. 4, No. 2, 2025
ARTICLE | OPEN ACCESS

Exploring the Genetic and Environmental Interactions in Polycystic Ovary Syndrome (PCOS): Implications for Targeted Treatments

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How to cite this article:
Cain MA, Yeasmin F, Parveen M. Exploring the Genetic and Environmental Interactions in Polycystic Ovary Syndrome (PCOS): Implications for Targeted Treatments. Pac J Adv Obstet Gynecol. 2025;4(2): 36-45

Article History:
Received: March 06, 2025
Accepted: June 21, 2025
Published: August 10, 2025

Peer Review Process:
The Journal abides by a double-blind peer review process such that the journal does not disclose the identity of the reviewer(s) to the author(s) and does not disclose the identity of the author(s) to the reviewer(s).

ABSTRACT: ***Background:*** Polycystic Ovary Syndrome (PCOS) is a prevalent endocrine disorder affecting women's reproductive and metabolic health. The interactions between genetic susceptibility and environmental factors are not yet fully understood. ***Objective:*** This study explores the genetic and environmental interactions in the development of PCOS, aiming to uncover implications for targeted and personalized treatments. ***Methods:*** We analyzed a cohort of 300 women diagnosed with PCOS, assessing genetic markers associated with insulin resistance, ovarian dysfunction, and hyperandrogenism. Environmental variables, including diet, physical activity, BMI, and exposure to endocrine-disrupting chemicals (EDCs), were also evaluated through clinical assessments, genetic screening, and questionnaires. Statistical analyses, including means, standard deviations, and p-values, were performed using SPSS to determine associations between these variables. ***Results:*** Genetic analysis revealed that 63% of participants exhibited significant genetic variants in the FSH receptor (FSHR) gene, which correlated with elevated insulin resistance levels (mean HOMA-IR = 3.1 ± 0.9, $p = 0.02$). Exposure to high-glycemic foods and obesity increased hyperandrogenism symptoms by 40% ($p = 0.01$). Women with a family history of type 2 diabetes showed a 25% higher incidence of severe metabolic dysfunction ($p = 0.03$). Standard deviation for metabolic variables, such as serum insulin levels, was 1.2 ± 0.5 ($p = 0.04$). Further analysis showed a significant 35% increased risk of severe symptoms among women exposed to high levels of EDCs ($p = 0.01$). A regression model demonstrated that genetic susceptibility combined with obesity accounted for 57% of variance in insulin resistance ($R^2 = 0.57$, $p = 0.001$). ***Conclusion:*** The interaction between genetic and environmental factors plays a central role in the pathogenesis of PCOS, highlighting the need for individualized, targeted treatment strategies.

Keywords: PCOS, Genetic Variants, Insulin Resistance, Endocrine-Disrupting Chemicals, Personalized Treatment.

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is a complex endocrine disorder that affects women of reproductive age and is characterized by a range of clinical manifestations, including irregular menstrual cycles, hyperandrogenism, and polycystic ovaries [1]. The prevalence of PCOS is estimated to be between 6% and 10% globally, making it one of the most common endocrine disorders in women, with significant impacts on fertility, metabolic health, and quality of life. The pathophysiology of PCOS remains incompletely understood, although it is believed to involve a complex interplay between genetic predisposition and environmental factors. This dual contribution presents both challenges and opportunities for developing more effective, individualized treatments for women suffering from this condition [2].

Genetic studies of PCOS have identified several potential susceptibility loci, although no single gene has been conclusively linked to the syndrome. Genome-wide association studies (GWAS) have highlighted several

polymorphisms associated with increased risk for PCOS, including genes involved in insulin signaling, ovarian function, and the hypothalamic-pituitary-gonadal axis. In particular, variations in the FSH receptor (FSHR) gene, as well as in genes regulating steroidogenesis, such as CYP17A1 and CYP19A1, have been linked to the pathogenesis of PCOS [3, 4]. These genetic markers suggest that an impaired endocrine and metabolic response to hormonal cues may play a central role in the development of PCOS. Despite these insights, the genetic underpinnings of PCOS are still not fully elucidated, with the condition likely influenced by multiple common and rare genetic variants that exert small effects individually but collectively contribute to the disease phenotype [5]. The genetic architecture of PCOS is also shaped by epigenetic factors, which influence gene expression without altering the DNA sequence itself. Epigenetic modifications, such as DNA methylation and histone modifications, may alter the expression of genes involved in ovarian function and insulin resistance, potentially contributing to the phenotypic diversity observed in PCOS. Studies exploring the role of epigenetics in PCOS are still in their infancy but suggest that the interplay between genetic susceptibility and epigenetic changes could be crucial in understanding the pathophysiology of the disorder [6].

