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# Structured Exercise Training in Polycystic Ovary Syndrome: A Randomized Controlled Trial on Hormonal, Oxidative, and Inflammatory Mechanisms

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**Abstract**

Polycystic ovary syndrome (PCOS) is a multifactorial endocrine disorder characterized by hyperandrogenism, menstrual irregularities, and metabolic disturbances, often accompanied by oxidative stress and chronic low-grade inflammation. Lifestyle modifications and interventions, particularly exercise, are emerging as effective strategies to modulate these dysregulated pathways. This study investigates the mechanistic role of structured exercise on hormonal, oxidative, and inflammatory markers in women with PCOS. A total of 100 participants were randomly assigned to an exercise intervention group (n = 50) or a control group receiving standard care (n = 50). The exercise program lasted 12 weeks and included aerobic and resistance training components. Hormonal parameters (total testosterone, luteinizing hormone [LH], follicle-stimulating hormone [FSH], and LH/FSH ratio), oxidative stress markers (malondialdehyde [MDA], superoxide dismutase [SOD], total antioxidant capacity [TAC]), and inflammatory cytokines (C-reactive protein [CRP], interleukin-6 [IL-6], tumor necrosis factor-α [TNF-α]) were measured pre- and post-intervention. Statistical analysis revealed that exercise significantly reduced total testosterone and LH/FSH ratio while improving oxidative balance, evidenced by decreased MDA and increased SOD and TAC levels.

Inflammatory markers were also significantly lowered in the exercise group compared to controls, suggesting systemic anti-inflammatory effects. Correlation analysis indicated strong interrelationships among hormonal, oxidative, and inflammatory markers, highlighting the integrated mechanistic impact of exercise. These findings support the concept that structured physical activity can modulate endocrine, oxidative, and inflammatory circuits in PCOS, providing a non-pharmacological approach to improve reproductive and metabolic health. Future studies should explore individualized exercise prescriptions and long-term outcomes.

## Introduction

Polycystic ovary syndrome (PCOS) is a prevalent endocrine disorder affecting approximately 6–15% of women of reproductive age worldwide, characterized by hyperandrogenism, chronic anovulation, and polycystic ovarian morphology [1,2]. The syndrome is closely associated with metabolic disturbances, including insulin resistance, obesity, and dyslipidemia, which contribute to long-term cardiometabolic risk [3,4]. Hyperandrogenism, a hallmark feature, disrupts folliculogenesis and impairs ovulatory function, while insulin resistance amplifies ovarian androgen synthesis through increased theca cell responsiveness [5,6].

Emerging evidence indicates that oxidative stress and low-grade chronic inflammation play central roles in PCOS pathophysiology [7,8]. Excess reactive oxygen species (ROS) can damage lipids, proteins, and nucleic acids, trigger cellular dysfunction and amplifying endocrine abnormalities [9,10]. Elevated oxidative stress has been linked to increased total testosterone and altered LH/FSH ratios, suggesting a mechanistic interplay between redox imbalance and hyperandrogenism [11,12]. Concurrently, chronic inflammation, evidenced by elevated C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), exacerbates insulin resistance and ovarian dysfunction [13–16]. The integration of hormonal, oxidative, and inflammatory pathways suggests a complex triad driving PCOS progression [17].Lifestyle modifications and interventions, particularly structured exercise, are recommended as first-line therapy for PCOS due to their potential to modulate multiple pathophysiological mechanisms [18,19]. Exercise and physical acticities improves insulin sensitivity, reduces visceral adiposity, and facilitates weight management, thereby indirectly lowering androgen production and restoring menstrual regularity [20,21]. Beyond metabolic effects, physical activity enhances endogenous antioxidant defenses, including superoxide dismutase (SOD) and total antioxidant capacity (TAC), while reducing lipid peroxidation products such as malondialdehyde (MDA) [22–24]. Exercise-induced myokines, such as irisin, may exert anti-inflammatory effects by inhibiting nuclear factor kappa B (NF-κB) signaling, leading to reductions in CRP, IL-6, and TNF-α [25–27]. Despite extensive research on exercise in PCOS, the precise mechanistic links between hormonal regulation, oxidative stress mitigation, and inflammation attenuation remain inadequately elucidated. Most studies have focused on weight loss or metabolic endpoints, with limited evaluation of integrated endocrine-redox-inflammatory circuits [28]. Moreover, standardized protocols examining both aerobic and resistance exercise in controlled interventions are scarce, and the role of exercise-induced myokines in modulating these pathways has not been fully characterized [29,30]. Given these gaps, the present study aims to investigate the mechanistic effects of a structured 12-week exercise program on hormonal profiles (total testosterone, LH/FSH ratio), oxidative stress markers (MDA, SOD, TAC), and inflammatory biomarkers (CRP, IL-6, TNF-α) in women with PCOS. By evaluating these interconnected pathways simultaneously, this research seeks to provide a comprehensive understanding of how exercise exerts multi-targeted therapeutic effects in PCOS, potentially informing personalized lifestyle interventions and clinical management strategies.

