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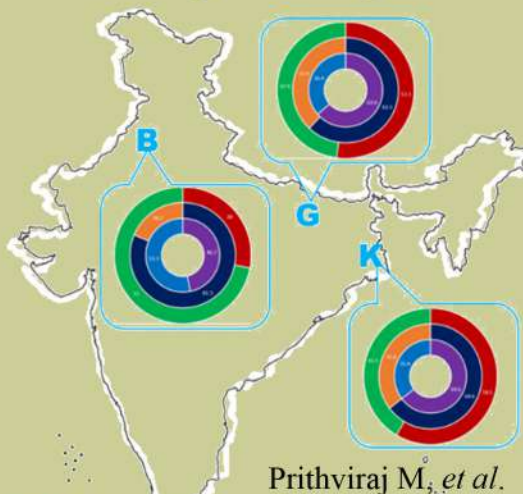
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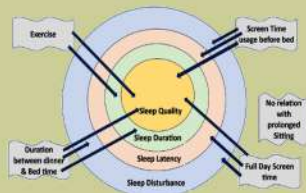
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Male ---Sex--- Female
Abnormal--IAT--Normal
Normal---ISI---Abnormal



Amit S. et al.

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THE PHYSIOLOGICAL SOCIETY OF INDIA (PSI)
Advancing Physiology • Inspiring Health • Shaping the Future

PSI INITIATIVE FOR SCHOOL CHILDREN
QUIZ COMPETITION & DRAWING COMPETITION
Explore, Learn, Compete, Excel!

DATE: 12th July 2026
VENUE: Online (Google Meet)
AGE GROUPS: 10-12 years, 13-15 years, 16-18 years
GOAL: To promote awareness about Physiology and its application in daily life.

CLASSES:
CLASS 10: Fundamentals of Physiology
CLASS 11: Human Physiology
CLASS 12: Advanced Physiology

EVENTS:
QUIZ COMPETITION
DRAWING COMPETITION
PSI FOUNDATION DAY SEMINAR BY UG STUDENTS

THE PHYSIOLOGICAL SOCIETY OF INDIA (PSI)
Advancing Physiology • Inspiring Health • Shaping the Future

PSI FOUNDATION DAY (13th July)
Physiology Unites – PSI Inspires the Future of Better Health!

13th July 2026
To Mark the 92nd Foundation Day

LECTURES BY DISTINGUISHED SPEAKERS:

01	Prof. Suparna Sengul University of Calicut, India	02	Prof. Nandini Kulkarni IISc, India
03	Dr. Dipankar Pal IISc, India	04	Prof. Suman Bhatnagar All India Institute of Medical Sciences, India
05	Prof. S.S. Shankaranarayanan IISc, India	06	Prof. S.S. Shankaranarayanan IISc, India

Topics: Role of Physiology in Human Health, Physiology: Theories and Experimental Evidence, Physiology: Theories and Experimental Evidence, Physiology: Theories and Experimental Evidence.

PSI FOUNDATION WEEK 2026
THE PHYSIOLOGICAL SOCIETY OF INDIA

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Topics: Role of Physiology in Human Health, Physiology: Theories and Experimental Evidence, Physiology: Theories and Experimental Evidence, Physiology: Theories and Experimental Evidence.

THE PHYSIOLOGICAL SOCIETY OF INDIA
Celebrating 92nd Foundation Day

INVITES TEACHERS TO A WORKSHOP ON:
Physiology Education and Teaching:
Way Forward and Improvements

13th July 2026
To Mark the 92nd Foundation Day

Interactive Sessions
Innovative Teaching Strategies
Latest Trends in Physiology Education

Venue: 800 Sankar Auditorium, University of Calicut (Kashipara Campus)
Time: 9:00 am to 5:00 pm