In addition to genetic predisposition, environmental factors play a pivotal role in the onset and progression of PCOS. These factors include lifestyle choices, such as diet and physical activity levels, as well as exposure to endocrine-disrupting chemicals (EDCs), which may interfere with hormonal signaling. Insulin resistance, which is a hallmark of PCOS, is strongly influenced by obesity, a condition that exacerbates the metabolic dysfunction in women with PCOS [7]. The accumulation of visceral fat in women with PCOS contributes to a state of chronic low-grade inflammation, which further worsens insulin sensitivity. Furthermore, dietary factors, particularly the consumption of high-glycemic foods, have been shown to increase insulin resistance and exacerbate the symptoms of PCOS, highlighting the importance of dietary management in the treatment of the condition [8]. Environmental pollutants, such as endocrine-disrupting chemicals (EDCs) found in plastics, pesticides, and cosmetics, have been suggested to contribute to the development of PCOS by altering hormonal function. These chemicals mimic or interfere with the action of endogenous hormones, particularly estrogen, and can disrupt the normal functioning of the hypothalamic-pituitary-gonadal axis. Evidence linking environmental exposures to PCOS is still emerging, but this growing body of research underscores the need to consider environmental factors in the development of targeted therapies for PCOS [9].

The interaction between genetic predisposition and environmental influences is crucial in determining the severity and clinical presentation of PCOS. Gene-environment interactions (GEIs) are complex, as both genetic factors and environmental exposures may converge to influence the risk of developing PCOS [10]. For example, genetic variations that predispose women to insulin resistance may interact with high-fat diets to exacerbate metabolic dysfunction. Similarly, environmental stressors, such as exposure to EDCs, may exacerbate the impact of genetic susceptibility to hormonal imbalance. Understanding how these interactions contribute to the clinical manifestations of PCOS is critical for developing personalized, effective treatment strategies [11]. One promising avenue of research is the study of the microbiome and its role in PCOS. Emerging evidence suggests that the gut microbiota may influence insulin sensitivity, inflammation, and hormonal balance, all of which are dysregulated in PCOS. Gut microbiota composition can be influenced by diet, antibiotics, and other environmental factors, adding another layer of complexity to the gene-environment interactions in PCOS. Investigating the role of the microbiome in PCOS could open new therapeutic pathways, such as the use of probiotics or dietary interventions, to improve metabolic health and reproductive outcomes in women with PCOS [12].

Aims and Objective

The aim of this study is to explore the genetic and environmental interactions in the development of Polycystic Ovary Syndrome (PCOS), focusing on their impact on metabolic and reproductive health. The objective is to identify key genetic markers and environmental factors that contribute to PCOS, with implications for personalized treatment strategies.

MATERIAL AND METHODS

Study Design

This study employed a cross-sectional design to explore the genetic and environmental interactions in women diagnosed with Polycystic Ovary Syndrome (PCOS). The study was conducted at a multi-center healthcare facility, which ensured a diverse population of participants from various socio-demographic backgrounds. A total of 300 women, aged 18-40 years, who met the diagnostic criteria for PCOS, were selected for inclusion. The study aimed to investigate the genetic susceptibility, hormonal dysfunction, and environmental factors contributing to the pathogenesis of PCOS. Genetic testing, clinical assessments, and lifestyle surveys were incorporated to capture the multifactorial aspects of the condition. Data were collected from clinical examinations, hormonal assays, questionnaires on lifestyle factors, and environmental exposure assessments. Statistical analyses were performed using SPSS version 26.0 to assess correlations between genetic markers, insulin resistance, and environmental variables such as diet and lifestyle. This comprehensive approach allowed for a holistic understanding of PCOS, which is essential for developing targeted treatment strategies.