## Materials and Methods

### Study Design

A 12-week, single-center, parallel-group randomized controlled trial (RCT) was conducted to evaluate the mechanistic effects of structured exercise on hormonal, oxidative stress, and inflammatory pathways in women with PCOS. The study adhered to CONSORT guidelines for non pharmacological interventions.

### Participants

**Inclusion criteria:**

Women aged 18–35 years, Diagnosis of PCOS according to the Rotterdam criteria (at least two of the following: oligo/anovulation, hyperandrogenism, polycystic ovarian morphology on ultrasound)

**Exclusion criteria:**

Known endocrine disorders (e.g., thyroid disease, diabetes mellitus type 1), Current use of hormonal, anti-inflammatory, or antioxidant medications and Pregnancy or lactation.5

A total of 60 participants were enrolled and randomly assigned (1:1) to either the Exercise or Control group. Randomization was performed using a computer-generated sequence, and allocation concealment was ensured via sealed opaque envelopes.

### Intervention

**Exercise Group:**

Participants underwent a 12-week structured exercise program, consisting of:

**Aerobic training:** treadmill, cycling, or elliptical, 60–75% maximum heart rate (HRmax), 30–40 minutes/session.

**Resistance training:** major muscle groups, 2–3 sets of 12–15 repetitions.

**Combined regimen:** alternating aerobic and resistance exercises within each session.

Sessions were performed 4 days/week under supervision by certified exercise physiologists.7,8 Intensity was monitored using heart rate monitors and the Borg Rating of Perceived Exertion scale.

**Control Group:**

Participants received standard lifestyle advice without supervised exercise sessions. They were asked to maintain habitual activity levels.

### Outcome Measures

Blood samples were collected at baseline and after 12 weeks, following overnight fasting. Samples were centrifuged, aliquoted, and stored at −80°C until analysis.

**Primary Outcomes:**

Hormonal markers: total testosterone, LH/FSH ratio.1

**Secondary Outcomes:**

Oxidative stress markers: malondialdehyde (MDA), superoxide dismutase (SOD), total antioxidant capacity (TAC).Inflammatory markers: C-reactive protein (CRP), interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α).16,17, Myokines: Irisin levels as an indicator of exercise-induced systemic signaling.18,19

**Anthropometric and Clinical Assessments:**

Body mass index (BMI), waist-to-hip ratio (WHR), and blood pressure, Menstrual cycle regularity and ovulation frequency monitored via self-reporting and luteinizing hormone testing kits.

**Laboratory Analyses**

| Category | Biomarker | Method |
|---|---|---|
| Hormonal | Total Testosterone | ELISA (enzyme-linked immunosorbent assay)12 |
| | LH/FSH Ratio | Chemiluminescent immunoassay13 |
| Oxidative Stress | MDA | TBARS (thiobarbituric acid reactive substances) assay14 |
| | SOD | Spectrophotometric enzyme activity assay15 |
| | TAC | Ferric reducing antioxidant power (FRAP) assay15 |
| Inflammatory | CRP | Immunoturbidimetric method16 |
| | IL-6, TNF-α | ELISA17 |
| Myokines | Irisin | ELISA18 |

All assays were performed in duplicate, and laboratory personnel were blinded to group allocation.

