QUALITY IN SPORT
eISSN 2450-3118 · Open Access · Peer-reviewed
apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

STENCELEWSKI, Damian, PAWLICKI, Jakub, GORZYNSKA-SCHULZ, Aleksandra, GORZYNSKA, Natalia, FALIS, Michał, NIERYCHLEWSKA, Alicja, BRONISŁAWSKA, Agata, BRONISŁAWSKI, Robert, SOLECKA, Alicja, and KIEŁBRATOWSKA, Lidia. Hyperandrogenism in Female Athletes: When Dermatological Signs Become an Endocrine and Sports Medicine Challenge. Quality in Sport. 2026;76:75440. https://doi.org/10.12775/QS.2026.76.75440

ARTICLE TIMELINE
Received: 18.08.2026. Revised: 30.08.2026. Accepted: 12.09.2026. Published: 23.09.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).
Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

Hyperandrogenism in Female Athletes: When Dermatological Signs Become an Endocrine and Sports Medicine Challenge

Damian Stencelewski[1] (D.S.)
e-mail: damian.sten777@gmail.com
ORCID: https://orcid.org/0009-0002-7359-5562

Jakub Pawlicki[2] (J.P.)
E-mail jakub.pawlicki96@gmail.com
ORCID: https://orcid.org/0009-0007-3888-4672

Aleksandra Gorzynska-Schulz[3] (A.G.S.)
e-mail: amjgorzynska@gmail.com
ORCID https://orcid.org/0009-0005-1719-8548

Natalia Gorzynska[4] (N.G).
e-mail: nataliagorzynska05@gmail.com
ORCID https://orcid.org/0009-0007-3891-9636

Michał Falis[1] (M.F.)
e-mail: michaljakubfalis@gmail.com
ORCID: https://orcid.org/0009-0002-1074-6036

Alicja Nierychlewska[2] (A.N.)
e-mail: alicjanierychlewska99@gmail.com
ORCID: https://orcid.org/0009-0009-6783-4601

Agata Bronisławska[2] (A.B.)
e-mail: agata.blach2@gmail.com
ORCID: https://orcid.org/0009-0003-3833-0638

Robert Bronisławski[2] (R.B.)
e-mail: robertbronislawski@gmail.com
ORCID: https://orcid.org/0009-0004-6494-4307

Alicja Solecka[1] (A.S.)
e-mail: alicjasolecka222@gmail.com
ORCID: https://orcid.org/0009-0001-0688-309X

Lidia Kiełbratowska[4] (L.K.)
e-mail: Lidkie03@gmail.com
ORCID https://orcid.org/0009-0000-6322-7446

Affiliation:
1 University Clinical Centre, Gdańsk, Poland
2 St. Wojciech Hospital, Jana Pawła II Avenue, 80-462 Gdańsk, Poland
3 Medical University of Gdańsk, Poland
4 Pomeranian Medical University, Szczecin, Poland

Corresponding author:
Damian Stencelewski
damian.sten777@gmail.com

ABSTRACT

Background. Hyperandrogenism in female athletes represents a complex intersection of endocrinology, dermatology, and sports medicine, where visible dermatological markers often signal underlying endocrine dysfunction or exogenous exposure.

Aim. This narrative review analyzes the pathophysiology, differential diagnosis, and clinical management of hyperandrogenism in female athletes, distinguishing between endogenous endocrine disorders, exercise-induced adaptations, and performance-enhancing drug use.

Material and methods. A literature search of PubMed and Google Scholar was conducted to identify recent clinical guidelines, endocrinological studies, and sports medicine literature regarding female hyperandrogenism, dermatological manifestations, and RED-S.

Results. Dermatological signs such as severe acne, hirsutism, and female pattern hair loss serve as critical clinical red flags. Differentiating pathological conditions like Polycystic Ovary Syndrome (PCOS) from Relative Energy Deficiency in Sport (RED-S) and exogenous androgen exposure is essential for accurate diagnosis and regulatory compliance in sports medicine.

Conclusions. Effective management of hyperandrogenism in female athletes requires a multidisciplinary approach, combining dermatological evaluation, rigorous endocrine profiling, and sports medicine oversight to safeguard both athletic health and long-term well-being.

