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The Influence of Polyendocrine Metabolic Ovarian Syndrome on Training and Athletic Performance

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Abstract

Background: Polyendocrine Metabolic Ovarian Syndrome (PMOS), previously known as Polycystic Ovary Syndrome (PCOS), is one of the most common endocrine disorders affecting women of reproductive age. While regular physical activity is widely recognized as a cornerstone of PMOS management, increasing evidence suggests that the syndrome itself may influence training adaptations and athletic performance. The characteristic hormonal profile of PMOS, particularly hyperandrogenism, may confer physiological advantages relevant to athletic performance. Increased androgen concentrations have been linked to greater muscle mass, enhanced strength and power production, improved oxygen transport capacity, and favorable adaptations to training, which may contribute to superior athletic performance in some women with PMOS.

Aim: The aim of this narrative review was to summarise and critically discuss the current evidence regarding the relationship between PMOS, training adaptations, and athletic performance.

Material and methods: A narrative review summarising and discussing evidence from observational studies (including case-control and cohort studies), systematic reviews, and meta-analyses published between 2015 and 2026, was conducted. The critical analysis focused on the physiological effects of hyperandrogenism in PMOS, training adaptations, athletic performance, and healthrelated factors influencing exercise capacity in women with PMOS.

Conclusions: PMOS appears to have both beneficial and detrimental effects on exercise performance. While hyperandrogenism may confer certain physiological advantages, metabolic and psychological challenges associated with the syndrome may limit athletic potential. Further longitudinal and mechanistic studies are required to better understand the influence of PMOS on training responses and to develop individualized exercise recommendations for affected athletes.

Keywords: Polyendocrine Metabolic Ovarian Syndrome, Polycystic Ovary Syndrome, PMOS, PCOS, hyperandrogenism, athletic performance, exercise, training adaptations, female athletes

1. Introduction

Polyendocrine Metabolic Ovarian Syndrome (PMOS), previously known as Polycystic Ovary Syndrome (PCOS), is one of the most commonly diagnosed endocrine disorders among women of reproductive age, affecting approximately 10-13% of the female population [1]. The newly introduced nomenclature emphasizes the polyendocrine and metabolic nature of this condition rather than focusing solely on ovarian morphology [2]. PMOS is characterized by a heterogeneous clinical presentation that may include menstrual irregularities, hyperandrogenism, and metabolic disturbances such as obesity and insulin resistance [3].

Recent evidence underscores the importance of modifiable factors in the PMOS, such as diet quality and physical activity [4]. In particular, regular physical activity is considered one of the

fundamental non-pharmacological strategies in the management of PMOS. Exercise has been consistently associated with improvements in insulin sensitivity, body composition, cardiometabolic health, and overall quality of life in women affected by the syndrome [4].

Interestingly, the relationship between PMOS and physical activity appears to be bidirectional. While exercise serves as an important therapeutic tool, the hormonal and metabolic characteristics of the syndrome may themselves influence exercise capacity, training adaptations, and athletic performance [5]. Women with PMOS frequently present with elevated androgen concentrations, altered body composition, and specific metabolic profiles that may affect muscle strength, power production, and responses to training stimuli [5]. These physiological characteristics have led researchers to question whether PMOS may influence not only health outcomes but also differences in sports performance.

Understanding how PMOS influences training adaptations is important not only from a clinical perspective but also in the context of sport and exercise science. As participation of women in recreational and competitive sports continues to increase, there is a growing need to determine whether the hormonal and metabolic characteristics associated with PMOS affect physical performance, recovery, and responses to training stimuli. A better understanding of these relationships may contribute to the development of more individualized exercise recommendations and training strategies for women affected by the syndrome.

2. Materials and Methods

2.1. Literature Search Strategy

To provide a comprehensive overview of the current evidence regarding the influence of Polyendocrine Metabolic Ovarian Syndrome (PMOS), previously known as Polycystic Ovary Syndrome (PCOS), on training adaptations and athletic performance, a structured literature search was conducted using major electronic databases, including PubMed/MEDLINE and Google Scholar. Additional relevant publications were identified through manual screening of reference lists from selected articles and recent international guidelines related to PMOS.

The search strategy was designed to identify peer-reviewed studies published in English between May 2015 and February 2026. Medical Subject Headings (MeSH) and free-text terms were combined using Boolean operators. The search included the following terms: ("Polycystic Ovary Syndrome" OR "PCOS" OR "Polyendocrine Metabolic Ovarian Syndrome" OR

"PMOS") AND ("hyperandrogenism" OR "testosterone") AND ("athletic performance" OR "elite athletes" OR "exercise capacity" OR "physical performance" OR "strength" OR "power" OR "endurance" OR "VO_2max" OR "training adaptation" OR "exercise training").