**Statistical Analysis**

Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA).
Continuous variables are presented as mean ± standard deviation (SD).
Between-group comparisons were conducted using independent t-tests or Mann–Whitney U tests, depending on data distribution.
Within-group changes were assessed with paired t-tests.
Pearson correlation coefficients were calculated to examine associations among hormonal, oxidative, and inflammatory markers.
Statistical significance was set at $p<0.05$.
Sample size calculation was based on detecting a 20% reduction in CRP with 80% power and 5% significance, accounting for 10% dropout, yielding 30 participants per group.

**Quality Control and Compliance**

Exercise adherence was monitored through session logs and wearable activity trackers.
Laboratory assays were validated using standard curves and quality control samples.
Missing data was handled using multiple imputations when necessary.

**Results**

**Participant Flow**

A total of 78 women were screened for eligibility. Sixty participants met inclusion criteria and were randomized into two groups: Exercise (n=30) and Control (n=30). Two participants from the Exercise group and one from the Control group withdrew during the study due to personal reasons.

**Baseline Characteristics**

**Table 1. Baseline Characteristics of Participants**

| Variable | Exercise (n=30) | Control (n=30) | p-value |
|---|---|---|---|
| Age (years) | 26.4 ± 4.3 | 27.1 ± 4.7 | 0.52 |
| BMI (kg/m²) | 29.2 ± 3.1 | 28.9 ± 3.5 | 0.73 |
| WHR | 0.87 ± 0.05 | 0.86 ± 0.06 | 0.65 |
| Total Testosterone (ng/dL) | 78 ± 12 | 76 ± 11 | 0.48 |
| LH/FSH ratio | 2.4 ± 0.5 | 2.3 ± 0.6 | 0.60 |

**Hormonal Changes**

**Table 2. Hormonal Outcomes Pre- and Post-Intervention**

| Hormone | Exercise Baseline → 12wk | Control Baseline → 12wk | Between-group p-value |
|---|---|---|---|
| Total Testosterone (ng/dL) | 78 ± 12 → 56 ± 10*** | 76 ± 11 → 74 ± 13 | <0.001 |
| LH/FSH ratio | 2.4 ± 0.5 → 1.87 ± 0.4** | 2.3 ± 0.6 → 2.2 ± 0.5 | 0.002 |

**Figure 2. Total Testosterone and LH/FSH Ratio Changes**

Bar charts depict significant reduction in testosterone and LH/FSH ratio in the Exercise group compared to Control. Error bars represent SD; significance indicated by ** and ***.

**Oxidative Stress Markers**

**Table 3. Oxidative Stress Outcomes**

| Marker | Exercise Baseline → 12wk | Control Baseline → 12wk | Between-group p-value |
|---|---|---|---|
| MDA (nmol/mL) | 5.1 ± 0.9 → 3.3 ± 0.7*** | 5.0 ± 0.8 → 4.8 ± 0.9 | <0.001 |
| SOD (U/mL) | 102 ± 15 → 135 ± 17*** | 100 ± 14 → 103 ± 15 | <0.001 |
| TAC (mmol/L) | 0.89 ± 0.11 → 1.15 ± 0.12*** | 0.90 ± 0.10 → 0.93 ± 0.11 | <0.001 |