Keywords: hyperandrogenism, female athletes, hirsutism, relative energy deficiency in sport

1. Introduction

Hyperandrogenism in female athletes represents a complex intersection of endocrinology, dermatology, and sports medicine (1,2). While elevated androgens are classically evaluated through conditions like Polycystic Ovary Syndrome (PCOS), their manifestation in female athletes introduces unique clinical and ethical challenges.Dermatological signs—such as severe acne, hirsutism, and female pattern hair loss—frequently serve as initial red flags (1,3). In high-performance sport, these markers require careful contextualization, as they may stem from endogenous pathologies, physiological training adaptations, or the exogenous use of performance-enhancing androgenic substances.Distinguishing pathological hyperandrogenism from exercise-induced disruptions, such as Relative Energy Deficiency in Sport (RED-S), is vital to prevent misdiagnosis. This review examines the pathophysiology, clinical presentation, and differential diagnosis of hyperandrogenism in female athletes, offering a practical framework for clinicians navigating athletic performance and long-term health(2,5).

2. Androgen Physiology in the Female Athlete

The androgen profile of a female athlete is a dynamic physiological state defined by the interplay between the hypothalamic-pituitary-gonadal (HPG) axis, adrenal androgen production, and peripheral tissue conversion(4). Unlike the sedentary population, the female athlete operates under the influence of chronic physical stress, metabolic flux, and fluctuating energy availability, all of which modulate androgen synthesis and bioavailability(1-4)(figure1). In healthy females, circulating androgens—primarily testosterone (T), androstenedione (A4), and dehydroepiandrosterone sulfate (DHEA-S)—are derived from the ovaries (approx. 50%) and the adrenal glands (approx. 50%). Testosterone circulates largely bound to sex hormone-binding globulin (SHBG) and albumin, with only a small fraction (1–2%) remaining as "free" or bioactive testosterone. In athletes, this balance is frequently altered: intense training can induce transient elevations in total testosterone, but it may also shift SHBG levels, complicating the interpretation of total androgen concentrations(3,7,8).

Peripheral tissues act as a critical metabolic hub, where enzymes like 5α-reductase convert T into the significantly more potent dihydrotestosterone (DHT). The expression and activity of these enzymes in hair follicles and sebaceous glands determine the clinical phenotype of hyperandrogenism, even when systemic androgen levels remain within the high-normal range. Consequently, the physiological threshold for "excess" is not merely defined by blood serum values but by the tissue-specific sensitivity and the efficacy of local steroid metabolism. Understanding this physiology is paramount, as the athletic body’s adaptation to training—

often involving suppressed gonadotropin-releasing hormone (GnRH) pulsatility—may mask or exacerbate these endocrine signals, making the distinction between physiological adaptation and pathological dysregulation a critical diagnostic challenge (3,9).

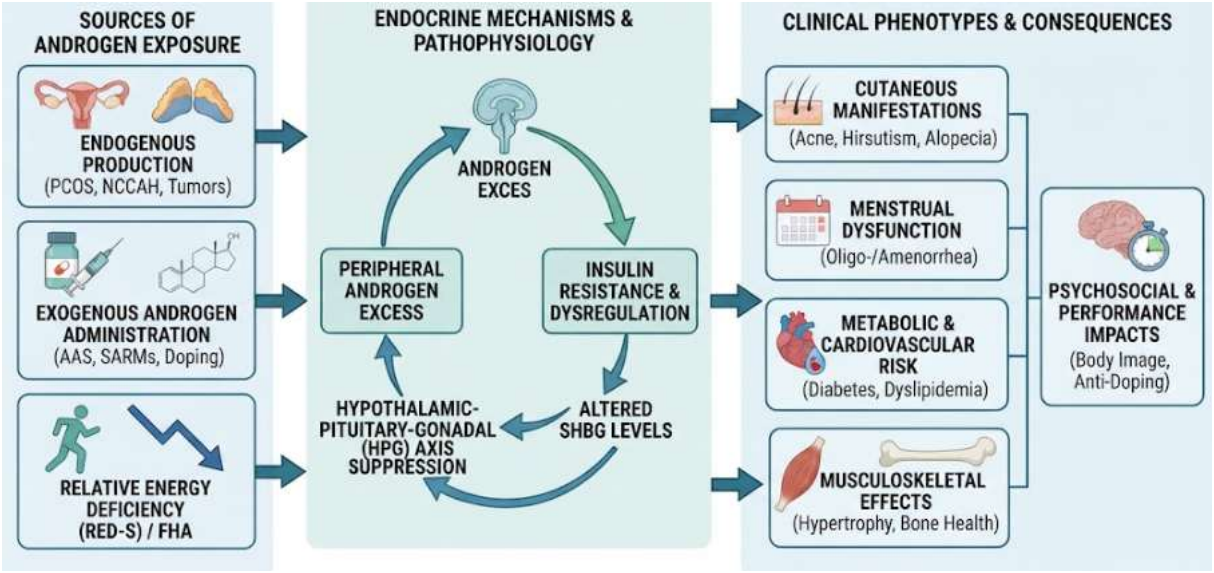

Figure 1. Conceptual framework linking androgen exposure, endocrine mechanisms, and clinical phenotype in female athletes (2,7,10).