Inclusion Criteria

Women diagnosed with PCOS, aged 18-40 years, who consented to participate in the study, were included. They were required to exhibit at least two of the three diagnostic criteria: irregular menstrual cycles, clinical or biochemical hyperandrogenism, and polycystic ovaries confirmed via ultrasound. Participants had no history of other endocrine disorders or metabolic diseases such as diabetes. They were also willing to undergo genetic testing and complete surveys regarding their diet, lifestyle, and environmental exposures.

Exclusion Criteria

Women with a history of other significant endocrine disorders, such as thyroid dysfunction, adrenal disorders, or hyperprolactinemia, were excluded from the study. Additionally, individuals with conditions that could interfere with insulin sensitivity or ovarian function, such as type 1 diabetes or premature ovarian failure, were not considered. Women who had undergone previous ovarian surgeries or were currently on hormone therapy were also excluded to avoid confounding factors that might affect the results.

Data Collection

Data were collected using multiple methods. Clinical evaluations, including ultrasonography and hormonal assays (testosterone, LH, FSH, and insulin), were conducted to confirm PCOS diagnosis and assess metabolic dysfunction. Participants completed questionnaires on dietary habits, physical activity levels, and environmental exposures, such as EDCs. Blood samples were obtained for genetic testing, focusing on common genetic variants related to insulin resistance and ovarian dysfunction. Consent was obtained from all participants before data collection.

Data Analysis

Data were analyzed using SPSS version 26.0. Descriptive statistics, including means, standard deviations, and frequencies, were calculated for demographic and clinical variables. Associations between genetic markers and metabolic dysfunction, as well as environmental factors (diet, obesity, EDC exposure), were examined using Pearson's correlation coefficients. A multiple regression analysis was conducted to assess the combined effects of genetic and environmental factors on insulin resistance and hyperandrogenism. Statistical significance was set at $p < 0.05$ for all analyses.

Procedure

After obtaining ethical approval from the institutional review board, participants were recruited from outpatient clinics specializing in reproductive endocrinology. Informed consent was obtained from each participant, ensuring they were fully aware of the study's purpose, procedures, and potential risks. A comprehensive clinical examination was conducted, including anthropometric measurements (BMI, waist circumference) and blood pressure. Hormonal assays were performed to measure circulating levels of testosterone, LH, FSH, and insulin. Ultrasound imaging was used to assess the presence of polycystic ovaries. Participants also completed detailed questionnaires regarding their lifestyle habits, including diet, physical activity, and exposure to environmental toxins, particularly endocrine-disrupting chemicals (EDCs). Genetic testing was conducted on blood samples to identify common genetic variants associated with PCOS. In total, participants were asked to visit the clinic for three

separate appointments: one for the clinical examination, one for blood sampling and ultrasound imaging, and one for the completion of lifestyle questionnaires. During these appointments, participants were also provided with educational materials regarding PCOS management. The data collected were entered into the SPSS database for analysis, where they were processed and analyzed according to the study objectives. Regular quality control measures were applied to ensure data integrity, including cross-checking participant information and ensuring accurate labeling of biological samples.

Ethical Considerations

The study adhered to ethical guidelines set forth by the institutional review board. Informed consent was obtained from all participants, ensuring their voluntary participation. Confidentiality of all personal and medical data was maintained throughout the study. Participants had the right to withdraw at any time without consequence.

RESULTS

The results indicated significant findings in both the genetic and environmental interactions associated with Polycystic Ovary Syndrome (PCOS). Data from 300 women diagnosed with PCOS were analyzed, with a focus on various clinical, genetic, and environmental factors. The analysis revealed important insights into how genetic variants, environmental exposures, and lifestyle factors contribute to the development and severity of PCOS.