Click here for Registration
Register Now! Free & Mandatory

For More Information:
Dr. Madhuvandana Deshpande | 9845111020 | 9845111020@gmail.com
The Physiological Society of India
Contact: 044-26111111, 044-26111112, 044-26111113, 044-26111114, 044-26111115, 044-26111116, 044-26111117, 044-26111118, 044-26111119, 044-26111120, 044-26111121, 044-26111122, 044-26111123, 044-26111124, 044-26111125, 044-26111126, 044-26111127, 044-26111128, 044-26111129, 044-26111130, 044-26111131, 044-26111132, 044-26111133, 044-26111134, 044-26111135, 044-26111136, 044-26111137, 044-26111138, 044-26111139, 044-26111140, 044-26111141, 044-26111142, 044-26111143, 044-26111144, 044-26111145, 044-26111146, 044-26111147, 044-26111148, 044-26111149, 044-26111150, 044-26111151, 044-26111152, 044-26111153, 044-26111154, 044-26111155, 044-26111156, 044-26111157, 044-26111158, 044-26111159, 044-26111160, 044-26111161, 044-26111162, 044-26111163, 044-26111164, 044-26111165, 044-26111166, 044-26111167, 044-26111168, 044-26111169, 044-26111170, 044-26111171, 044-26111172, 044-26111173, 044-26111174, 044-26111175, 044-26111176, 044-26111177, 044-26111178, 044-26111179, 044-26111180, 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Neurocognitive profiling in Polyendocrine Metabolic Ovarian Syndrome (PMOS): A minireview of current evidence

Soumen Manna*, Saloni Kumari

ABSTRACT

Background: Polyendocrine metabolic ovarian syndrome (PMOS) or polycystic ovary syndrome (PCOS) is a common long-term heterogeneous endocrine disorder characterized by hyperandrogenism, insulin resistance, and ovulatory dysfunction. Evidence suggests that hormonal and metabolic disruptions inherent to PCOS may also impair neurocognitive functioning. **Objective:** This mini-review aims to consolidate existing evidence regarding the cognitive profile of women with PCOS and the impact of hormonal treatments on these outcomes. **Methods:** A comprehensive search was conducted in Web of Science, Scopus, and PubMed for studies published through February 2026 that examined cognitive domains in patients with PCOS. **Results:** PCOS women demonstrated lower performance in executive functioning, attention, visuospatial ability, and verbal memory than healthy controls. Although hypergonadism was expected to be associated with improved male-favoring cognitive tasks, it appears "decremental" to both male- and female-favoring domains in patients with PCOS. The results of pharmacological interventions to modulate testosterone levels in patients with PCOS and cognitive performance are mixed. Some studies have shown significant cognitive improvement using anti-androgens or oral contraceptives, whereas others have shown no significant cognitive gains despite reduced testosterone levels. However, neuroimaging studies have correlated these cognitive deficits with structural and functional brain changes, including reduced brain volumes in the frontal and parietal cortices, decreased white matter integrity, and aberrant functional connectivities. **Conclusion:** Significant cognitive dysfunction is observed in patients with PCOS in addition to reproductive and endocrine problems. Hyperandrogenism and insulin resistance are associated with impaired cognitive function. Clinical management should evolve beyond reproductive health to include the systematic assessment and treatment of neurocognitive and mental health symptoms.

Keywords: Polycystic ovary syndrome, Polycystic ovarian syndrome, Neurocognition, Cognition, Hyperandrogenism

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INTRODUCTION

Polyendocrine metabolic ovarian syndrome (PMOS), the new internationally consensus-driven name for polycystic ovarian syndrome (PCOS), is a long-term, heterogeneous endocrine disorder that affects a high percentage of women of reproductive age. The global health challenge is intensifying, as evidenced by a dramatic 89% increase in prevalence and an 87% rise in disability-adjusted life years (DALYs) between 1990 and 2021.¹ In India, current estimates indicate a pooled prevalence of approximately 11.33% among women of reproductive age.² The scale of the problem is further highlighted by broader regional trends in South Asia, which saw a 246% increase in prevalence between 1990 and 2021 and the highest increase in incidence rates globally.² PCOS is primarily diagnosed using three major sets of criteria that have evolved to standardize the identification of the disorder: (1) National Institutes of Health (NIH) Criteria (1990) was established as the first formal criteria for the syndrome. The NIH criteria require the presence of clinical and/or biochemical signs of hyperandrogenism and ovulatory dysfunction in the form of irregular or absent menstruation.³ (2) The Rotterdam criteria are currently the most widely used criteria in clinical research and diagnosis. A diagnosis under this framework requires the presence of at least two of the following three conditions: a) oligo-anovulation or anovulation, b) clinical or biochemical hyperandrogenism, and c) polycystic ovaries defined via ultrasonography as