3. Dermatological Manifestations

Dermatological signs are frequently the earliest visible indicators of hyperandrogenism in female athletes, acting as cutaneous windows into systemic endocrine status. Because the skin and its appendages (sebaceous glands and hair follicles) are highly responsive target tissues for androgens, alterations in systemic hormone levels or local tissue sensitivity manifest rapidly in these structures. In the athletic population, these clinical features require careful interpretation, as intense physical training, sweat accumulation, occlusion from athletic gear, and metabolic shifts can either mimic, trigger, or exacerbate cutaneous pathologies(1,8,11).

Acne

Acne vulgaris in the context of hyperandrogenism is driven by androgen-stimulated sebaceous gland proliferation and increased sebum production. Androgens bind to nuclear receptors in sebocytes, promoting lipogenesis and altering the lipid composition of sebum, which creates a favorable environment for *Cutibacterium acnes* proliferation and subsequent follicular hyperkeratinization(4,7).

In female athletes, acne often presents or worsens along the jawline, chin, neck, and back. A high index of suspicion for underlying hyperandrogenism is warranted when acne is:

Severe, cystic, or treatment-resistant to conventional topical therapies and standard systemic antibiotics.

Accompanied by other stigmata of androgen excess, such as hirsutism or menstrual irregularities.

Aggravated by certain nutritional supplements or performance-enhancing substances (e.g., anabolic-androgenic steroids or high-glycemic sports formulations).

Hirsutism

Hirsutism is defined as the excessive growth of terminal hair in androgen-dependent areas (upper lip, chin, chest, upper and lower abdomen, back, and inner thighs) following a male-like distribution pattern. It is evaluated clinically using the modified Ferriman-Gallwey (mFG) scoring system, where a score threshold (typically ≥ 8 in many populations, though lower cutoffs are sometimes applied depending on ethnicity) indicates pathological hirsutism(10,11,12,13).

In female athletes, hirsutism is one of the most specific markers of elevated circulating androgens or heightened peripheral 5-alpha-reductase activity. Unlike transient physiological changes, structural terminal hair growth reflects chronic androgen exposure(8,10,12,14,15). Clinicians must differentiate between mild, familial/ethnic variations in hair growth and true pathological hirsutism driven by ovarian disorders (such as Polycystic Ovary Syndrome) or adrenal hyperandrogenism(13,14,16).

Female Pattern Hair Loss

Female pattern hair loss (FPHL) is characterized by a gradual reduction in hair density, typically starting at the central parting and progressing to diffuse thinning over the crown, while characteristically sparing the frontal hairline(12,14,15).

Androgens contribute to FPHL by inducing the miniaturization of hair follicles, progressively shortening the anagen (growth) phase and prolonging the telogen (resting) phase. In female athletes, FPHL can be compounded by physiological stressors. Relative Energy Deficiency in Sport (RED-S) and nutritional deficiencies (such as iron or zinc depletion common in endurance athletes) can induce telogen effluvium, which may overlap with or accelerate androgen-driven hair loss. Therefore, comprehensive evaluation must isolate endocrine-mediated follicular miniaturization from stress- or nutrient-induced shedding(9,15-17).

Acanthosis Nigricans

Acanthosis nigricans presents as symmetric, hyperpigmented, velvety thickening of the skin, most commonly found in skin folds such as the axillae, posterior neck, groin, and inframammary regions(12,14).

While less directly driven by androgens themselves, acanthosis nigricans serves as a classic cutaneous marker of severe **hyperinsulinemia**. Insulin acts synergistically with luteinizing hormone (LH) to stimulate ovarian androgen production and simultaneously suppresses hepatic synthesis of Sex Hormone-Binding Globulin (SHBG), thereby increasing the bioavailability of free circulating testosterone. In female athletes, the presence of acanthosis nigricans—even amidst low body fat percentages—strongly points toward underlying insulin resistance, metabolic dysregulation, or metabolic-associated phenotypes of Polycystic Ovary Syndrome (PCOS)(2,10).