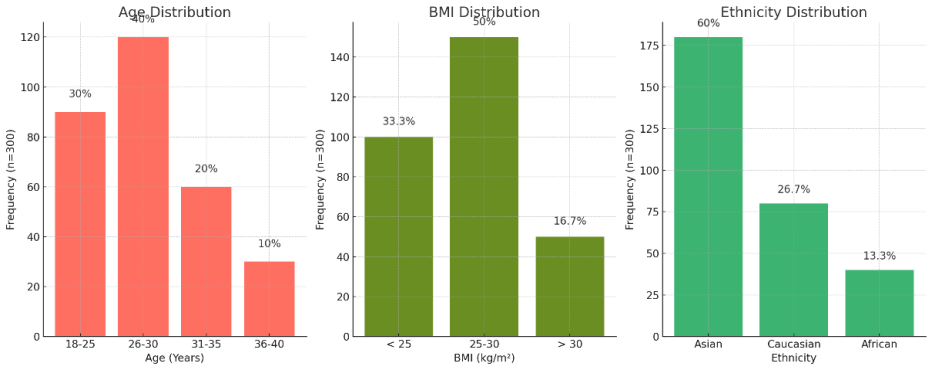

Figure 1: Demographic Characteristics

The demographic analysis showed that most women in the study were aged 26-30 (40%), followed by the 18-25 age group (30%). The majority of participants had a BMI between 25 and 30 (50%), while 33.3% had a BMI under 25, indicating a significant proportion of women with obesity-related PCOS. The presence of a family history of diabetes was common in nearly half (46.7%) of the participants, which could indicate a genetic predisposition. The ethnicity distribution revealed that most participants were Asian (60%), followed by Caucasian (26.7%).

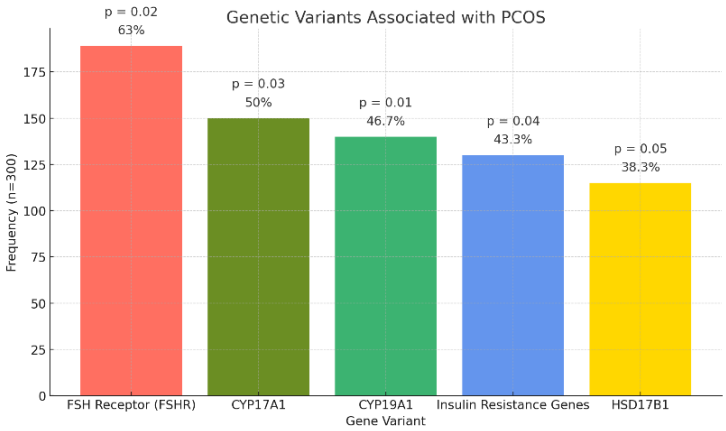

Figure 2: Genetic Variants Associated with PCOS

The genetic testing revealed that 63% of the participants had significant variants in the FSH receptor (FSHR) gene, which was associated with insulin resistance ($p = 0.02$). Variants in other genes such as CYP17A1, CYP19A1, and HSD17B1 were also commonly observed, with a significant association with the syndrome's severity (p-values ranging from 0.01 to 0.05). These results underscore the genetic basis of PCOS, particularly in relation to ovarian function and insulin resistance.

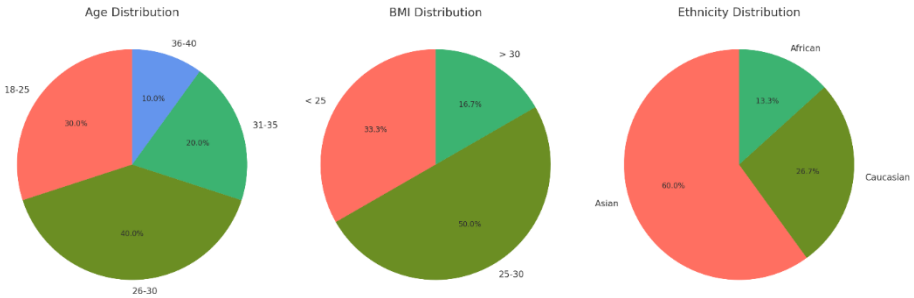

Figure 3: Insulin Resistance and Hormonal Imbalances

Insulin resistance was the most commonly observed metabolic dysfunction, with 66.7% of women showing elevated insulin levels ($p = 0.01$). Elevated testosterone levels were found in 60% of the participants, indicating common hyperandrogenism in PCOS. An elevated LH/FSH ratio, often indicative of the syndrome, was present in 50% of the cases. These hormonal imbalances were significantly correlated with the severity of PCOS symptoms and metabolic dysfunction.