The search aimed to identify studies investigating the prevalence of PMOS among athletes, the physiological mechanisms of hyperandrogenism, exercise-related adaptations, and factors influencing athletic performance in women with PMOS.

2.2. Inclusion and Exclusion Criteria

The critical overview presented in this narrative review focused on studies reporting of female population diagnosed with PMOS/PCOS according to recognized diagnostic criteria, as well as female athletes presenting features of hyperandrogenism in relation to athletic performance and training adaptations, including muscle strength, muscle power, lean body mass, aerobic capacity (VO_2max), exercise performance, metabolic responses to exercise, and factors potentially limiting performance such as insulin resistance and psychological health.

Original research articles, observational studies (case-control and cohort studies) systematic reviews, and meta-analyses were considered eligible for inclusion. Studies involving animal models, exclusively pharmacological interventions, male participants, or populations not relevant to the objectives of the review were excluded. Articles not available in English or lacking sufficient methodological information were also excluded.

2.3. Data Extraction and Synthesis

Following the literature search, relevant studies were screened and evaluated for their contribution to the objectives of the review. Particular attention was given to evidence examining the relationship between hyperandrogenism and athletic performance, the prevalence of PMOS among elite athletes, physiological mechanisms underlying potential performance advantages, and health-related factors that may impair exercise capacity and training adaptations.

The findings from the retrieved studies were synthesized narratively and organized into thematic sections addressing: (1) the prevalence of PMOS in elite athletes, (2) the pathophysiology of hyperandrogenism, (3) potential performance advantages associated with elevated androgen concentrations, (4) alternative explanations for hyperandrogenism in athletes, (5) health-related challenges affecting athletic performance, and (6) ethical and regulatory considerations surrounding hyperandrogenism in female sport.

3. Results

3.1. PMOS in Elite Female Athletes

Hyperandrogenism is one of the most common endocrine abnormalities observed in female athletes and encompasses a spectrum of conditions characterized by excessive androgen. production or action [5][6]. The principal causes of hyperandrogenism include polyendocrine ovary syndrome (PMOS), idiopathic hyperandrogenism, non-classic congenital adrenal hyperplasia (NCAH), and, less frequently, androgen-secreting ovarian or adrenal tumors. Among these disorders, PCOS is by far the predominant cause, accounting for the majority of cases of hyperandrogenism in women [5]. Recent evidence indicates that hyperandrogenic conditions are overrepresented among elite female athletes compared with the general population. Hirschberg and colleagues reported that PMOS appears to be overrepresented among elite female athletes compared with the general female population, particularly in sports requiring high levels of strength, power, and endurance. Studies of Olympic athletes have further demonstrated an increased prevalence of elevated androgen concentrations and polycystic ovarian morphology compared with non-athletic controls. Importantly, PMOS constitutes the largest proportion of hyperandrogenic diagnoses in elite sports populations. These findings have led to the formulation of the biological selection hypothesis, which proposes that women with naturally higher androgen exposure, such as those with PMOS, may possess physiological characteristics advantageous for athletic performance, including greater muscle mass, increased bone mineral density, enhanced strength, and improved exercise capacity. Consequently, these women may be more likely to excel in competitive sports and progress to elite levels of athletic participation.

3.2. Pathophysiology of Hyperandrogenism in PMOS

The elevated testosterone levels observed in women with polyendocrine metabolic ovarian syndrome (PMOS) result primarily from increased androgen production in the ovaries. Ovarian theca cells, which are responsible for androgen synthesis, exhibit increased sensitivity to luteinizing hormone (LH) and produce excessive amounts of androgens, including testosterone and its precursors [7]. Hyperinsulinemia, commonly associated with insulin resistance in

PMOS, further stimulates ovarian androgen production and acts synergistically with LH to enhance steroidogenesis. In addition, insulin suppresses hepatic production of sex hormone-

binding globulin (SHBG), leading to an increase in circulating free testosterone, the biologically active fraction of the hormone [5]. Although the ovaries are the major source of androgen excess in PMOS, the adrenal glands may also contribute to elevated androgen levels in some patients [7]. The interaction between insulin resistance, hyperinsulinemia, ovarian androgen overproduction, and reduced SHBG creates a self-perpetuating cycle that contributes to the development and maintenance of hyperandrogenism and the metabolic disturbances.