4. Menstrual Dysfunction and the Differential Diagnosis with RED-S

Menstrual dysfunction is a frequent clinical finding in female athletes, ranging from subclinical ovulatory disturbances to oligomenorrhea and primary or secondary amenorrhea. In the context of hyperandrogenism, tracking menstrual regularity is essential, as the ovaries are both primary producers of androgens and the organs most sensitive to systemic hormonal imbalances. However, diagnosing the root cause of menstrual disruption in athletes presents a complex diagnostic puzzle due to the overlapping phenotypes of endocrine pathology and exercise-induced energy deficits(12,15). The primary differential diagnostic challenge lies in distinguishing Polycystic Ovary Syndrome (PCOS)—the most common endocrine cause of hyperandrogenism—from Relative Energy Deficiency in Sport (RED-S) and its precursor, functional hypothalamic amenorrhea (FHA). PCOS is characterized by chronic oligo- or anovulation, clinical or biochemical signs of hyperandrogenism, and/or polycystic ovarian morphology on ultrasound, stemming from intrinsic ovarian and neuroendocrine dysregulation often compounded by hyperinsulinemia. Conversely, RED-S and FHA are caused by chronic low energy availability (LEA), where dietary energy intake is insufficient to support the energy expenditure required for training, downregulating the hypothalamic-pituitary-gonadal axis and suppressing pulsatile GnRH secretion(17)(table1).

Differentiating between these two entities is notoriously difficult in athletic populations due to overlapping phenotypes, complex androgen profiles, and dual pathological states where an athlete may possess an underlying predisposition to PCOS while simultaneously experiencing RED-S due to rigorous weight-class or aesthetic pressures. Both conditions carry severe long-

term consequences, with PCOS increasing cardiometabolic and endometrial risks, while RED-S severely compromises bone mineral density, endothelial function, and psychological well-being. Consequently, accurate diagnosis requires a meticulous history assessing dietary intake, training volume, weight history, and the timeline of menstrual changes relative to training shifts, alongside targeted biochemical screening including gonadotropins, estradiol, thyroid function, and metabolic markers to isolate whether menstrual dysfunction stems from hyperandrogenic ovarian drive or central hypothalamic suppression(15,18).

Phenotype	**Key Signs**	**Primary Cause**	**Main Distinction**
PCOS	Acne, hirsutism, irregular cycles	Ovarian excess, insulin resistance	Androgen excess & metabolic overactivation
RED-S / FHA	Amenorrhea, low bone density	Low energy availability	Energy deprivation & low/normal androgens
Exogenous Doping	Rapid virilization, voice deepening	AAS or SARM use	Profoundly suppressed LH, FSH, and SHBG
NCCAH	Hirsutism, acne mimicking PCOS	Partial 21-hydroxylase defect	Elevated morning 17-OHP levels
Androgen Tumors	Rapid-onset severe virilization	Neoplastic production	Total testosterone > 200 ng/dL

Table 1. Clinical Phenotypes and Differentiation.

5. PCOS in the Athletic Population

Polycystic Ovary Syndrome (PCOS) is the most prevalent endocrine disorder among women of reproductive age, affecting an estimated 8% to 13% of the female population(19), yet its specific presentation and implications within the athletic cohort remain uniquely nuanced(figure 2). Diagnosing PCOS in female athletes is frequently confounded by physiological adaptations to rigorous physical training. For instance, endurance and strength training can naturally induce skeletal muscle remodeling and metabolic improvements that mask or alter classical metabolic symptoms, while strenuous exercise can also lead to morphological changes in the ovaries—such as an increased follicle count—that overlap with the ultrasound criteria for polycystic ovarian morphology (PCOM) in eumenorrheic athletes(13,18).

The clinical manifestation of PCOS in athletes often diverges from the sedentary phenotype. While the classic presentation involves insulin resistance, central adiposity, and metabolic syndrome, athletic women with PCOS frequently present with a lean or metabolically favorable body composition, driven by high daily energy expenditures. However, these athletes still experience the core endocrine hallmarks: hyperandrogenism (clinical manifestations like persistent acne and hirsutism) and ovulatory dysfunction. This creates a paradox where an athlete may exhibit elite physical performance and low body fat percentage while simultaneously struggling with debilitating hyperandrogenic symptoms, anovulatory subfertility, and chronic fatigue related to underlying metabolic inflexibility(20).

Furthermore, managing PCOS in competitive athletes requires carefully balancing endocrine therapy with sports regulations and performance goals. Standard pharmacological treatments, such as oral contraceptive pills (OCPs) used to regulate cycles and suppress ovarian androgen production, can negatively impact parameters like maximal oxygen uptake, plasma volume, and lean mass accrual, or raise concerns regarding anti-doping compliance if substances contain prohibited progestins(3,18,20). Consequently, therapeutic strategies often prioritize lifestyle modifications—optimizing macronutrient intake, tailoring resistance and endurance training blocks, and utilizing insulin-sensitizing agents like metformin—to manage hyperandrogenism and restore regular menstrual cycles without compromising athletic output or violating sports governance frameworks(1,10,13).