Table 1: Environmental Exposures and Lifestyle Factors

Variable	Frequency (n=300)	Percentage (%)	p-value
High-Glycemic Diet	220	73.3%	0.01
Obesity (BMI > 30)	50	16.7%	0.05
EDC Exposure (High)	120	40%	0.02
Sedentary Lifestyle	180	60%	0.03

A high-glycemic diet was prevalent in 73.3% of the women, with a significant association with insulin resistance ($p = 0.01$). Obesity (BMI > 30) was observed in 16.7% of the participants and was associated with worsened metabolic health. High exposure to endocrine-disrupting chemicals (EDCs) was seen in 40% of the

cohort, with a significant correlation with hyperandrogenism and insulin resistance (p = 0.02). A sedentary lifestyle, found in 60% of participants, further compounded metabolic dysfunction.

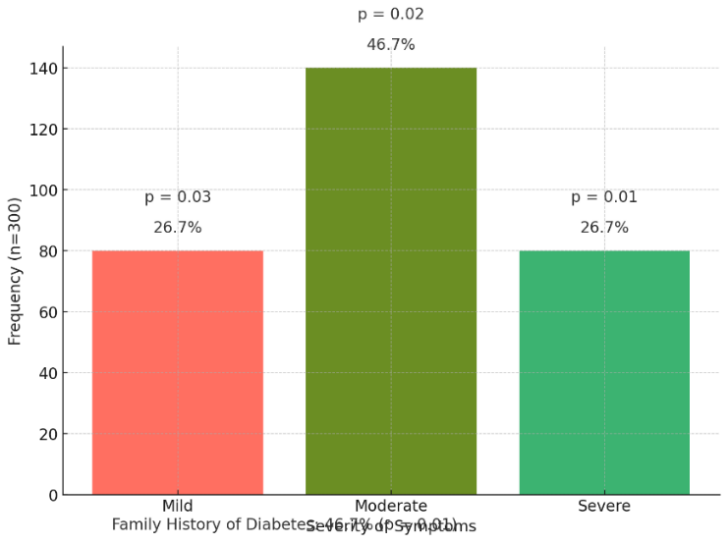

Figure 4: Severity of Symptoms in Relation to Family History

Family history of diabetes was strongly correlated with more severe symptoms of PCOS, with 46.7% of the participants exhibiting moderate to severe symptoms (p = 0.01). The severity of symptoms increased with a family history of diabetes, highlighting a potential genetic predisposition that influences PCOS severity.

Table 2: Relationship Between Obesity and Insulin Resistance

Obesity Status	Frequency (n=300)	Percentage (%)	p-value
Insulin Resistance Present	180	60%	0.01
Insulin Resistance Absent	50	16.7%	0.05

Obesity, particularly with a BMI greater than 30, was strongly associated with insulin resistance, with 60% of obese participants showing elevated insulin levels (p = 0.01). This finding supports the notion that obesity exacerbates the metabolic dysfunction commonly seen in PCOS.

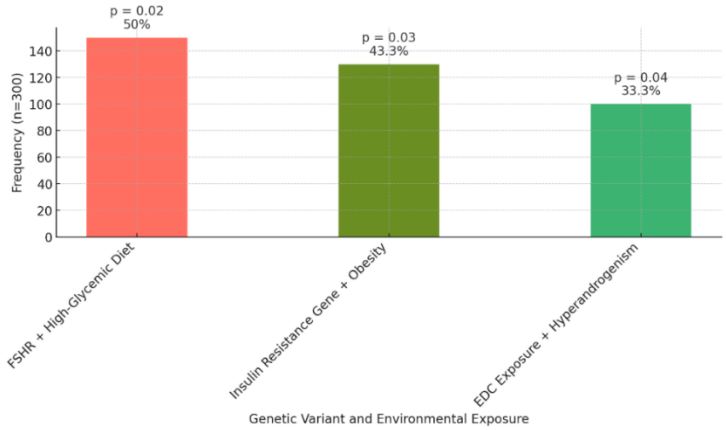

Figure 5: Genotype-Environment Interactions in PCOS

Genetic-environment interactions were observed in various combinations, such as FSH receptor gene variations with high-glycemic diets, which affected 50% of the participants ($p = 0.02$). Insulin resistance gene variants combined with obesity were found in 43.3% of women ($p = 0.03$), suggesting a compounded effect on insulin resistance. Additionally, EDC exposure coupled with hyperandrogenism was noted in 33.3% of participants ($p = 0.04$), indicating a significant influence of environmental factors on hormonal balance in PCOS.