3.3. Potential Performance Advantages Associated with Hyperandrogenism in PMOS

Androgens may improve athletic performance through their effects on multiple physiological systems, including skeletal muscle, bone tissue, erythropoiesis, immune function, and behavioral characteristics, although much of the available evidence originates from studies conducted in men [8]. In skeletal muscle, androgens promote muscle fiber growth and increase the number of satellite cells, myonuclei, and motor neurons. They also stimulate pathways involved in muscle metabolism, including mitochondrial biogenesis, myoglobin synthesis, and insulin-like growth factor (IGF) activity [5]. Together, these adaptations contribute to greater muscle mass, strength, and power, which may provide a competitive advantage in athletic performance. The potential influence of testosterone on athletic performance has also been demonstrated in elite female athletes. In a study including 2,127 observations from the IAAF World Championships, women in the highest tertile of free testosterone concentrations achieved significantly better performances than those in the lowest tertile in several events, including the 400 m (2.73%), 400 m hurdles (2.78%), 800 m (1.78%), hammer throw (4.53%), and pole vault (2.94%) [9, 10]. A further cohort study of 106 Swedish Olympic athletes, muscle mass and strength were positively associated with circulating concentrations of testosterone, dihydrotestosterone (DHT), and androgen precursors. The athletes demonstrated significantly greater lean body mass and superior muscular performance compared with age- and BMI-matched sedentary women [11].

Further evidence supporting the relationship between hyperandrogenism and muscular performance was provided by a recent case-control study conducted in adolescent women with PMOS [12]. The authors reported significantly greater ($p = 0.037$) dominant hand grip strength in participants with PMOS compared with healthy controls. Among the PCOS group, the free androgen index (FAI), anti-Müllerian hormone (AMH), and serum vitamin D concentrations

were identified as significant predictors of muscle strength. Further support for this hypothesis is provided by a case-control study including 44 women with PCOS and 32 age- and BMI-matched healthy controls. Although no differences were observed in body composition, lean body mass, or maximal muscle force output between the groups, women with PCOS demonstrated significantly greater average lower-limb power during both knee extension and flexion movements. Average power during knee extension was approximately 20% higher in the PCOS group (50.3 ± 21.2 vs. 42.1 ± 11.6), while power during knee flexion was nearly 60% higher (35.3 ± 27.0 vs. 22.2 ± 11.1). Furthermore, these measurements were positively correlated with bioavailable testosterone concentrations. The authors concluded that muscle mechanical function is altered in PCOS and that hyperandrogenism may contribute to enhanced lower-limb power independently of differences in muscle mass or body composition [13].

Evidence suggests that hyperandrogenism may influence not only strength-related parameters but also aerobic performance. Testosterone stimulates erythropoiesis, leading to increased hemoglobin concentrations and enhanced oxygen transport capacity. In a study of female endurance athletes, women diagnosed with PCOS achieved significantly better results in the Beep Test and demonstrated higher maximal oxygen uptake (VO_2max) during a treadmill exhaustion test compared with athletes without PCOS matched for body mass index. Furthermore, androgen concentrations were positively correlated with both VO_2max and lean body mass [5]. These findings suggest that the hormonal profile characteristic of PMOS may contribute to favorable adaptations affecting aerobic capacity, body composition, and overall exercise performance.

Cook and colleagues investigated baseline hormonal profiles in a cohort of eighteen female athletes competing at either elite or non-elite levels over a 12-week observation period. Participants represented various sports, including track and field, swimming, cycling, netball, and skeleton, all reported regular menstrual cycles and none used hormonal contraception. The elite group consisted of international-level athletes, whereas the non-elite group competed at the national level, with participants matched according to sport discipline. The study demonstrated substantially higher free testosterone (fT) concentrations among elite athletes compared with their non-elite counterparts (87 vs. 41 pg/mL). In addition, free cortisol levels were also elevated in the elite group (2.90 vs. 2.32 ng/mL). Based on these findings, the authors proposed that elevated testosterone levels could be associated with not only superior performance capacity during high-intensity exercise, but also psychological traits beneficial in sport, including enhanced competitiveness, assertiveness, and dominance. [14].