Department of Physiology, Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi, India

***Corresponding author:** Soumen Manna, Department of Physiology, Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi, India, Email: drsoumen.manna@gmail.com

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the presence of 12 or more follicles measuring 2–9 mm in each ovary or an increased ovarian volume of more than 10 ml.⁴ (3) The Androgen Excess Society (AES) Criteria (2006) identifies PCOS as a predominantly hyperandrogenic syndrome. It requires the presence of clinical or biochemical hyperandrogenism in combination with either oligo/anovulation or polycystic ovaries.⁵ Patients with PCOS are characterized by hyperandrogenemia in the form of hirsutism, acne, androgenic alopecia, or biochemical evidence of increased testosterone and androstenedione levels; menstrual irregularities, including oligomenorrhea or amenorrhea; and polycystic ovaries on ultrasonography.⁶ In addition, a high percentage of patients with PCOS also display insulin resistance, which

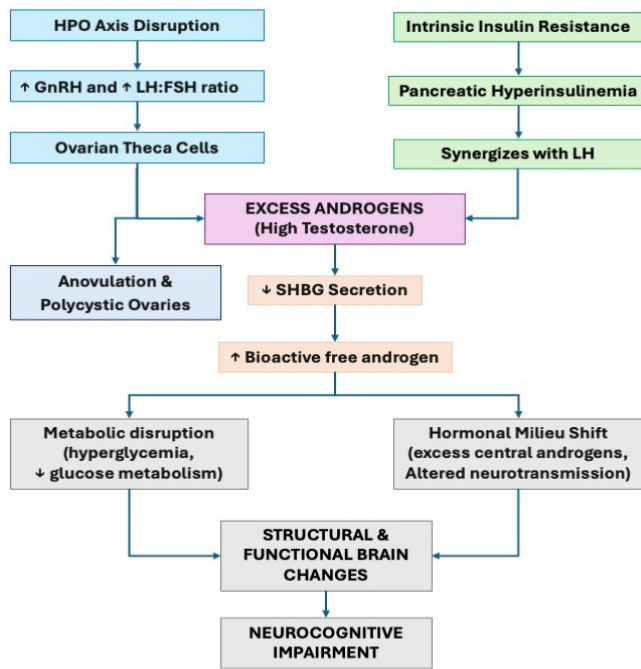


Figure 1: Mechanistic framework linking neuroendocrine and metabolic dysregulation to neurocognitive decline in PCOS

leads to an increased risk of diabetes mellitus (DM). This unfavorable metabolic profile is the main pathogenic factor responsible for hyperandrogenism, characterized by elevated testosterone levels, slightly elevated estrogen levels, and other metabolic manifestations observed in patients with PCOS.^{6,7} In addition to endocrine abnormalities, cognitive function is impaired in patients with PCOS, partly because of hyperandrogenism and hyperestrogenism.^{8,9}

Cognitive functioning comprises multiple domains, including memory, executive functioning, working memory (WM) capacity, attention, perception, and language.¹⁰ There is abundant evidence that sex steroid hormones exert profound effects on brain activity by interacting with receptors in brain regions that are critical for cognitive functions.¹¹⁻¹³ Sexual dimorphism in cognitive task performance has been well documented. Women typically demonstrate higher proficiency in verbal recall, fine motor coordination, and perceptual precision, whereas men tend to show a comparative advantage in visuospatial orientation and mathematical problem-solving.¹⁴⁻¹⁷ Therefore, in PCOS, the characteristic state of hyperandrogenism, often compounded by insulin resistance, disrupts the delicate hormonal milieu of the brain and predisposes patients with PCOS to cognitive deficits (Figure 1).

