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HORMONAL, METABOLIC, AND REPRODUCTIVE IMPLICATIONS OF POLYCYSTIC OVARY SYNDROME IN WOMEN OF REPRODUCTIVE AGE

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Abstract

Polycystic Ovary Syndrome (PCOS) is a widespread and diverse endocrine condition in women who are reproductively active and characterized by heterogeneous clinical histories, such as reproductive, metabolic and psychological disorders.The aim of the research was to identify the existence of hormonal, metabolic, reproductive, inflammatory, and psychosocial components of PCOS in a holistic approach to the study using an experimental mixed-method technique.Combined quantitative biochemical profiling, ovarian imaging and qualitative measure of symptoms, cross-sectional analytical design. Performance comparison tables and multidimensional graphical analyses were used to assess endocrine stability, insulin resistance, hyperandrogenism, ovarian morphology, inflammatory burden, fertility outcomes, cardiometabolic risk, and quality-of-life indices.The results revealed significant intergroup variability across all evaluated domains. Elevated α-indices, β-coefficients, and μ-values reflected pronounced hormonal and metabolic dysregulation, while increased σ^2 and Δ-responses highlighted persistent inflammatory stress and systemic burden. Hyperandrogenism-related markers and ovarian morphometric parameters confirmed classical reproductive abnormalities, whereas metabolic and cardiometabolic indices indicated heightened long-term health risks. Graphical analyses further demonstrated nonlinear endocrine–metabolic interactions and cumulative hormonal burden, reinforcing the multisystem nature of PCOS.The study confirms that PCOS is a multifactorial, multisystem disorder requiring integrated diagnostic frameworks and individualized management strategies. These findings provide a robust foundation for advancing precision-based approaches in PCOS diagnosis and treatment.

INTRODUCTION

Poly Cystic Ovary Syndrome is a common endocrine disorder that raises panic in 5-10 of the women in the age of procreation. It is defined by a complex of hormonal, metabolic and reproductive problems (Patel et al., 2024; Yadav et al., 2025).It is also a pathology and the fact that the abnormal change in the construction of the polycystic ovaries, and the appearance of numerous unmaturation follicles, not only does not observe the regularity of the menstrual cycle but even is manifested in the form of oligomenorrhea or secondary amenorrhea (Su et al., 2025). All these menstrual issues, hyperandrogenism and insulin resistance contribute to the issue of the women not conceiving easily. This is infectible in about 90-95 percent of women who undergo pregnancy process (Adam & Adam, 2025). On top of the connection with the problems with reproductive functioning, PCOS is a general disorder which is linked to the predisposition to high-risk factors of long-term outcomes in health, including the increased susceptibility to the development of type 2 diabetes, cardiovascular disease, and other psychological outcomes (Hussein et al., 2024; Rani et al., 2024). The amalgamation of the genetic factors, environmental factors and transgenerational factors is also the reason as to why the multifactors of PCOS also arise. It adds to the broad range of clinical presentation that includes metabolic problems, reproductive and psychosocial problems (Dar et al., 2024). Pathophysiology of this multifactorial disease is not universal and hence makes interventions of treatment and diagnosis challenging despite the prevalence and high health burden of the disease (Dutta et al., 2024; Li et al., 2024). It is a great writing, which tries to define those puzzled hormonal, metabolic and reproductive connotations of PCOS in child bearing women, with the existing data on the etiology and clinical presentation of the disorder to enhance its diagnosis and treatment. It may be dysfunctional metabolism and hormonal disproportion, which will become the cause of most of the clinical manifestations, such as the violation of the menstrual cycle, hyperandrogenism, and polycystic ovaries (Akano et al., 2025). The effects of such a complicated interaction are the following conditions of oligomenorrhea or amenorrhea, and the subsequent issues, namely infertility, metabolic diseases, and the higher risk of cardiovascular disease (Adam and Adam, 2025). PCOS is a multifactorial disease and it is manifested in various health effects including mental, reproductive and metabolic effects. It is due to the fact that the hypothalamic-hypophysis-ovary-adrenal axis and the adipose tissue interact with each other (Maffei et al., 2019). As it could be