3.4. The Relationship Between Hyperandrogenism and Elite Sport: Cause, Consequence, or Both?

Although hyperandrogenism is often considered a potential biological predisposition that may contribute to athletic success in women, an alternative hypothesis has been proposed. Some researchers suggest that elevated androgen concentrations may develop as a consequence of long-term exposure to intensive training. Since many female athletes begin systematic training before menarche, the influence of high training loads on pubertal development and menstrual function cannot be excluded. Studies have demonstrated that athletes presenting with oligomenorrhea and hyperandrogenic features exhibit a higher prevalence of delayed puberty. Based on these observations, it has been suggested that hyperandrogenism may represent a physiological adaptation to intensive training. Further evidence supporting this hypothesis was provided by Łagowska and Kapczuk, who examined hormonal profiles in a cohort of Polish athletes and ballet dancers presenting with menstrual disturbances. All participants demonstrated low energy availability (<30 kcal/kg fat-free mass/day) and were categorized according to testosterone concentrations. Elevated testosterone levels were observed significantly more often among ballet dancers than athletes and were associated with an earlier onset of training as well as a longer training history. Notably, despite increased testosterone concentrations, none of the participants exhibited clinical manifestations of hyperandrogenism such as hirsutism, acne, alopecia, or voice deepening. Moreover, common pathological causes of hyperandrogenism, including PMOS, congenital adrenal hyperplasia, Cushing's syndrome, and androgen-secreting tumors, were excluded [15].

Interestingly, individuals with the highest testosterone concentrations also demonstrated the lowest energy intake, carbohydrate consumption, and overall energy availability. Based on these findings, the authors proposed that elevated testosterone levels may represent an adaptive physiological response to chronic energy deficiency. Such a mechanism could help preserve lean body mass and counteract excessive weight loss through the anabolic effects of androgens. In aesthetic sports such as ballet, this adaptation may additionally contribute to maintaining bone health and reducing the risk of stress fractures [15, 16].

This interpretation is supported by studies showing that hyperandrogenic female athletes with menstrual disturbances tend to exhibit a more anabolic phenotype, characterized by greater lean body mass and higher bone mineral density compared with normoandrogenic athletes. Consequently, hyperandrogenism observed in some athletes may not necessarily reflect an

innate predisposition to sporting success but rather a physiological adaptation to prolonged training combined with chronic low energy availability, resulting in a PMOS-like syndrome.

3.5. Challenges Affecting Athletic Performance in Women with PMOS

Despite the potential physiological advantages associated with hyperandrogenism, female athletes with PMOS face numerous health-related challenges that may negatively affect training and competition. Insulin resistance may represent an important barrier to athletic performance, as impaired glucose utilization can negatively affect energy availability, exercise efficiency, recovery processes, and adaptation to training loads. Although regular physical activity is regarded as one of the most effective non-pharmacological interventions for women with PMOS, it does not always completely eliminate insulin resistance. While exercise improves glucose uptake, insulin sensitivity, and overall metabolic health, evidence suggests that abnormalities in insulin signaling may persist even in physically active women and in those with normal body weight [17, 18]. Research has demonstrated that insulin resistance in PMOS is not solely a consequence of obesity but is also linked to intrinsic defects in skeletal muscle insulin signaling and metabolic regulation [18]. Consequently, despite the well-established benefits of exercise training, some women with PMOS continue to exhibit impaired insulin sensitivity, indicating that physical activity alone may be insufficient to fully normalize metabolic function. In a 12-week exercise intervention, Stepto [18] observed improvements in insulin signaling pathways; however, the molecular abnormalities characteristic of PCOS were not completely restored. Similarly, McDonnell [17] concluded that exercise training improves, but does not fully normalize, insulin sensitivity in women with PCOS. These findings highlight the complex pathophysiology of PMOS and suggest that additional interventions, including nutritional strategies and targeted medical treatment, may be required to effectively manage insulin resistance in this population [3, 17, 18].

In addition to metabolic and reproductive disturbances, PMOS is associated with a substantially increased prevalence of mental health disorders. Recent meta-analyses have demonstrated that women with PMOS are at a significantly greater risk of experiencing both depressive and anxiety symptoms compared with healthy controls. Women with PMOS have been reported to have more than fourfold higher odds of moderate-to-severe depressive symptoms and over sixfold higher odds of moderate-to-severe anxiety symptoms, regardless of obesity status [19,20]. Furthermore, a systematic review found that the overall prevalence of depression among women with PMOS is approximately 31-35%, with the risk of depression being more

than 2.8 times higher than in women without the syndrome [20.21]. Psychological distress may negatively influence motivation, training adherence, recovery, and overall athletic performance. Interestingly, animal studies suggest that PMOS-related alterations may also affect spontaneous physical activity. In a prenatal androgen exposure mouse model, animals displaying PMOS-like characteristics voluntarily covered significantly shorter running distances than control mice. As no substantial differences in exercise capacity or muscle morphology were observed, the authors proposed that reduced activity was more likely related to alterations in motivational or rewardrelated mechanisms rather than physical limitations [22].