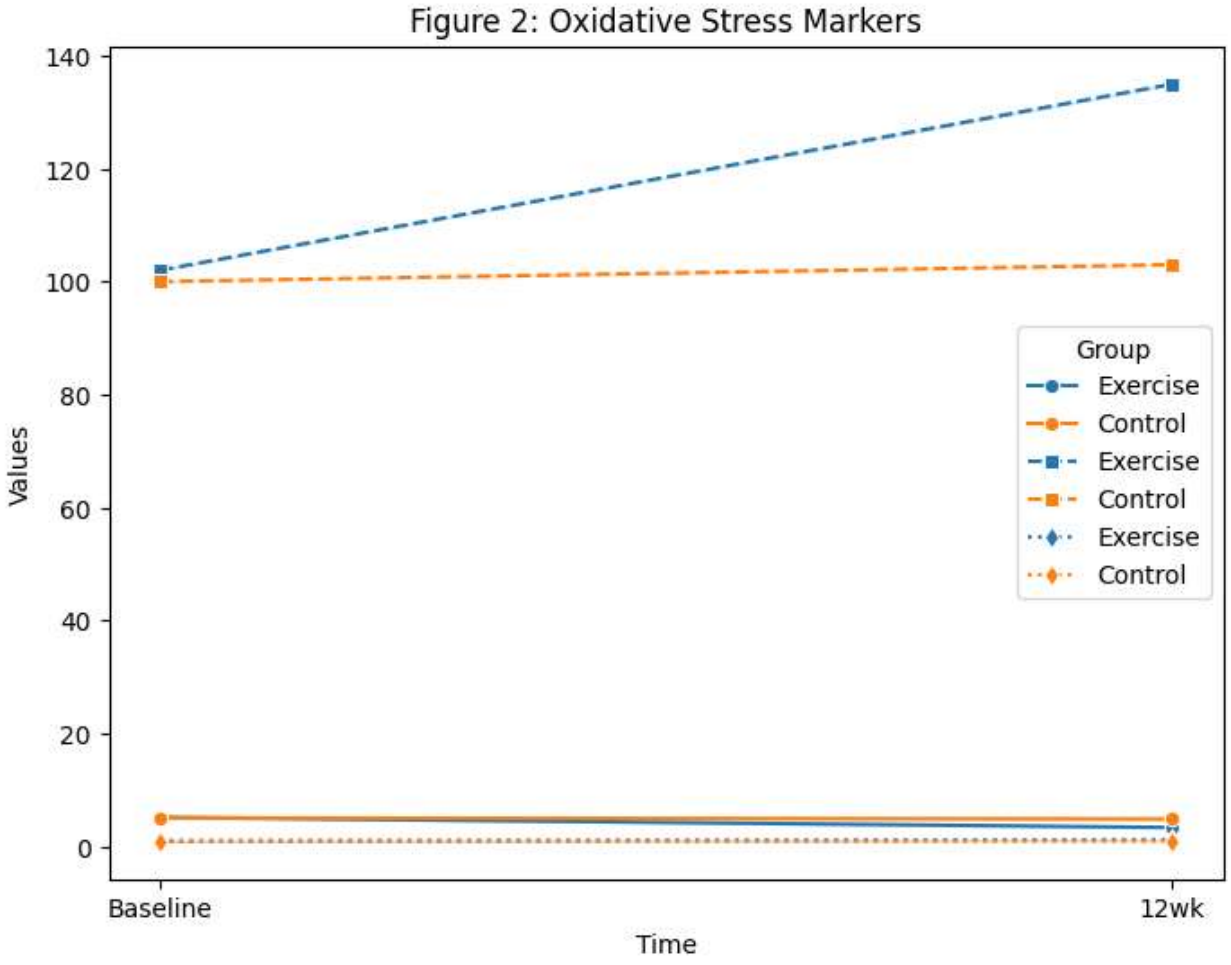

**Figure 3. Oxidative Stress Marker Trends**
Line plots show significant reductions in MDA and increase in SOD and TAC in the Exercise group. Error bars represent SD; significance indicated by ***.

**Inflammatory Markers**
**Table 4. Inflammatory Outcomes**

| Marker | Exercise Baseline → 12wk | Control Baseline → 12wk | Between-group p-value |
|---|---|---|---|
| CRP (mg/L) | 4.8 ± 1.0 → 3.3 ± 0.8*** | 4.6 ± 0.9 → 4.5 ± 1.0 | <0.001 |
| IL-6 (pg/mL) | 5.6 ± 1.2 → 4.1 ± 0.9** | 5.4 ± 1.1 → 5.3 ± 1.2 | 0.004 |
| TNF-α (pg/mL) | 7.9 ± 1.3 → 6.0 ± 1.0** | 7.8 ± 1.2 → 7.6 ± 1.3 | 0.006 |