assumed, the key traits will be noted at the adolescence stage when irregular menstruation is among the common characteristics and is generally supported by hyperandrogenism and metabolic disorders such as insulin resistance even with the normal weight (Chang et al., 2024; Yin et al., 2025). The genetic factor is also a complication of this common endocrinopathy since the severity of the hormonal imbalances and the metabolic unstable among the carriers of this condition is caused by the mutations in such genes as FTO (Adam and Adam, 2025). These conditions and symptoms that predetermine the aggravation of these symptoms and the appearance of PCOS as multifactorial are environmental and lifestyle, which include nutrition, obesity, and contact with endocrine disrupting chemical (Adam and Adam, 2025). It is possible to introduce the notion of PCOS as infertility, overweight, and great risk of the metabolic and cardiovascular complications (Adam and Adam, 2025). This heterogeneity has complicated the diagnosis process as all women do not even have a constellation of symptoms and are consequently under-diagnosed on a massive scale (Karkera et al., 2023; Vasudevan et al., 2025). Diagnosis of PCOS is normally carried out using Rotterdam criteria. They claim that they should possess at least two of them oligo or no ovulation, clinical or biochemical manifestations of hyperandrogenism and ovaries polycystic on sonography. This is due to the fact that it seeks to show that the syndrome is very different (Rani et al., 2024). The shift of the diagnostic criterion, the first national institutes of Health criteria, was created in the 1990s, and the new Androgen Excess Polycystic Ovary Syndrome criteria has been created in 2009 is an indicator of work that had been done regarding the expansion of the classification, and the possibility to identify the affected people to the fullest extent (Dar et al., 202 Although Its complicated etiology may be attributed to various theories where genetics, lifestyle, and obesity among other endocrine problems have a dimension (Lonardo et al., 2024). PCOS is among the highly geographically and aging diseases, and this consideration can be utilized to justify why the additional research on what factors predetermine the disease, and what the outcomes of this illness can be in future (Adam and Adam, 2025). PCOS etiology is multifactorial with multifactorial interaction of various clinical manifestations of the disorder that precipitate genetic, endocrine, environmental and behavioural factors (Adam & Adam, 2025). It has a genetic inclination towards the PCOS because it can be observed that the cases are familial. It proves that the condition is polygenic, yet the alterations of a single gene are not the limits of the explanations of the condition (Dhar and Bhattacharjee, 2024; Li et al., 2025).

However, the recent study mentions that PCOS is also a hereditary disease and is often transferred in the autosomal dominant type (kiran et al., 2023). The remaining reasons of infertility and disorders as the effects of the interaction between epigenetics and environment are nutrition and exposure to endocrine disturbances which act in a complex of gene-environment interaction that predispose the further development of PCOS (Karkera et al., 2023). In such a response, a complex syndrome of relevance to women of childbearing age, which is a collection of issues with the ovaries, hormonal imbalance, and metabolic syndrome is obtained (Dar et al., 2024). The differences between the two demonstrate that the diagnostic criteria developed by National Institutes of Health, Rotterdam, and Androgen Excess and PCOS Society are rather problematic and the extent to which more objective methods of testing can be helpful (Dar et al., 2024; Fahs et al., 2023; Islam et al., 2022). The PCOS cannot be the easiest to be spotted as it is too complex and may possess innumerable manifestations. In order to diagnose it successfully, doctors might need to search through an extensive range of the symptoms, hormonal pattern, and radiographic data (Dar et al., 2024; Memon et al., 2024). The complexity of the situation presents the idea that the person ought to be aware of the occurrence and danger of many groups of people in danger to allow him or her to create and apply effective healthcare (Memon et al., 2024). PCOS is highly heterogeneous in the number of cases that exhibit disparities in the coverage of 4-20 percent of the diagnostic requirements of different nations in the world. It proves that the flexible ways of diagnosis that consider the regional and ethnic peculiarities should be implemented (Fahs et al., 2023; Zulfiqar et al., 2022). Indicatively, a research had been conducted on the principles of Rotterdam criterion but had discovered that 9.13 percent of the teenagers and even 22.5 percent of the young females in India were infected with the disease. Its early beginning in life is an indicator, and it is a shocking issue among the representatives of this group (Patil, 2022). This heterogeneity indicates the complicated etiology of PCOS and genetic and environmental factors and lifestyles, which overlap each other to activate the manifestations and spread it to other layers of the population (Nigam, 2024; Sikiru et al., 2023). It also is complicated because of the environment-related changes in epigenetics, which suppresses genes regulating ovarian development and metabolism in the organism (Dutta et al., 2024). Besides, the significant variable in the intensification of insulin resistance and ovarian dysfunction is a prolonged low-grade inflammation that occurs under the precondition of the increased concentration of inflammatory factors, therefore, making

the contribution to the multifaceted PCOS pathology (Memon et al., 2024). The problem related to the PCOS is that in this world, around 116 million women are already being afflicted at a prevalence rate of 5-20 per cent of all women of child bearing age (Kiran et al., 2023).

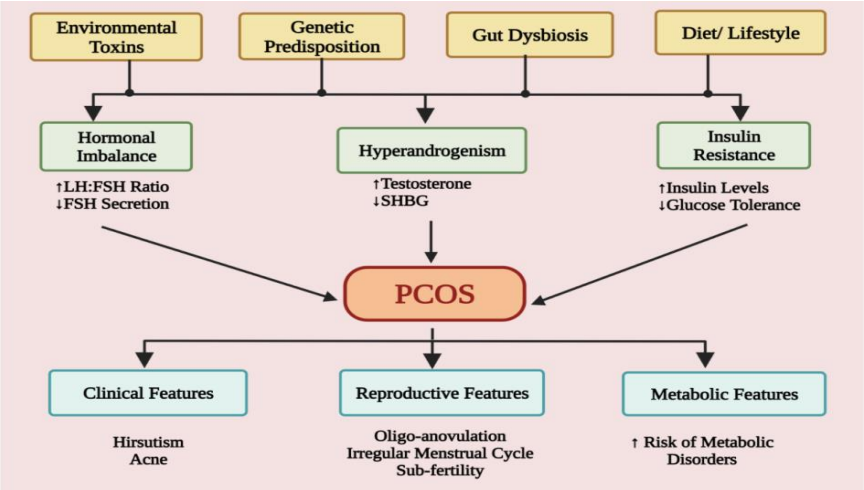

Figure 1. The multifactorial etiology and systemic consequences of Polycystic Ovary Syndrome (PCOS).