Guoqing *et al.* showed that postmenopausal women with PCOS had a significantly higher prevalence of cerebral infarction and white matter lesions than healthy controls.¹⁸ Another study by Rees *et al.* using diffusion tensor magnetic resonance imaging (DTI) showed a widespread reduction in axial white matter fibers in the corpus callosum in women with PCOS compared to healthy controls.¹⁹ Cognitive

performance was also poorer in the PCOS group than in the control group. Using functional magnetic resonance imaging (fMRI), Lai *et al.* demonstrated altered functional connectivity in certain brain regions, such as the prefrontal cortex, which is responsible for visuospatial working memory and episodic memory, in patients with PCOS compared with healthy controls.²⁰ Accordingly, multiple studies have shown reduced visuospatial ability in women with PCOS compared to healthy controls.^{21, 22}

Castellano *et al.* demonstrated that women with PCOS had a 10 to 17% lower brain volume, particularly in the frontal and parietal cortices, than that of the control group (23). These women also had significantly lower cerebral metabolic rates of glucose in these brain areas. However, compared to the control group, the PCOS group did not show any significant cognitive impairment.²³ Similarly, Soleman *et al.* (24) showed that women with PCOS used more neural resources during a working memory task than those in the control group. Using functional magnetic resonance imaging (fMRI), the study showed increased activation of the right superior and inferior parietal lobes in women with PCOS compared with the control group during a working memory task. However, task performance in terms of speed and accuracy was similar between the PCOS and control groups.²⁴

Based on the aforementioned studies, it is speculated that abnormal gonadal hormone levels and metabolic abnormalities in PCOS may serve as precursors to altered neurophysiology, ultimately contributing to cognitive deficits in the patient population. Therefore, this mini-review aims to consolidate the available information on the cognitive profiles of women with PCOS.

METHODOLOGY

This review explored the status of cognitive function in women with PCOS. An electronic literature search was conducted independently by two authors using the Web of Science, Scopus, and PubMed databases. The keywords used in the search strategies were "polycystic ovarian syndrome," "polycystic ovary syndrome," "PCOS," "Stein-Leventhal Syndrome," "cognition," "cognitive function," "neurocognition," "cognitive impairment," and "cognitive performance," with Boolean operators to refine and broaden the search scope. This review included all published articles from 2005 to February 2023.

Studies were included if they explicitly examined cognitive domains (e.g., executive function, working memory, attention, visuospatial ability, verbal memory/fluency) or neural correlates (e.g., fMRI, DTI, structural MRI) in women diagnosed with PCOS. Human clinical trials, cross-sectional studies, case-control studies, and randomized controlled trials (RCTs) were included in the review. To filter our search results, the exclusion criteria were: (i) studies not written in English, (ii) exclusive of animal studies, and (iii) those lacking a clear finding related to the cognitive functions of women with PCOS.

The initial screening was performed by evaluating the titles and abstracts of the studies to eliminate duplicates and irrelevant literature. Subsequently, a full-text screening was conducted based on the inclusion and exclusion criteria of this review. Based on the above criteria, 11 unique primary articles met the specific keywords, search parameters, and eligibility boundaries.

The findings of the selected studies were organized to provide an overview of the existing evidence. This organization includes an analysis of patterns and discrepancies in results to understand the current status of cognition in patients with PCOS.

Hormones and PCOS

PCOS is a disease with a complex pathophysiology and unknown etiology. The three primary hormones most strongly linked to the development of PCOS are elevated androgen, estrogen, and anti-Müllerian hormone (AMH).²⁵ Hyperandrogenism (higher testosterone) is often driven by a disruption in the hypothalamus–pituitary–ovarian axis signaling process and abnormal gonadotropin-releasing hormone (GnRH) regulation. This leads to abnormal GnRH pulsatility and an elevated LH-to-FSH ratio, ultimately stimulating the ovaries to overproduce testosterone (Figure 1). Abnormalities in reproductive hormones, such as LH and FSH, with an increased ratio of LH and FSH, and decreased FSH production, are key features in patients with PCOS.²⁶ Another cause could be the intrinsic dysfunction of theca cells or diminished cortisol secretion. Increased LH drives theca cells in the ovaries to produce androgens; however, decreased FSH concentrations and insufficient conversion of androgens to estradiol result in failure to select a dominant follicle and chronic anovulation. This phenomenon typically results in polycystic morphology in the ovaries.²⁷ This is due to decreased cortisol hypothalamic adrenocorticotrophic hormone levels, stimulation of adrenal steroidogenesis, and adrenal hyperandrogenism.²⁵

Anti-Müllerian hormone (AMH) levels are higher in patients with newly diagnosed PCOS than in healthy women of the same age.²⁸ Although elevated AMH levels correlate with the presence of positive preantral and small antral follicles in the ovaries, the exact etiology of higher AMH levels in PCOS remains unknown. However, in patients with PCOS, a positive correlation exists between androgen levels and AMH expression, suggesting that high testosterone levels may drive excessive follicle production, which is a characteristic of this syndrome.²⁹