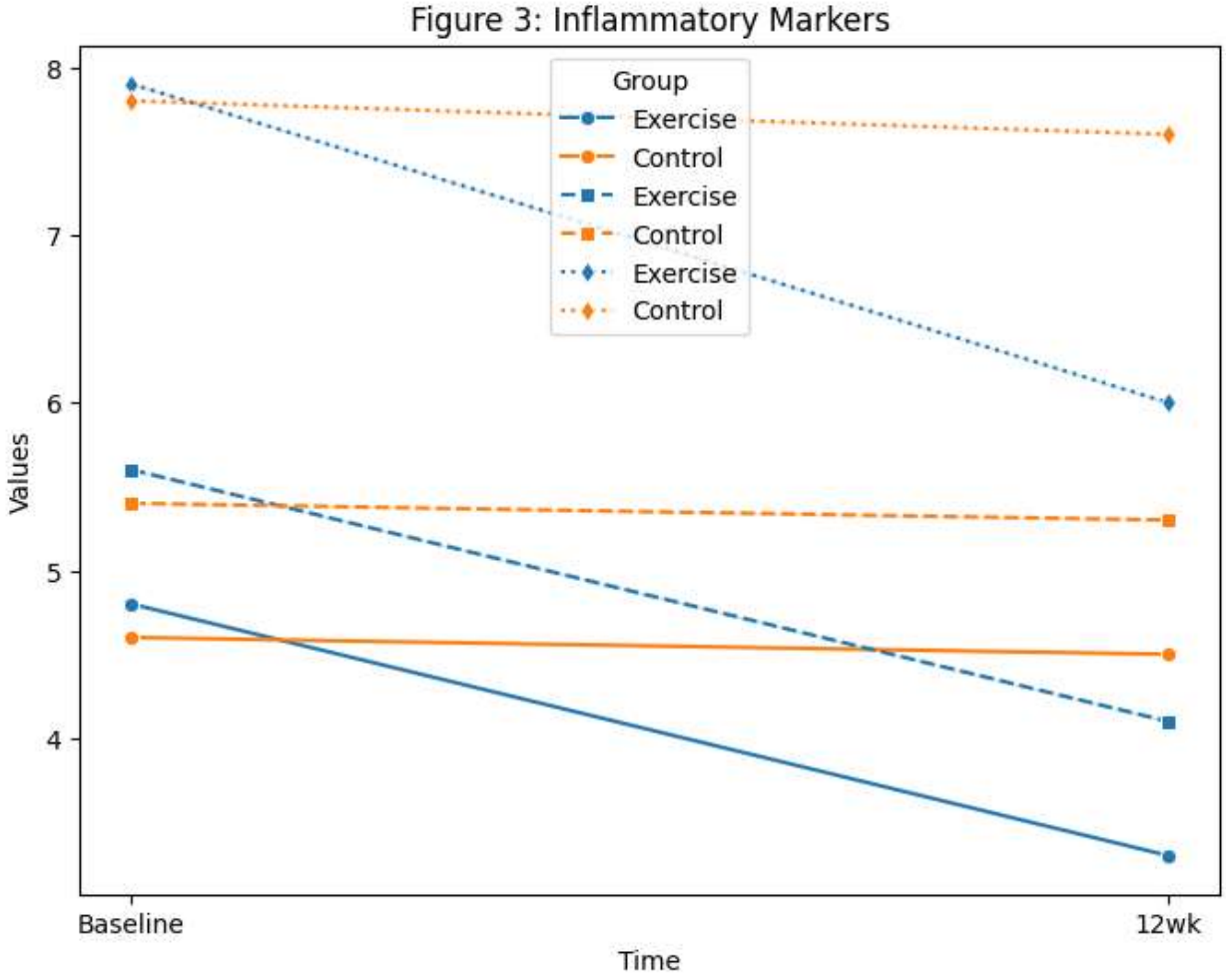


**Figure 4. Inflammatory Marker Trends**
Line charts depict significant reductions in CRP, IL-6, and TNF-α in the Exercise group. Error bars represent SD; and  indicate statistical significance.