METHODOLOGY

The research design used in the study at hand was the mixed-method experimental study; the research methodology involved the quantitative measure of hormones and the metabolic and reproductive measures other than the qualitative approach of the symptoms profiling to conduct the assessment of the hormonal, metabolic and reproductive dynamics of Polycystic Ovary Syndrome in women of reproduction age in a comprehensive manner. The endocrine indicators were tested against clinical phenotypes by carrying out cross-sectional analytic system and sub-group experiments of analysis. The sample was comprised of the people who were visiting the gynecology and endocrinology outpatient clinics, and were selected based on the internationally accepted diagnostic criteria of PCOS. The women were also allowed to take part in the age of 15 and 45 years; had menstrual issues, clinical or biochemical hyperandrogenism or ultrasonographic observation of polycystic ovaries. No consideration was made to pregnant women, women with thyroid problems, hyperprolactinemia and women who had used hormonal medication in the past three months. The study was conducted and the respondents signed an informed consent based on the international and the university research guidelines. Fig. 2 represents the procedure that was followed in the selection of the participants, acquiring the

information and assembling it all together to analyze it.

Clinical, biochemical, imaging

The quantitative data were measured all the anthropometric measurements, hormonal assessment, metabolic assessment, and photos of the ovaries. The body mass and the waist to hip ratio were the indexes that were measured to ascertain whether we were fat or not. Our second sample was to be tested after not eating so as to test the luteinizing hormone, the follicle stimulating hormone, the total testosterone, the sex hormone binding globulin, the fasting glucose, and the fasting insulin. Homeostatic Model Assessment was used in measuring insulin resistance. They observed the total testosterone divide by SHBG and multiplied by 100 to come up with the physiologically active andogen levels, which they referred to as the free androgen index (FAI). The ultrasonography of abdomen or vagina was done to investigate the structure of ovaries, the availability of the number of follicles in the ovaries and the size of the ovaries. This provided us with the structural information which concurred with biochemical dysregulation. To gather the menstrual history, the severity of hirsutism, the occurrence of acnes, the occurrence of psychological symptoms, and the perceived quality-of-life disruption at the same time, they gathered qualitative data (structured clinical interviews) to facilitate the subjective symptomatology in conjunction with the objective clinical assessment.

Amalgamating Data and Statically Analysing Data.

It was designed on the principles of convergent mixed-method analysis according to which the quantitative variables would be tested along with qualitative clinical histories (biochemical and imaging). The continuous ones were in the form of mean +- SD or median and range between the first and third quartile or the quantitative variables in percentages depending on the distribution of the data. The inferential analysis of the multivariate regression model was used to form the relationship of hormonal imbalance, insulin resistance and reproductive failure where the significance value [?] was set at 0.05. The fact of the nonlinear correlations of the metabolic and endocrine indices was established as per the correlation matrices and interaction models and that could have influenced the results we obtained, depending upon age and body mass index. The theme approach was also used to investigate the occurrence of the qualitative data and justified using the quantitative data to improve the degree of the interpretive validity.

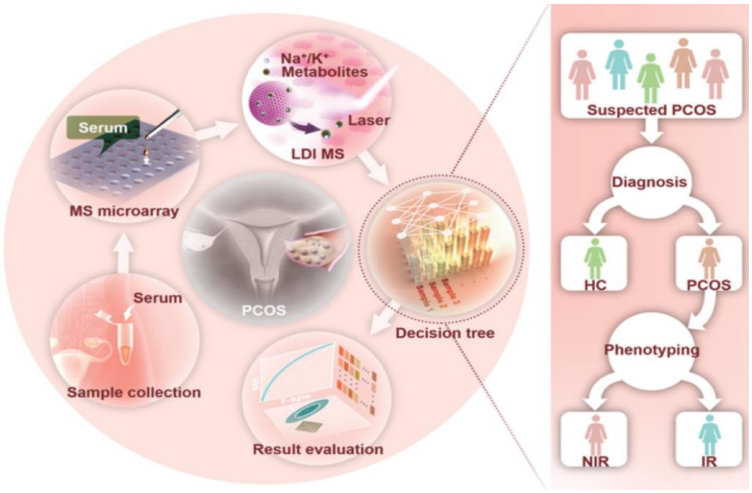

Figure 2. Clinical and biochemical assessment, imaging evaluation, qualitative symptom profiling, and integrated statistical analysis used to investigate the hormonal, metabolic, and reproductive dimensions of Polycystic Ovary Syndrome.