Insulin resistance and hyperglycemia are other important players in the pathogenesis of PCOS, particularly in obesity-associated PCOS. Regardless of the degree of obesity or androgen levels, women with PCOS exhibit intrinsic insulin resistance. Due to insulin resistance, increased pancreatic insulin secretion directly stimulates the pituitary gland to release excess luteinizing hormone (LH). This surge in LH, combined with insulin's effect on the ovaries, drives excess

androgen secretion and affects folliculogenesis in the ovaries. Elevated insulin and androgen (testosterone) levels inhibit the secretion of sex hormone-binding globulin (SHBG). This decrease in SHBG increases the levels of bioactive free androgens, which are key hormones in the pathophysiology of PCOS.²⁷

Neurocognition in PCOS: Current Evidence

Women with PCOS demonstrate significantly lower cognitive performance in areas such as executive functioning, attention, and visual–spatial ability compared to healthy women without PCOS (Table 1).³⁰ Recent evidence has shown that women with PCOS are less accurate in facial emotion recognition, particularly fear and disgust, than women without PCOS.³¹

Barnard *et al.* (8) conducted a large internet-based study on right-handed women with PCOS (no anti-androgen group, $n = 112$; age, 30 ± 6.4 years, and anti-androgen therapy group, $n = 109$; age, 19 ± 5.6 years) and demonstrated that women with PCOS had impaired performance in comparison to the control group on various male-favoring visuo-spatial tasks, especially in speed and accuracy on word recognition tasks.⁸ In addition, they did not find any differences in the mental rotation test and spatial location tasks between the PCOS and control groups. Therefore, this study did not support the hypothesis that women with PCOS would excel at sexually dimorphic 'male-favoring' cognitive functioning tasks because of hyperandrogenism in PCOS. Sukhapure *et al.* (32) also demonstrated that higher free testosterone levels in women with PCOS ($n = 81$, age 18.7 ± 7.4 years) were significantly correlated with impaired cognitive function, specifically in the domains of psychomotor speed and visuospatial learning.³² Surprisingly, this finding did not support the notion that the PCOS group would outperform the control group in male-favoring tasks. A substantial threshold shift in testosterone levels may be required to exert a measurable impact on spatial cognition in women of reproductive age.³²

However, Schattmann and Sherwin (33) found no statistically significant differences in performance between the PCOS ($n = 22$, age 27.7 ± 5.5 years) and control groups ($n = 29$, age 26.0 ± 6.0 years) in males, favoring visuospatial tasks such as the mental rotation task ($p = 0.04$), although the performance of the PCOS group was worse than that of the control group.³³ However, they also found a statistically significant difference in performance between the PCOS and control groups ($p < 0.05$) on the backward portion of the Block Tapping task, which is a male-favoring task in the working memory domain.³⁴ Moreover, they found that elevated free testosterone levels in PCOS are also detrimental to female-favoring tasks, such as verbal fluency, verbal memory, and manual dexterity, as shown by multiple previous studies.³³ Mehrabadi *et al.* (30) assessed cognitive performance using the Montreal Cognitive Assessment (MoCA) and demonstrated that women with PCOS ($n = 53$, age 28.4 ± 6.2 years) had significantly poorer