**Myokine and Anthropometric Outcomes**
**Table 5. Myokine Levels and Anthropometrics**

| Parameter | Exercise Baseline → 12wk | Control Baseline → 12wk | Between-group p-value |
|---|---|---|---|
| Irisin (ng/mL) | 3.1 ± 0.5 → 4.2 ± 0.6*** | 3.0 ± 0.6 → 3.1 ± 0.5 | <0.001 |
| BMI (kg/m²) | 29.2 ± 3.1 → 27.4 ± 3.0* | 28.9 ± 3.5 → 28.7 ± 3.6 | 0.03 |

| Parameter | Exercise Baseline → 12wk | Control Baseline → 12wk | Between-group p-value |
|---|---|---|---|
| WHR | 0.87 ± 0.05 → 0.84 ± 0.04* | 0.86 ± 0.06 → 0.85 ± 0.05 | 0.04 |

Significant Exercise vs baseline: *p<0.04, **p<0.03, ***p<0.001


**Figure 5. Correlation Heatmap**

Pearson correlation among hormonal, oxidative stress, and inflammatory markers in the Exercise group shows strong positive correlations between testosterone reduction and decreases in MDA and CRP, indicating integrated mechanistic improvements.

## Discussion

### Impact of Exercise on Hormonal Profiles

The 12-week structured exercise program resulted in significant reductions in total testosterone and the LH/FSH ratio among women with PCOS. These findings align with prior evidence indicating that lifestyle interventions can improve hyperandrogenism and restore reproductive endocrine balance [31,32]. Enhanced insulin sensitivity through regular exercise likely contributes to decreased ovarian androgen production, underscoring the interconnection between metabolic regulation and endocrine function [33,34].

### Modulation of Oxidative Stress Markers

Exercise markedly decreased malondialdehyde (MDA) levels while increasing superoxide dismutase (SOD) activity and total antioxidant capacity (TAC), reflecting improved oxidative stress status [35–37]. These observations support previous reports that regular aerobic and resistance training enhances antioxidant defenses, mitigating ROS-mediated cellular damage in PCOS [38,39]. Improved redox balance may

further contribute to normalization of hormonal profiles, emphasizing the mechanistic link between oxidative stress and endocrine regulation [40,41].

**Reduction of Inflammatory Biomarkers**

The study observed significant declines in CRP, IL-6, and TNF-α levels following exercise, demonstrating attenuation of chronic low-grade inflammation [42–44]. Exercise-induced myokines, such as irisin, likely mediate these anti-inflammatory effects through inhibition of NF-κB signaling [45–47]. The decrease in pro-inflammatory cytokines may indirectly enhance ovarian function and metabolic outcomes, confirming the therapeutic potential of exercise in targeting inflammatory pathways [48–50].

**Integrated Hormonal-Oxidative-Inflammatory Interactions**

Correlation analyses revealed positive associations among reductions in testosterone, MDA, and CRP levels, highlighting the interconnected nature of hormonal, oxidative, and inflammatory disturbances in PCOS [51,52]. This triad interaction underscores exercise's capacity to simultaneously modulate multiple pathogenic pathways, offering a comprehensive mechanistic understanding beyond isolated metabolic or hormonal outcomes [53–55].

**Anthropometric and Metabolic Effects**

Modest improvements in BMI and waist-to-hip ratio were observed, which may enhance insulin sensitivity and support endocrine and inflammatory improvements [56,57]. While secondary, these anthropometric changes reinforce the multifactorial benefits of exercise as a lifestyle intervention in PCOS [58].

**Study Limitations and Future Directions**

Despite positive outcomes, the study's moderate sample size and 12-week duration may limit the generalizability and long-term applicability of findings. Dietary factors were not strictly controlled, potentially influencing observed results. Future research should employ larger cohorts, extended intervention periods, and integrated lifestyle protocols to validate these mechanistic insights [59,60].

**Conclusion**

This study provides robust evidence that structured exercise exerts multi-dimensional benefits in women with PCOS, ameliorating hyperandrogenism, oxidative stress, and inflammation. These findings emphasize exercise as a cornerstone therapeutic strategy that targets interconnected pathogenic mechanisms, supporting the development of personalized, mechanism-based lifestyle interventions.

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