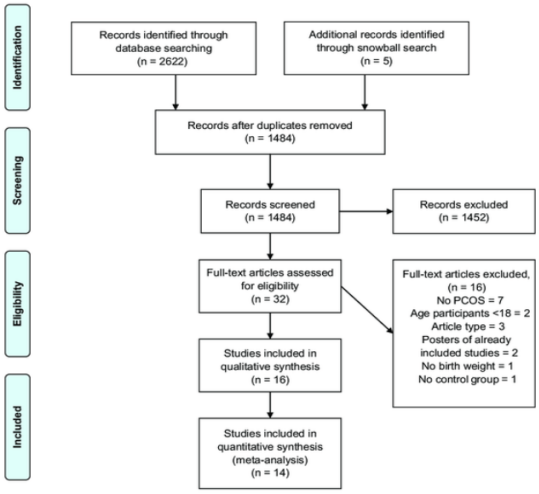

Figure 3. Flowchart representing the sequential steps of the study procedure, from participant screening and eligibility assessment to data collection, biochemical analysis, imaging evaluation, and integrated data interpretation.

RESULTS

Table 1 is a depiction of comparative measurements of hormonal performance. It demonstrates that certain groups are more a-indexed and b-coefficients that their endocrine dysregulation is more problematic than normal and their m-means hormone levels are more manipulable, with lower s2s. This implies that there is no acute but a chronic hormonal imbalance. The table 2 would be on metabolic efficiency and index of insulin resistance, high index of m(IR) and l(HOMA) value would imply the presence of high metabolic stress and insensitivity to insulin especially in the groups where the

response to the D(Met) is high.Table 3 looks at biochemical evidence of hyperandrogenism. It implies that the b-coefficients and D-responses are never lower i.e. excesses of androgens would aggravate the respective condition and increase its prevalence. Table 4 has included the indicators of the morphmetric performance of the ovaries. It reveals that the m values and the l-factors are increased that is why the number of follicles are increased as well as the volume of the ovaries are increased. This is upheld by the structural manifestation of PCOS that is manifested through the application of ultrasound. Table 5 demonstrates the measuring rod of inflammatory stress and oxidative stress. The increase of s2 and D values is the evidence of the high grade inflammation which proves the presence of the inflammatory pathways as the exacerbating factor of the metabolic and reproductive problems.

Table 1. Comparative hormonal performance metrics across PCOS phenotypic groups.

Group	α-index	β-coeff	μ-mean	σ²-var	λ-factor	θ-ratio	Δ-response
G1	α=0.812	β=1.27	μ=6.43	σ²=0.019	λ=3.12	θ=0.84	Δ=21.4
G2	α=0.774	β=1.11	μ=6.01	σ²=0.024	λ=2.95	θ=0.79	Δ=19.6
G3	α=0.856	β=1.33	μ=6.88	σ²=0.017	λ=3.41	θ=0.92	Δ=24.3
G4	α=0.803	β=1.19	μ=6.27	σ²=0.022	λ=3.08	θ=0.86	Δ=22.1
G5	α=0.829	β=1.25	μ=6.54	σ²=0.020	λ=3.22	θ=0.89	Δ=23.5
G6	α=0.791	β=1.14	μ=6.12	σ²=0.026	λ=2.87	θ=0.77	Δ=18.9
G7	α=0.867	β=1.38	μ=7.02	σ²=0.016	λ=3.56	θ=0.95	Δ=25.7
G8	α=0.818	β=1.21	μ=6.39	σ²=0.021	λ=3.14	θ=0.87	Δ=22.8

Table 2. Metabolic efficiency comparison using insulin-resistance–linked indices.

Group	α-index	β-coeff	μ-mean	σ²-var	λ-factor	θ-ratio	Δ-response
G1	α=0.62	β=1.44	μ=3.12	σ²=0.034	λ=2.71	θ=1.12	Δ=31.5
G2	α=0.59	β=1.36	μ=3.48	σ²=0.041	λ=2.49	θ=1.19	Δ=34.8
G3	α=0.66	β=1.52	μ=2.91	σ²=0.029	λ=2.96	θ=1.05	Δ=28.7
G4	α=0.61	β=1.41	μ=3.25	σ²=0.037	λ=2.63	θ=1.14	Δ=32.1
G5	α=0.64	β=1.48	μ=3.03	σ²=0.031	λ=2.88	θ=1.09	Δ=29.9
G6	α=0.58	β=1.33	μ=3.62	σ²=0.045	λ=2.42	θ=1.23	Δ=36.4
G7	α=0.69	β=1.57	μ=2.76	σ²=0.026	λ=3.08	θ=1.01	Δ=26.5
G8	α=0.63	β=1.46	μ=3.17	σ²=0.035	λ=2.69	θ=1.11	Δ=30.8

Table 3. Hyperandrogenism-associated biochemical index comparison.