Table 1: Summary of Evidence on Neurocognition and Brain Mapping in PCOS

Study & Year	Study Design & Sample Size (n)	Key Variables/Treatments Evaluated	Main Findings	Conclusion
Barnard et al. (2007) ⁶	Internet-based study • PCOS–No anti-androgen (n = 112, age: 30 ± 6.4) • PCOS–Anti-androgen (n = 109, age: 19 ± 5.6)	Hyperandrogenism vs. anti-androgen therapy; male-favoring visuospatial tasks	Impaired speed and accuracy on word recognition tasks. No differences in mental rotation or spatial location	Failed to support the hypothesis that hyperandrogenism in PCOS provides an advantage in male-favoring tasks
Sukhapure et al. (2022) ³²	Clinical study. • PCOS (n = 81, age: 18.7 ± 7.4)	Correlation between free T and cognitive performance	Impaired psychomotor speed and visuospatial learning	Higher free T significantly correlated with poorer cognitive functioning, refuting any male-favoring task advantage
Schattmann & Sherwin (2007) ³³	Case-control study • PCOS (n = 22, age: 27.7 ± 5.5) • Control (n = 29, age: 26.0 ± 6.0)	Performance on male- and female-favoring visuospatial and WM tasks	Significant impairment (p<0.01) in verbal fluency, verbal memory, and fine motor coordination	Elevated free T is highly detrimental to cognitive tasks where women traditionally excel.
Mehrabadi et al. (2020) ³⁰	Cross-sectional study • PCOS (n = 53, age: 28.4 ± 6.2) • Control (n = 50, age: 29.9 ± 6.2)	Overall cognitive profiling using the Montreal Cognitive Assessment (MoCA)	Significantly poorer performance in executive function, attention, and visuospatial abilities.	Validated a broad profile of neurocognitive vulnerabilities in young women with PCOS
Schattmann & Sherwin (2007) ³⁵	RCT • Hormone group (n = 8, cyproterone acetate + estrogen) Placebo group (n = 11)	Pharmacological manipulation / reduction of free T levels over time.	Substantial decline in unbound testosterone levels occurred, but showed no measurable effect on cognitive performance	Drastic changes or reductions in free T did not directly impact or restore cognitive execution in this cohort
Franik et al. (2019) ³⁶	Clinical study. • PCOS (n = 55, age: 17–30)	Impact of sex hormones and metabolic markers on mood and cognition.	Positive correlation between free T and verbal psychomotor speed; androstenedione correlated with worse executive function	Different circulating androgens can exert conflicting, complex effects across separate cognitive sub-domains.
Sukhapure et al. (2022) ³⁷	Treatment study. • PCOS patients (mean age: 29.2)	Pharmacological management with spironolactone (n = 22) and OC (n = 16)	Significant improvements in depression and anxiety, but cognitive improvements were not statistically significant.	Traditional baseline clinical treatments alleviate psychological distress but may fall short of resolving cognitive deficits.
Dinsdale et al. (2023) ³⁸	Comparative clinical study. • PCOS (n = 123, age: 26.8 ± 4.6)	Impact of anti-androgen (spironolactone) medication vs. unmedicated states.	Medicated patients demonstrated higher Reading the Eyes in the Mind Task (RMET) scores.	Hormonal tracking and anti-androgen interventions positively influence and mediate social cognition.
Kumari et al. (2025) ³⁹	Clinical intervention study. • PCOS (n = 35, age: 21.1 ± 4.2)	COC treatment containing levonorgestrel + ethinyl estradiol.	Significant improvements observed across working memory, executive functions, and attention.	COCs can improve multi-domain cognitive performance, though lack of a control group restricted purely isolating the pathology.
Soleman et al. (2016) ²⁴	fMRI & Treatment study • PCOS (n = 14, age: 29.3 ± 5.6) • Control (n = 20, age: 25.6 ± 6.2)	WM task performance during fMRI; pre- and post-treatment (cyproterone acetate + estrogen).	PCOS group showed hyper-activation of the right superior and inferior parietal lobes during tasks. Post-treatment T reduction led to improved task performance.	Women with PCOS recruit more neural resources to achieve matching baseline speeds/accuracy. Anti-androgen therapy successfully optimized cognitive tracking here.
Li et al. (2020) ⁴⁰	Functional MRI study. • PCOS vs. Healthy controls	Resting-state brain activity and functional connectivity.	Decreased amplitude of low-frequency fluctuation (ALFF) in the left middle frontal gyrus (MFG). Increased connectivity between left MFG and left inferior frontal gyrus positively correlated with serum testosterone.	Left MFG alterations may represent the direct underlying neuropathological mechanism causing working memory and executive dysfunction.