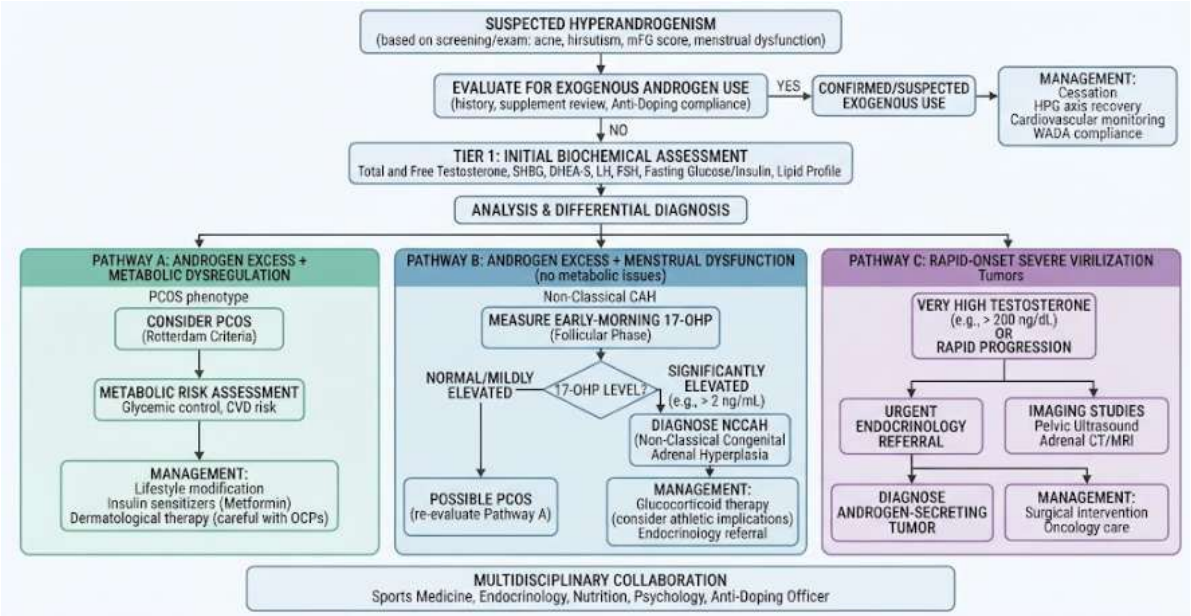

Figure 2. Practical diagnostic pathway for suspected hyperandrogenism in a female athlete.

6. Performance-Enhancing Androgen Exposure

The landscape of hyperandrogenism in female athletes is uniquely complicated by the potential exogenous administration of performance-enhancing androgens, predominantly anabolic-androgenic steroids (AAS), selective androgen receptor modulators (SARMs), and exogenous testosterone. Within elite and competitive sports, the use of these substances introduces a critical diagnostic distinction: differentiating between endogenous pathological hyperandrogenism (such as PCOS or congenital adrenal hyperplasia) and exogenous doping-induced androgen excess. Uncovering this etiology is essential not only for medical management and safeguarding long-term athlete health, but also for addressing anti-doping compliance and regulatory frameworks governed by bodies such as the World Anti-Doping Agency (WADA)(13,17).

Exogenous androgen exposure profoundly disrupts the female endocrine system, frequently producing a clinical picture that mimics or drastically magnifies endogenous hyperandrogenism(15,17). Dermatological signs often appear with rapid onset and extreme severity, including adult-onset cystic acne extending aggressively across the back and chest, profound male-pattern hair loss, and rapid-onset hirsutism. Biochemically, the introduction of exogenous androgens exerts negative feedback on the hypothalamic-pituitary-gonadal (HPG) axis, leading to the marked suppression of endogenous gonadotropins (luteinizing hormone

and follicle-stimulating hormone), low or undetectable native estradiol, and frequently suppressed levels of sex hormone-binding globulin (SHBG)(19,20).

Differentiating between a natural endocrine disorder and illicit substance use requires a rigorous, multi-layered clinical evaluation. A thorough history must explore training milestones, sudden changes in physical strength or muscle mass disproportionate to training history, and psychological shifts sometimes associated with AAS use (e.g., mood lability, aggression). In sports medicine practice, suspicious biochemical profiles—such as profoundly suppressed gonadotropins in the presence of marked clinical hyperandrogenism—should prompt immediate coordination with formal anti-doping protocols, carbon isotope ratio mass spectrometry (IRMS) testing, and biological passport monitoring. Recognizing the systemic sequelae of exogenous androgens—including hepatotoxicity, dyslipidemia, left ventricular hypertrophy, and irreversible vocal cord deepening or clitoromegaly—is paramount for sports physicians tasked with both protecting clean sport and managing the profound endocrine recovery required when an athlete ceases usage(15).