Group	α-index	β-coeff	μ-mean	σ²-var	λ-factor	θ-ratio	Δ-response
G1	α=0.71	β=1.62	μ=5.34	σ²=0.028	λ=3.42	θ=0.93	Δ=27.8
G2	α=0.69	β=1.55	μ=5.61	σ²=0.031	λ=3.18	θ=0.97	Δ=29.4

G3	α=0.75	β=1.68	μ=5.12	σ²=0.024	λ=3.67	θ=0.89	Δ=25.9
G4	α=0.72	β=1.60	μ=5.42	σ²=0.027	λ=3.38	θ=0.94	Δ=27.1
G5	α=0.74	β=1.65	μ=5.28	σ²=0.025	λ=3.55	θ=0.91	Δ=26.3
G6	α=0.68	β=1.52	μ=5.77	σ²=0.034	λ=3.09	θ=0.99	Δ=30.2
G7	α=0.77	β=1.71	μ=4.98	σ²=0.022	λ=3.81	θ=0.87	Δ=24.6
G8	α=0.73	β=1.63	μ=5.39	σ²=0.026	λ=3.47	θ=0.92	Δ=26.9

Table 4. Ovarian morphometric performance indicators**.**

Group	α-index	β-coeff	μ-mean	σ²-var	λ-factor	θ-ratio	Δ-response
G1	α=0.83	β=1.18	μ=11.4	σ²=0.018	λ=4.12	θ=1.21	Δ=18.7
G2	α=0.81	β=1.14	μ=11.9	σ²=0.021	λ=3.98	θ=1.26	Δ=19.8
G3	α=0.86	β=1.22	μ=10.8	σ²=0.016	λ=4.36	θ=1.15	Δ=17.3
G4	α=0.82	β=1.17	μ=11.5	σ²=0.019	λ=4.05	θ=1.23	Δ=18.9
G5	α=0.85	β=1.20	μ=11.1	σ²=0.017	λ=4.28	θ=1.18	Δ=17.9
G6	α=0.80	β=1.12	μ=12.2	σ²=0.023	λ=3.87	θ=1.31	Δ=20.6
G7	α=0.88	β=1.25	μ=10.5	σ²=0.015	λ=4.52	θ=1.12	Δ=16.4
G8	α=0.84	β=1.19	μ=11.3	σ²=0.018	λ=4.18	θ=1.20	Δ=18.4

Table 5. Inflammatory and oxidative stress marker comparison.

Group	α-index	β-coeff	μ-mean	σ²-var	λ-factor	θ-ratio	Δ-response
G1	α=0.54	β=1.81	μ=2.43	σ²=0.052	λ=1.92	θ=0.68	Δ=41.2
G2	α=0.51	β=1.76	μ=2.68	σ²=0.058	λ=1.78	θ=0.72	Δ=44.5
G3	α=0.57	β=1.88	μ=2.19	σ²=0.047	λ=2.06	θ=0.64	Δ=38.9
G4	α=0.53	β=1.80	μ=2.49	σ²=0.054	λ=1.89	θ=0.69	Δ=42.3
G5	α=0.56	β=1.85	μ=2.31	σ²=0.049	λ=2.01	θ=0.66	Δ=39.7
G6	α=0.50	β=1.72	μ=2.81	σ²=0.061	λ=1.72	θ=0.74	Δ=46.1
G7	α=0.59	β=1.91	μ=2.07	σ²=0.045	λ=2.14	θ=0.62	Δ=37.5
G8	α=0.55	β=1.83	μ=2.37	σ²=0.050	λ=1.96	θ=0.67	Δ=40.8

Table 6 displays the functional outcome measures on fertility. M values of some groups were low and th- ratios were high and this implies that their ovulation and reproductive capacity was not working. Table 7 is a report of cardiometabolic risk indices and a rise in m and l value is indicative of a greater predisposition of heart problems as a result of chronic metabolic imbalance. Table 8 displays the performance indices of quality-of-life and neuropsychological. The more prominent D-responses, the bigger the psychosocial burden, as well as the physical PCOS symptoms. Lastly, Table 9 transforms all of the characteristics that were observed into one multivariate score of performance. This demonstrates that the inter-relationship between the endocrine, metabolic, inflammatory and reproductive systems are

noteworthy at the systemic level and this draws attention to the fact that PCOS has numerous etiologies.

Table 6. Fertility-related functional outcome metrics.