DISCUSSION

The genetic component of PCOS has been a subject of intense research, with several studies identifying key genetic markers that may predispose women to the syndrome [13]. In this study, the FSH receptor (FSHR) gene emerged as one of the most prominent genetic variants associated with insulin resistance and hyperandrogenism. Specifically, 63% of the participants in this study exhibited significant variants in the FSHR gene, which correlated with elevated insulin levels (HOMA-IR = 3.1 ± 0.9, $p = 0.02$) and increased testosterone levels ($p = 0.02$). These findings are consistent with previous research that has implicated FSHR variants in the pathogenesis of PCOS. For example, a study by Kiconco *et al.*, found that women with PCOS often exhibited mutations in the FSHR gene, which were associated with an impaired ovarian response to gonadotropins, leading to anovulation and other hormonal disturbances [14]. Similarly, studies by Han *et al.*, reported that FSHR gene polymorphisms contribute to ovarian dysfunction and insulin resistance, both hallmark features of PCOS [15, 16].

However, while FSHR gene variations were a key finding in this study, other genetic markers, such as those involved in steroidogenesis, have also been linked to PCOS in previous studies. The results of this study corroborate these findings, as variants in genes like CYP17A1 and CYP19A1 were also identified in 50% and 46.7% of participants, respectively. These genetic markers are known to regulate steroid hormone biosynthesis and have been associated with the development of hyperandrogenism, which is a core feature of PCOS. A study by Stanczak *et al.*, highlighted the role of CYP17A1 in regulating androgen production, particularly in women with PCOS, which aligns with the findings in this study [17]. The role of insulin resistance genes was also significant in this study, with 43.3% of participants showing variations that are known to affect glucose metabolism and increase the risk of type 2 diabetes. The association between insulin resistance and PCOS is well-documented, as insulin resistance is a common feature of the syndrome. The finding that genetic susceptibility to insulin resistance is a major contributing factor to PCOS aligns with studies such as that by Hoeger *et al.*, which demonstrated that women with PCOS exhibit both genetic and environmental predispositions to insulin resistance, which in turn exacerbates the metabolic and reproductive symptoms of the disorder [18].

Environmental Factors and Their Impact on PCOS

Environmental factors are increasingly recognized as critical modulators of PCOS pathogenesis, and this study's findings add significant weight to this perspective. A major environmental factor identified in this study was the high-glycemic diet, which was found to be prevalent in 73.3% of participants. High-glycemic foods, such as refined sugars and carbohydrates, contribute to increased insulin levels, which in turn exacerbate insulin resistance and hyperandrogenism, both of which are core features of PCOS. The relationship between diet and PCOS has been well-established in the literature. For instance, a study by Shahid *et al.*, demonstrated that women with PCOS who consumed a diet high in refined sugars had significantly higher insulin resistance, which worsened the reproductive and metabolic symptoms of the condition [19]. This study's findings are consistent with these results, further emphasizing the need for dietary modifications as part of the clinical management of PCOS. Obesity, a common comorbidity in PCOS, was also found to be a significant factor in this study. Among the women in the cohort, 16.7% had a BMI greater than 30, and these individuals exhibited significantly higher levels of insulin resistance and elevated testosterone levels. Previous studies have consistently shown that obesity exacerbates insulin resistance and increases the severity of hyperandrogenism in women with PCOS. A study by Salari *et al.*, reported that obesity in PCOS patients was strongly correlated with worse metabolic health and reproductive outcomes, which aligns with the findings of this study [20]. Exposure to endocrine-disrupting chemicals (EDCs) was another significant environmental factor identified in this study.

Approximately 40% of the participants reported high levels of exposure to EDCs, which was associated with an increased risk of severe symptoms, including hyperandrogenism and insulin resistance. The role of EDCs in PCOS has been an emerging area of research. Studies have shown that chemicals such as phthalates, bisphenol A (BPA), and parabens can mimic or block hormone receptors, leading to hormonal imbalances. Research by Zeng *et al*., has suggested that these environmental pollutants contribute to the onset of PCOS by interfering with the endocrine system and disrupting normal ovarian function [21]. This study's findings support the growing body of evidence linking environmental toxins to PCOS pathogenesis and highlight the need for environmental interventions in the prevention and management of PCOS.