7. Laboratory Evaluation

A comprehensive laboratory evaluation is essential to accurately diagnose the etiology of hyperandrogenism in female athletes, differentiating between endogenous endocrine disorders, physiological adaptations, and exogenous substance use. Because clinical stigmata like acne or hirsutism reflect tissue-level sensitivity rather than systemic hormone concentrations alone, biochemical profiling must look beyond basic panels to assess the complete hormonal and metabolic milieu(table 2).

The baseline biochemical investigation should be timed strategically, ideally during the early follicular phase (days 2 to 5) of a spontaneous menstrual cycle if the athlete is eumenorrheic, though adjustments are necessary for those experiencing oligomenorrhea or amenorrhea. The core laboratory panel includes:

Androgen Profile: Total testosterone, free testosterone (preferably measured via equilibrium dialysis or calculated using total testosterone and Sex Hormone-Binding Globulin [SHBG]), androstenedione, and dehydroepiandrosterone sulfate (DHEA-S). Elevated DHEA-S strongly points toward an adrenal source of hyperandrogenism, whereas isolated or disproportionate elevations in testosterone and androstenedione typically suggest an ovarian origin(4,15).

Binding Proteins: Sex Hormone-Binding Globulin (SHBG). SHBG is a critical metric because conditions like insulin resistance suppress hepatic SHBG synthesis, elevating free (bioactive) testosterone even when total testosterone appears normal.

Gonadotropins and Reproductive Hormones: Luteinizing hormone (Luteinizing Hormone [LH]), follicle-stimulating hormone (Follicle-Stimulating Hormone [FSH]), and 17\beta-estradiol. An elevated LH-to-FSH ratio (often $> 2:1$ or $3:1$) is a hallmark of PCOS, whereas globally suppressed gonadotropins and estradiol point toward central suppression via Relative Energy Deficiency in Sport (RED-S) or the negative feedback of exogenous anabolic-androgenic steroids.

Metabolic Markers: Fasting blood glucose, fasting insulin, and a lipid profile (total cholesterol, HDL, LDL, triglycerides). These are vital for identifying underlying insulin resistance, metabolic syndrome, or metabolic-associated PCOS phenotypes, even in lean athletes.

Exclusionary and Adrenal Screenings: 17-hydroxyprogesterone (17-OHP) measured in the morning to rule out non-classical congenital adrenal hyperplasia (NCCAH) due to 21-hydroxylase deficiency, along with thyroid-stimulating hormone (TSH) and prolactin to exclude secondary causes of menstrual dysfunction and hyperandrogenemia.

In sports medicine, interpreting these results requires great contextual care. Transient fluctuations in total testosterone can occur following exhaustive endurance or high-intensity resistance training sessions, meaning blood draws should ideally be scheduled following a standardized rest period. Furthermore, when biochemical profiles reveal profound suppression of endogenous gonadotropins alongside clinical signs of hyperandrogenism in the absence of an identifiable energy deficit, clinicians must maintain a high index of suspicion for exogenous androgen exposure, prompting collaboration with specialized anti-doping protocols and confirmatory analytical testing(13,17).

Test Category	Specific Biomarkers	Clinical Purpose
Androgens	Total testosterone, free testosterone, androstenedione, DHEA-S	Differentiate adrenal versus ovarian sources of androgen excess
Binding Proteins	Sex Hormone-Binding Globulin (SHBG)	Assess bioavailable (free) testosterone and metabolic status
Reproductive Hormones	LH, FSH, 17β-estradiol	Evaluate HPG axis activity and distinguish PCOS from RED-S
Metabolic Markers	Fasting glucose, fasting insulin, lipid profile	Identify underlying insulin resistance, dyslipidemia, or metabolic syndrome
Adrenal Screening	Early-morning 17-hydroxyprogesterone (17-OHP)	Rule out non-classical congenital adrenal hyperplasia (NCCAH)

Table 2. Suggested Laboratory Assessment

8. Differential Diagnosis

Establishing an accurate diagnosis for a female athlete presenting with signs of hyperandrogenism requires a systematic and rigorous differential diagnostic process. Because clinical presentations frequently overlap—such as menstrual irregularities accompanied by cutaneous stigmata—clinicians must carefully delineate between intrinsic endocrine pathologies, exercise-induced physiological adaptations, and illicit pharmacological exposures. The primary entities that must be systematically differentiated include Polycystic Ovary Syndrome (PCOS), the most common internal endocrine disorder diagnosed via the Rotterdam criteria, and Relative Energy Deficiency in Sport (RED-S) alongside functional hypothalamic amenorrhea (FHA), which is characterized by central suppression of the hypothalamic-pituitary-gonadal axis due to chronic low energy availability. Other critical considerations