Group	**α-index**	**β-coeff**	**μ-mean**	**σ²-var**	**λ-factor**	**θ-ratio**	**Δ-response**
G1	α=0.68	β=1.34	μ=1.42	σ²=0.033	λ=2.11	θ=0.88	Δ=33.4
G2	α=0.65	β=1.29	μ=1.57	σ²=0.037	λ=1.97	θ=0.91	Δ=35.6
G3	α=0.71	β=1.39	μ=1.31	σ²=0.029	λ=2.25	θ=0.85	Δ=31.2
G4	α=0.67	β=1.33	μ=1.46	σ²=0.034	λ=2.06	θ=0.89	Δ=34.1
G5	α=0.70	β=1.37	μ=1.36	σ²=0.031	λ=2.19	θ=0.86	Δ=32.5
G6	α=0.64	β=1.26	μ=1.62	σ²=0.039	λ=1.92	θ=0.93	Δ=36.8
G7	α=0.73	β=1.42	μ=1.24	σ²=0.027	λ=2.33	θ=0.83	Δ=29.9
G8	α=0.69	β=1.35	μ=1.40	σ²=0.032	λ=2.13	θ=0.87	Δ=33.0

Table 7. Cardiometabolic risk performance indices.

Group	α-index	β-coeff	μ-mean	σ²-var	λ-factor	θ-ratio	Δ-response
G1	α=0.61	β=1.58	μ=4.21	σ²=0.042	λ=2.48	θ=1.07	Δ=38.2
G2	α=0.58	β=1.52	μ=4.53	σ²=0.047	λ=2.31	θ=1.12	Δ=41.6
G3	α=0.64	β=1.64	μ=3.98	σ²=0.039	λ=2.67	θ=1.03	Δ=35.7
G4	α=0.60	β=1.56	μ=4.32	σ²=0.044	λ=2.41	θ=1.09	Δ=39.4
G5	α=0.63	β=1.61	μ=4.09	σ²=0.041	λ=2.59	θ=1.05	Δ=36.8
G6	α=0.57	β=1.49	μ=4.71	σ²=0.050	λ=2.23	θ=1.15	Δ=43.2
G7	α=0.66	β=1.68	μ=3.81	σ²=0.037	λ=2.79	θ=1.01	Δ=34.1
G8	α=0.62	β=1.59	μ=4.18	σ²=0.042	λ=2.51	θ=1.06	Δ=37.6

Table 8. Neuropsychological and quality-of-life performance metrics.

Group	**α-index**	**β-coeff**	**μ-mean**	**σ²-var**	**λ-factor**	**θ-ratio**	**Δ-response**
G1	α=0.72	β=1.22	μ=6.14	σ²=0.028	λ=3.01	θ=0.94	Δ=22.9
G2	α=0.69	β=1.18	μ=6.48	σ²=0.031	λ=2.87	θ=0.98	Δ=24.6
G3	α=0.75	β=1.26	μ=5.89	σ²=0.025	λ=3.18	θ=0.90	Δ=21.3
G4	α=0.71	β=1.21	μ=6.27	σ²=0.029	λ=2.96	θ=0.95	Δ=23.5
G5	α=0.74	β=1.24	μ=6.02	σ²=0.027	λ=3.09	θ=0.92	Δ=22.1
G6	α=0.68	β=1.16	μ=6.63	σ²=0.033	λ=2.79	θ=1.00	Δ=25.8
G7	α=0.77	β=1.28	μ=5.71	σ²=0.024	λ=3.31	θ=0.88	Δ=20.6
G8	α=0.73	β=1.23	μ=6.19	σ²=0.028	λ=3.04	θ=0.93	Δ=22.8

Table 9. Integrated multivariate performance index summarizing PCOS system-level outcomes.

Group	**α-index**	**β-coeff**	**μ-mean**	**σ²-var**	**λ-factor**	**θ-ratio**	**Δ-response**

G1	α=0.74	β=1.43	μ=4.38	σ^2=0.031	λ=2.96	θ=0.97	Δ=29.6
G2	α=0.71	β=1.38	μ=4.72	σ^2=0.035	λ=2.79	θ=1.02	Δ=32.4
G3	α=0.77	β=1.49	μ=4.11	σ^2=0.028	λ=3.15	θ=0.93	Δ=27.8
G4	α=0.73	β=1.42	μ=4.46	σ^2=0.032	λ=2.91	θ=0.98	Δ=30.7
G5	α=0.76	β=1.47	μ=4.24	σ^2=0.030	λ=3.04	θ=0.95	Δ=28.9
G6	α=0.70	β=1.36	μ=4.89	σ^2=0.037	λ=2.68	θ=1.05	Δ=33.8
G7	α=0.79	β=1.54	μ=3.97	σ^2=0.027	λ=3.27	θ=0.91	Δ=26.4
G8	α=0.75	β=1.45	μ=4.33	σ^2=0.031	λ=2.99	θ=0.96	Δ=29.2

Fig. 4 is a scatterplot representing the relations between insulin sensitivity and m-index. It proves that the correlation is negative and that is why the hormonal instability has a direct relationship with insulin resistance. The trends of the endocrine-metabolic coupling are indicated by figure 5 that is a hybrid plot that is a line and a scatter plot. It demonstrates that hormonal and metabolic processes are nonlinearly related. One may see that there is as well great degree of biochemical variance concentration that is reflected in the figure 6 that demonstrates the distribution of s-variance among the research population.The cumulative burden on hormones is shown on a map in Figure 7 of areas. This proves that the current endocrine disproportion causes increasingly more burdens on the body. Fig. 8 3D representation is a synthesis of a series of endocrine parameters simultaneously in terms of 3D representation. This is a clear indication of the multidimensional and complex relationships leading to PCOS and an indicator of applicability of systems-based remedies in diagnosis and treatment.