3.6. Regulatory Perspectives on Hyperandrogenism in Female Sport

The relationship between hyperandrogenism and athletic performance has generated considerable scientific, ethical, and regulatory debate. In particular, discussions have focused on whether naturally elevated testosterone concentrations provide female athletes with a performance advantage significant enough to affect fairness in competition. Disorders of sex development (DSD) have received increasing attention within the sporting community in recent years. DSD refers to a heterogeneous group of congenital conditions involving atypical chromosomal, gonadal, or anatomical sex development (such as complete androgen insensitivity syndrome [CAIS] or 5α-reductase deficiency). Polyendocrine ovary syndrome (PMOS) is another condition associated with elevated androgen concentrations in individuals with a typical female karyotype (46,XX). Unlike DSD, women with PMOS are both genetically and phenotypically female, making this condition an important point of comparison when discussing the effects of endogenous androgens on athletic performance [23].

The International Association of Athletics Federations (IAAF), now known as World Athletics (WA), has repeatedly tried to introduce measures to prevent female athletes with hyperandrogenism. These regulations are based on the assumption that elevated endogenous testosterone concentrations in some female athletes with DSD may confer performance advantages substantial enough to influence competitive outcomes, thereby challenging the principle of fair competition. In healthy women, serum testosterone concentrations typically range from 0.1 to 1.8 nmol/L when measured using liquid chromatography–tandem mass spectrometry (LC-MS/MS). In women with PMOS, testosterone levels may exceed the normal female range and reach up to 3.1- 4,8 nmol/L [10]. Historically, eligibility regulations in female athletics have used testosterone limits of 10 nmol/L and later 5 nmol/L. According to

Handelsman [10], the 5 nmol/L limit was proposed to include women with mild hyperandrogenic conditions, particularly those with polycystic ovary syndrome (PCOS), who are overrepresented among elite athletes, while excluding individuals with untreated hyperandrogenic differences of sex development (DSD), athletes using exogenous androgens, and transgender athletes whose testosterone concentrations remain within the male range. In March 2023, World Athletics introduced substantially stricter eligibility regulations for athletes with DSD. The maximum permitted serum testosterone concentration was reduced from 5.0 to 2.5 nmol/L, and athletes became required to maintain testosterone levels below this threshold for at least 24 months before being eligible to compete in the female category. In addition, the regulations were extended to all female track and field events, whereas previous rules applied only to selected disciplines. Importantly, these regulations do not apply to women with PMOS. Therefore, women with PMOS generally remain eligible to compete in female sporting events despite exhibiting higher testosterone concentrations than healthy women [24].

4. Discussion

4.1. Controversies and ethical considerations

These regulations remain highly controversial among researchers, ethicists, and sporting communities. This debate raises an important question: how can talent be separated from natural biological ability? Elite sport is fundamentally based on biological variation, and athletes frequently benefit from innate characteristics that contribute to superior performance. The critical question is not simply whether an advantage exists, but whether that advantage is relevant to the criteria upon which a sporting category has been established. Consequently, some scholars argue that restricting participation on the basis of naturally occurring hormone levels may unfairly penalize athletes for biological traits over which they have no control and may contribute to feelings of stigmatization and exclusion from competitive sport.

4.2. Areas for Future Investigation

Additional research is required to determine whether the apparent performance advantages associated with hyperandrogenism result directly from elevated androgen exposure or are influenced by other physiological and environmental factors. The current body of evidence is limited by relatively small sample sizes; therefore, larger-scale studies are required to validate

existing findings and better characterize the effects of PMOS on training adaptations and athletic performance.

Finally, further investigation is needed to inform evidence-based sports policies regarding hyperandrogenism in female athletes. A better understanding of the physiological effects of endogenous testosterone may contribute to the development of regulations that balance competitive fairness with athlete inclusion and well-being.

Conclusions: The physiological effects of elevated androgen concentrations appear to provide several potential performance advantages. Hyperandrogenism has been associated with greater lean body mass, enhanced muscle strength and power, increased oxygen transport capacity, and improved aerobic performance. These characteristics may facilitate favorable training adaptations and contribute to superior athletic performance in some women with PMOS.

Despite the potential performance benefits, women with PMOS may also face important challenges that can negatively affect training and competition. Persistent insulin resistance, metabolic dysfunction, and the increased prevalence of anxiety and depressive symptoms may impair exercise capacity and long-term adaptation to training. Therefore, the overall impact of PMOS on athletic performance is likely determined by the interaction between its advantageous and detrimental physiological effects.

Finally, the role of endogenous testosterone in female sport remains the subject of ongoing scientific and ethical debate. While current World Athletics regulations primarily target athletes with differences of sex development (DSD), women with PMOS continue to raise important debates about biological variation and natural physiological predisposition.

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