include Non-Classical Congenital Adrenal Hyperplasia (NCCAH) caused by partial 21-hydroxylase deficiency, rare but dangerous androgen-secreting ovarian or adrenal tumors characterized by rapid-onset severe virilization and profoundly elevated testosterone levels, exogenous androgen exposure via anabolic-androgenic steroids or SARMs presenting with suppressed gonadotropins, and systemic endocrine disorders such as Cushing's syndrome and hyperprolactinemia (table 3). A structured diagnostic algorithm utilizing baseline biochemical profiling, thorough dietary and training history reviews, and targeted imaging ensures that underlying pathologies are neither misattributed to routine athletic stress nor overlooked beneath the guise of high-performance training(13,14,17).

Condition	Key Features	Main Laboratory Findings
PCOS	Acne, hirsutism, irregular cycles, insulin resistance	High testosterone, high LH:FSH ratio, low SHBG
RED-S / FHA	Amenorrhea, low bone density, energy deficit	Low LH, FSH, and estradiol; normal/low androgens
Exogenous Doping	Rapid-onset severe virilization and voice deepening	Suppressed LH/FSH, low SHBG
NCCAH	Hirsutism and acne mimicking PCOS	Elevated morning 17-OHP
Androgen Tumors	Rapid, progressive, severe virilization	Total testosterone > 200 ng/dL
Cushing's / Prolactin	Central adiposity, striae, or galactorrhea	High cortisol or elevated prolactin

Table 3. Differential diagnosis of hyperandrogenic features in female athletes

9. Hyperandrogenism Versus RED-S

Differentiating between hyperandrogenism and Relative Energy Deficiency in Sport (RED-S) represents one of the most intellectually demanding diagnostic challenges in sports endocrinology(13,17). At first glance, these two conditions appear to occupy opposite ends of the endocrine spectrum: hyperandrogenism is defined by androgen excess and metabolic overactivation, whereas RED-S is characterized by energy deprivation, metabolic downregulation, and reproductive suppression. However, in the high-performance female athlete, their clinical presentations frequently intersect, creating diagnostic ambiguity. Both conditions can manifest with identical secondary consequences, most notably menstrual dysfunction (ranging from oligomenorrhea to secondary amenorrhea) and dermatological concerns, as chronic physiological stress can alter skin and hair homeostasis in both states(9,13).

The fundamental divergence lies in their pathophysiology and primary biochemical signatures. Hyperandrogenism—whether driven by Polycystic Ovary Syndrome (PCOS), ovarian or adrenal pathology, or exogenous substance use—stems from intrinsic hormonal excess or tissue hypersensitivity, often featuring elevated free testosterone, altered LH-to-FSH ratios, and sometimes underlying hyperinsulinemia. In stark contrast, RED-S is rooted in chronic low energy availability (LEA), where the body downregulates the hypothalamic-pituitary-gonadal (HPG) axis to conserve energy, resulting in suppressed gonadotropins (LH and FSH), low estradiol, and low-to-normal androgen levels. Yet, the diagnostic complexity escalates when considering that an athlete with an underlying, subclinical predisposition to PCOS may simultaneously experience severe RED-S due to weight-class pressures or intense caloric restriction. In these complex dual presentations, the energy deficit of RED-S can mask the classic biochemical markers of PCOS, only for hyperandrogenic symptoms to surge explosively once energy availability is restored and the HPG axis reactivates(1,15).

For the sports physician and clinician, distinguishing between the two requires looking beyond the menstrual cycle to evaluate metabolic, bone, and cardiovascular health parameters. While PCOS increases long-term cardiometabolic and endometrial risks, RED-S poses immediate threats to endothelial function, psychological stability, and bone mineral density, significantly elevating stress fracture risk. Misdiagnosing an athlete with RED-S as having simple PCOS—and prescribing hormonal contraceptives without addressing the underlying energy deficit—can disastrously accelerate bone loss and compromise long-term athletic health. Conversely, treating RED-S while ignoring an underlying hyperandrogenic pathology leaves metabolic and aesthetic symptoms unmanaged. Therefore, a comprehensive assessment integrating dietary

history, training loads, body composition tracking, and meticulous hormonal profiling is vital to untangle these overlapping syndromes and establish an appropriate, safe therapeutic intervention(9,13).