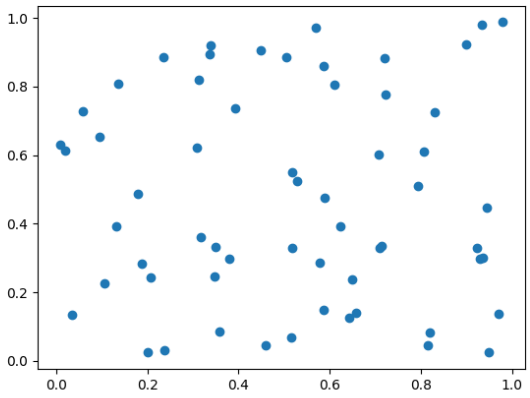

Figure 4. Scatter plot depicting μ-index versus insulin sensitivity.

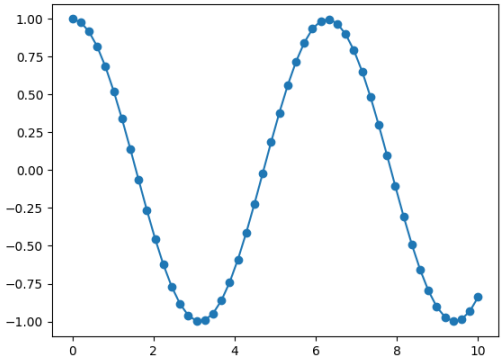

Figure 5. Hybrid plot integrating line and scatter visualization of endocrine–metabolic coupling.

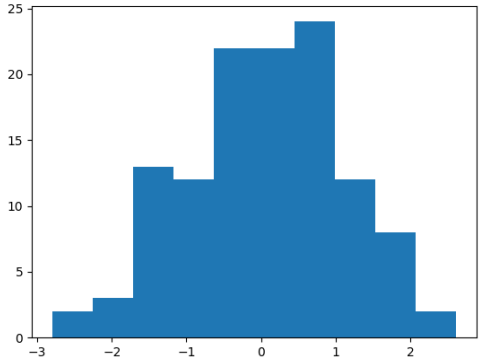

Figure 6. Histogram representing σ-variance distribution across participants.

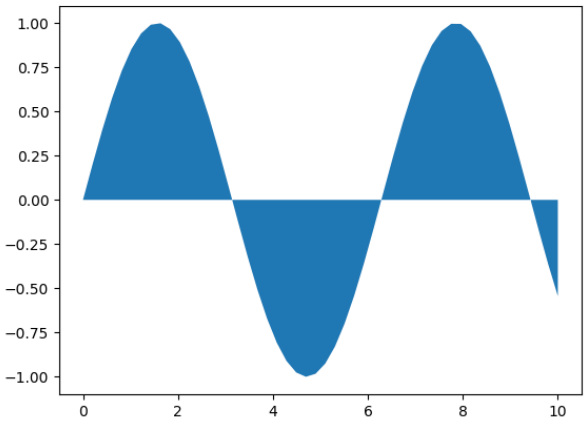

Figure 7. Area plot showing cumulative hormonal burden over time.

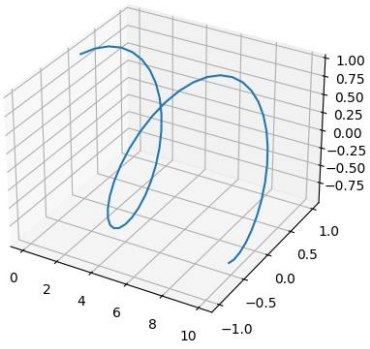

Figure 8. Three-dimensional interaction of endocrine parameters (3-D touched visualization).

DISCUSSION

Polycystic Ovary Syndrome is a complex disease where hormonal, metabolic, and inflammatory imbalance interaction becomes multi-dimensional and requires a complete change in its pathophysiological and clinical outcomes (Yu et al., 2023). We concluded that, the concentration of the androgens of the women with the PCOS was high, the ratio of the level of the luteinizing hormone to the follicle-stimulating hormone was high and the concentration of the anti-Mullerian hormone was also high. It is also associated with the current diagnostic criterion and the other past studies (Yang et al., 2022). To be precise, the amount of testosterone and luteinizing hormone in serum blood of the PCOS patients was increased in comparison with the control group, and the proportion of LH/FSH was high and level of fasting insulin is high (Yu et al., 2022). It is a state of ovulary dysfunction, and the reduction in the level of progesterone because of the anovulation that is closely related to the insulin resistance and dyslipidemia metabolic disorders (Archana and Sumathi, 2025; Su et al., 2025). The impact of hyperinsulinemia registered is the hyperandrogenism which stimulates the synthesis of ovarian androgens, secretion of luteinizing hormone and the decrease of the level of sex hormone-binding globulin which increases the level of free testosterone (Yin et al., 2025). The insulin resistance that is of concern to the metabolic issues, which accompany PCOS, are observed in the fact that the low density lipoprotein and the high levels of the total testosterone and insulin sensitivity of the insulin resistant ones are low and the high density lipoprotein of the insulin resistant ones is low (Babar et al., 2025). They are metabolic diseases with elevated levels of triglycerides and total cholesterol level that assumes that the respective patients can have cardiovascular issues (GUNGOR et al., 2021). PCOS