T: Testosterone; WM: Working memory; RCT: Randomized Controlled Trial; OC: Oral contraceptives; COC: Combined oral contraceptive

cognitive performance in executive function, attention, and visual-spatial abilities compared to healthy controls ($n = 50$, age 29.9 ± 6.2 years).³⁰

To investigate the effect of pharmacological manipulation of free testosterone on cognition in women with PCOS, Schattmann and Sherwin (35) conducted a randomized placebo-controlled trial on 19 women with PCOS. The women were randomly allocated to the hormone group (HRM) and placebo (PL) group (HRM; $n = 8$, with cyproterone acetate + estrogen and PL; $n = 11$) groups.³⁵ However, the study found that although hormonal treatment (cyproterone acetate + estrogen) in the HRM group (age 29.3 ± 5.6 years) caused a substantial decline in unbound testosterone levels, it did not affect cognitive performance, suggesting that changes in free testosterone did not impact cognitive performance in patients with PCOS. However, Franik et al. reported that higher free testosterone levels were correlated with better verbal psychomotor speed in women with PCOS ($n = 55$, age 17-30). However, they also noted a positive correlation between androstenedione and worse executive functioning scores in women with PCOS.³⁶

In a recent study, Sukhaphure *et al.* showed that when women with PCOS (mean age = 29.2 years) were treated with various medications, predominantly spironolactone ($n = 22$) and oral contraceptives ($n = 16$), there was a significant improvement in depression and anxiety symptoms, although the cognitive function improvement was not significant in comparison to the control group.³⁷ However, a previous study by Soleman *et al.* (24) demonstrated that treatment with cyproterone acetate + estrogen in patients with PCOS ($n = 14$, age 29.3 ± 5.6 years) caused a significant reduction in free testosterone levels, and this reduction was associated with improved performance in comparison to the control group ($n = 20$, age 25.6 ± 6.2 years).²⁴ Similarly, Dinsdale et al. reported that women with PCOS ($n = 123$, age 26.8 ± 4.6 years) treated with antiandrogens (spironolactone) showed higher Reading the Eyes in the Mind Task (RMET) scores than their unmedicated counterparts, suggesting that hormonal treatment influences social cognition.³⁸

In our recent study, we demonstrated that women with PCOS ($n = 35$, age 21.1 ± 4.2 years) treated with a combined oral contraceptive (COC) containing levonorgestrel + ethinyl estradiol showed significantly improved performance in multiple cognitive domains, including working memory, executive functions, and attention.³⁹ However, the lack of a control group in this study restricted our ability to definitively attribute the observed cognitive differences to the pathophysiology of PCOS alone.

The exact pathophysiology of cognitive impairment in patients with PCOS remains unexplored. A recent study using MRI revealed that resting-state brain activity and functional connectivity in women with PCOS were aberrant compared to those in the control group, suggesting that these changes might be linked to cognitive dysfunction.⁴⁰ A decreased amplitude of low-frequency fluctuation (ALFF) of the left

middle frontal gyrus (MFG) was related to poor executive functions in patients with PCOS, and increased functional connectivity between the left MFG and left inferior frontal gyrus was positively correlated with serum testosterone levels. The decrease in ALFF value in the left MFG might be a neuropathological mechanism underlying impaired working memory function in patients with PCOS.⁴⁰

DISCUSSION

Over the past decade, data have shown that patients with PCOS have a high risk of cognitive dysfunction and mental health conditions, particularly depression and anxiety, and decreased quality of life (QoL). Standard clinical guidelines primarily focus on oral contraceptive treatment (OCT) as a first-line therapy for regulating hormonal imbalances.⁴¹ However, OCT alone does not address cognitive impairment or the mental health challenges faced by these patients. Young women with PCOS continue to report mild-to-moderate anxiety, psychological distress, and cognitive difficulties that traditional pharmacological baseline treatments fail to resolve.

The characteristic state of hyperandrogenism and insulin resistance disrupts the brain's delicate hormonal environment in women with PCOS, and studies suggest that elevated free testosterone levels may be detrimental to female-favoring cognitive tasks, such as verbal memory and manual dexterity. Imaging has revealed reduced brain volume in the frontal and parietal cortices, widespread reductions in white matter fiber density, and aberrant resting-state functional connectivity associated with impaired executive function in patients with PCOS. Obesity, which affects a substantial subset of patients with PCOS, fundamentally amplifies their metabolic and cognitive vulnerabilities. Excess visceral adipose tissue undergoes structural remodeling, promoting chronic, low-grade systemic inflammation characterized by elevated circulating levels of pro-inflammatory cytokines.⁴² These peripheral inflammatory mediators can compromise BBB integrity,⁴³ which is highly consistent with neuroimaging studies demonstrating widespread white matter lesions and reduced frontal-parietal volumes in metabolic-heavy PCOS phenotypes.