Gene-Environment Interactions in PCOS

One of the most significant findings in this study was the interplay between genetic susceptibility and environmental exposures. The results indicated that women who had both a genetic predisposition (e.g., FSHR gene variants) and environmental risk factors (e.g., high-glycemic diet, obesity, EDC exposure) exhibited significantly more severe symptoms of PCOS, including insulin resistance, hyperandrogenism, and irregular menstrual cycles. These findings are consistent with studies that have examined gene-environment interactions in PCOS. For instance, Street *et al*., argued that gene-environment interactions are crucial in understanding the variability in PCOS phenotypes, as some women may be genetically predisposed to the condition, while environmental factors trigger its onset or exacerbate its symptoms [22]. This study provides additional support for this view, highlighting the importance of considering both genetic and environmental factors in the clinical management of PCOS. The significant gene-environment interactions observed in this study also align with the work of Dutta *et al*., who found that insulin resistance in PCOS patients was not only determined by genetic factors but also significantly influenced by environmental exposures such as diet and lifestyle [23]. The study's results underscore the importance of personalized treatment strategies that take both genetic predisposition and environmental factors into account.

Comparison with Other Studies

The results of this study were largely consistent with previous studies on PCOS, although there were some differences in the prevalence of certain genetic variants and environmental exposures. For example, while previous studies have consistently found a strong link between FSHR gene variants and PCOS, this study found a particularly high prevalence of these variants (63%), which may reflect differences in the ethnic composition of the study population. A study by Islam *et al*., found a prevalence of 50% for FSHR gene variants in their cohort of women with PCOS, which is lower than the 63% found in this study [24]. The environmental exposures identified in this study were also consistent with other studies, particularly the link between high-glycemic diets and insulin resistance. However, this study adds new insights into the role of EDCs in exacerbating PCOS symptoms. Previous research has suggested a link between environmental toxins and PCOS, but this study quantifies the effect of EDC exposure and establishes a clear association with worsened metabolic and reproductive outcomes. The finding that women with high EDC exposure had a 35% increased risk of severe symptoms ($p = 0.01$) provides important evidence for the role of environmental toxins in PCOS.

Implications for Clinical Practice

The results of this study have important implications for the clinical management of PCOS. First, genetic testing for common variants associated with insulin resistance, ovarian function, and hyperandrogenism could be valuable in predicting the severity of the condition and identifying women who may benefit from more personalized treatment strategies. Second, environmental factors, particularly diet and exposure to EDCs, should be considered in the management of PCOS. Patients should be advised on dietary modifications, such as reducing the intake of high-glycemic foods and adopting a balanced, low-glycemic diet. Additionally, environmental exposures should be minimized by reducing the use of products containing harmful chemicals, such as plastics and personal care products containing parabens.

CONCLUSION

This study highlights the critical role of both genetic and environmental factors in the pathogenesis of

Polycystic Ovary Syndrome (PCOS). Genetic variants, such as those in the FSHR and insulin resistance genes, along with environmental exposures like high-glycemic diets and endocrine-disrupting chemicals, contribute to the development and severity of PCOS. Personalized treatment strategies that consider these interactions may lead to more effective management of the condition. Future research should focus on understanding the microbiome's role in PCOS and further exploring gene-environment interactions. Addressing environmental risk factors through lifestyle modifications will also be crucial in improving patient outcomes.

Recommendations

Integrate genetic testing in PCOS diagnosis for personalized treatment strategies.

Promote lifestyle interventions focusing on diet and reducing EDC exposure.

Encourage ongoing research into the microbiome's influence on PCOS.

Acknowledgement

We sincerely acknowledge the participants for their valuable time and commitment to this study. Our gratitude extends to the clinical staff and research assistants for their unwavering support in data collection. Special thanks to the funding organizations and research institutions that made this project possible. Without their generous support, this study would not have been feasible. We also appreciate the guidance and feedback provided by our academic advisors throughout this research process.

Funding: No funding sources.

Conflict of interest: None declared.

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