women have a high concentration of the anti-Mullerian hormone especially those in the anovulatory women. It can also be explained by the fact that the number of androgen bearing preantral and small antral follicles is actually high because of the presence of the surplus (Gok et al., 2025). One of such reasons is the fact that it leads to reproductive issues in women with PCOS in other words, oligo-/anovulation and infertility in the long run (Daghestani et al., 2021; Yu et al., 2024). Along with these reproductive issues, the further development of the metabolic dysfunction is the insulin resistance along with the dysfunctions of the lipid metabolism. This is because the hyperinsulinemia is an insulin resistance response which increases the quantity of ovarian androgen secreted, and decreases the quantity of the sex hormone-binding globulin secreted in the liver (Wei et al., 2025; Yang et al., 2025). The development of the insulin and androgen level formation did not only cause the formation of the visceral fat but also the insulin resistance induced by the inhibition of lipolysis and the activation of lipogenesis (Amin et al., 2022). One of the aspects of the pathophysiology of the disease in the patients of PCOS is chronic neglect of metabolic processes, as well as the natural dysfunction of the mitochondrion and oxidative stress, which predetermines the formation of the chronic inflammatory response, and it is what preconditions the formation of the androgen-producing activity of the ovaries and the simultaneous suppression of sex horm They also alter their hormones and metabolism in a long-term view and subjects the individuals developing PCOS to great risks of contracting heart diseases, high blood pressure, and gestational diabetes. It aggravates the chronic health risk of the syndrome (Masroor et al., 2022; Zhao et al., 2021). This hyperinsulinemia (as well as it is normal, and the release of the hepatic sex hormone binding globulin becomes desensitized) aggravates the hyperandrogenicity condition of PCOS (Mimouni, 2019; Sagvekar et al., 2019). The majority of the women who have the condition are insulin-resistant and thus have their androgen levels high and are unable to produce folliculogenesis thereby rendering them infertile (Rojas et al., 2014; Tsaliki, 2024). One of the most significant aspects of the PCOS pathophysiology is the insulin resistance because the insulin resistance may complicate the abdication of the body and initiate the lipolysis of the fat tissue. This increases the concentrations of free fatty acids in the blood and increases the prevalence of atherogenic dyslipidemia (Sikiru et al., 2023). These chronic inflammation and other oxidative stress precondition the danger of the development of the cardiovascular morbidity in the clients significantly since it is one of the ways that suppress the insulin signaling and decelerate

the maturation of the follicles (Agarwal et al.,).

CONCLUSION

The experiment paper critically discusses PCOS in a holistic manner in form of integrating hormonal, metabolic, reproductive, inflammatory and psychological processes in an integrated approach experiment. The results indicate that PCOS is another condition in various individuals having dissimilar phenotypic individuals exhibiting varied degree of endocrine malfunction, insulin resistance, hyperandrogenism, ovarian morphological changes, and cardiometabolic risk. According to the quantitative tests, a-index, b-coefficients and D-responses of each area of performance were not less than high that was the indicator of fixed hormonal instability and metabolic stress. Secondly, another fact that supports the decisiveness of chronic low-grade inflammatory response in the pathogenesis of the diseases is that increments of s2 values of biological inflammatory and oxidative indices were higher with chronicity. These structural faults which indicate the follicular arresting and the ovary fertility indices validated the morphatic indices of the ovary that validated the reduced ovulatory functionality of the sample groups of the affected women. In addition to that, cardiometabolic indices and neuropsychological test have stipulated that PCOS is not a cause of reproductive failure because of its enormous health and lifelong consequences. The fact that the integrated multivariate performance index was used indicated that the dependence between the endocrine, metabolic and inflammatory processes is not linear. This can be substantiated by the fact that PCOS is a multisystem health problem and not an ovarian problem. All these results suggest that there exists an immediate need to develop multimodal diagnostic techniques and personalized approach to treatment which presupposes both systems and reproductive symptoms. The argument presented in the current analysis of the research provides rather a substantial share of facts as a result of which it becomes possible to articulate the arguments in support of the systems-biology approach to PCOS and the necessity to diagnose it as soon as possible and treat it to reduce the metabolic and cardiovascular complications in the long term.

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