While some studies have shown that anti-androgen treatment or COC can improve cognitive domains, such as working memory, attention, and social cognition, other trials have found that reducing testosterone levels does not always lead to measurable cognitive improvements. Finally, while the exact pathophysiology remains under investigation, the management of PCOS must evolve beyond reproductive health to include the assessment and treatment of neurocognitive and mental health symptoms of PCOS.

The existing data suffer from heterogeneity due to varying cognitive assessment tools (e.g., MoCA, computer-based tasks, Internet-based testing), heterogeneous diagnostic criteria for PCOS, and small sample sizes. Future research must employ long-term longitudinal tracking of adolescent

and young adult PCOS cohorts from the initial diagnosis onward. Moreover, establishing a standardized, consensus-based neurocognitive battery tailored to endocrine-driven phenotypes is essential to enable cross-study comparisons and meta-analyses. Given the mixed cognitive outcomes observed with traditional oral contraceptive therapies and anti-androgens, future RCTs must be rigorously designed with cognitive performance as a primary or secondary endpoint. Patients should be stratified by distinct PCOS phenotypes (e.g., hyperandrogenic vs. metabolic/insulin-resistant) to evaluate whether insulin-sensitizing agents yield superior neurocognitive recovery compared to or in combination with anti-androgen regimens.

CONCLUSION

Accumulating evidence established over the past decade confirms that PCOS is a complex, multi-system disorder whose manifestations extend far beyond traditional reproductive and metabolic boundaries into the domain of neurocognitive health. Women with PCOS consistently demonstrate significantly lower performance across vital cognitive domains, including executive functioning, attention, visuospatial ability, working memory, and verbal memory. Rather than conferring a “male-favoring” cognitive advantage due to hyperandrogenism, the disruption of the central hormonal and metabolic milieu by elevated free testosterone and intrinsic insulin resistance acts as a distinct neuropathological stressor in women with PCOS. While therapeutic interventions such as COC and anti-androgens successfully downregulate circulating free testosterone, their ability to reverse cognitive deficits remains inconsistent and mixed across clinical trials. Consequently, standard clinical paradigms must rapidly evolve beyond an exclusive focus on metabolic and reproductive health to systematically incorporate neurocognitive and psychological assessments.

AI DECLARATION

The authors acknowledge the use of Gemini 3 to enhance the manuscript's language, grammar, and readability. After using this tool, the authors reviewed and edited the manuscript as needed and took full responsibility for its content.

STATEMENT AND DECLARATIONS

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ETHICAL APPROVAL & CONSENT

Not applicable.

DATA AVAILABILITY

No new datasets were generated in this study. All data used in this study are included in this manuscript.

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PEER-REVIEWED CERTIFICATION

During the review of this manuscript, a double-blind peer-review policy has been followed. The author(s) of this manuscript received review comments from a minimum of two peer-reviewers. Author(s) submitted revised manuscript as per the comments of the assigned reviewers. On the basis of revision(s) done by the author(s) and compliance to the Reviewers' comments on the manuscript, Editor(s) has approved the revised manuscript for final publication.

Gymguard-SCD Clinical Decision Support System

GYM GUARD-SCD: THE FUTURE OF SPORTS CARDIOLOGY AND PREVENTIVE CARDIOLOGY



DR. SUJOY MUKHERJEE,
MBBS, MD (Physiology),
DM Resident, AIIMS Patna

Dr. Sujoy Mukherjee (Inventor):
dr.sujoywbmes123@gmail.com
Mobile: +91-8620937923

SYSTEM AND METHOD FOR PRE-EXERCISE DYNAMIC ELECTRO-AUTONOMIC INSTABILITY DETECTION

Assistant Professor, WBMES

Contact number: +91-8620937923
Email ID: dr.sujoywbmes123@gmail.com

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* GymGuard-SCD is an advanced clinical support system designed for multi-parametric sudden cardiac death risk stratification. This innovative device performs pre-exercise electro-autonomic instability detection, representing the future of sports cardiology and supporting the vision of Atmanirbhar Bharat.