10. Cardiovascular and Metabolic Considerations

The intersection of hyperandrogenism and elite athletic training creates a complex cardiovascular and metabolic profile. While regular physical exercise is classically cardioprotective, the presence of pathological hyperandrogenism—particularly when driven by Polycystic Ovary Syndrome (PCOS), insulin resistance, or the clandestine use of performance-enhancing drugs—introduces significant long-term health risks that can counteract the protective adaptations of high-level training(12-14). From a metabolic perspective, hyperandrogenism is frequently intertwined with hyperinsulinemia and peripheral insulin resistance. Insulin acts synergistically with luteinizing hormone to stimulate ovarian androgen production while simultaneously suppressing hepatic sex hormone-binding globulin (SHBG) synthesis. In sedentary populations, this metabolic triad markedly increases the risk of impaired glucose tolerance, type 2 diabetes mellitus, and dyslipidemia (characterized by elevated triglycerides and decreased high-density lipoprotein cholesterol)(2,7,12). In female athletes, high daily energy expenditures and enhanced muscular glucose uptake can partially mask these metabolic disturbances, allowing athletes to maintain favorable body compositions while underlying insulin resistance silently compromises metabolic flexibility (1,7). Cardiovascular health is similarly modulated by the underlying etiology of hyperandrogenism. Endogenous hyperandrogenism, such as PCOS, is associated with early markers of cardiovascular risk, including endothelial dysfunction, increased arterial stiffness, and heightened systemic inflammation. Furthermore, when hyperandrogenism stems from the exogenous administration of anabolic-androgenic steroids (AAS) or selective androgen receptor modulators (SARMs), the cardiovascular toll is profound and rapid. Exogenous androgens frequently induce atherogenic lipid profiles (severe suppression of HDL and elevation of LDL)(13,17), systemic hypertension, direct myocardial toxicity, and left ventricular hypertrophy. Consequently, sports physicians must look beyond athletic performance metrics. Comprehensive cardiovascular and metabolic risk stratification—including regular fasting lipid panels, glycemic evaluations, and blood pressure monitoring—is vital, ensuring that the pursuit of athletic excellence does not compromise long-term metabolic health and cardiovascular longevity(10).

11. Management

The management of hyperandrogenism in female athletes requires a delicate, highly individualized multidisciplinary approach that balances symptom resolution and long-term metabolic health with athletic performance preservation, anti-doping compliance, and nutritional status. Therapeutic interventions span lifestyle optimization, targeted medical treatments, and sports-specific adaptations. Lifestyle and nutritional optimization serve as the cornerstone of management—particularly for PCOS-related phenotypes and metabolic dysregulation—focusing on correcting subtle energy deficits, optimizing macronutrient distribution, and managing carbohydrate intake to improve insulin sensitivity while preventing inadvertent overlap with Relative Energy Deficiency in Sport (RED-S) (13,14). When localized treatments fail to control severe dermatological manifestations, systemic therapies may be required, though choices like spironolactone or oral contraceptive pills (OCPs) must be carefully weighed against athletic needs, potential performance limitations regarding peak oxygen uptake and muscle hypertrophy, and World Anti-Doping Agency (WADA) regulations. For athletes exhibiting marked insulin resistance or metabolic-associated PCOS phenotypes, agents like metformin can improve metabolic flexibility and reduce circulating androgens without the performance-limiting side effects of hormonal contraceptives. In cases where hyperandrogenism stems from illicit or inadvertent performance-enhancing substance use, management shifts toward immediate cessation under medical supervision, comprehensive cardiovascular and hepatic monitoring, and structured endocrine rehabilitation to support the recovery of the suppressed hypothalamic-pituitary-gonadal axis. Ultimately, successful management bridges sports medicine, endocrinology, dermatology, and sports psychology, ensuring that the athlete's competitive career and long-term somatic health are both vigorously protected(3,4).

12. Practical Screening in Sports Medicine

Implementing a structured, practical screening protocol in sports medicine is essential for the early detection and management of hyperandrogenism in female athletes. Because clinical signs such as acne, hirsutism, or menstrual irregularities are frequently dismissed as normal side effects of rigorous training or overlooked due to the athlete's high physical fitness, sports physicians require a systematic approach during pre-participation physical evaluations (PPPE) and routine periodic health assessments(7,19). A comprehensive screening framework should incorporate several key pillars, beginning with targeted history-taking that explicitly screens for endocrine and reproductive health, including detailed inquiries about menstrual history,

cycle regularity, and recent changes in cycle frequency. This must be paired with a granular assessment of training volume and dietary habits to identify potential risks of Relative Energy Deficiency in Sport (RED-S)(3,6,11). During physical examination, clinicians should perform a focused assessment for cutaneous stigmata of hyperandrogenism, utilizing the modified Ferriman-Gallwey score to quantify hirsutism, evaluating for female pattern hair loss, and inspecting skin folds for acanthosis nigricans, which serves as a vital clinical marker for insulin resistance. When findings raise clinical suspicion, tiered biochemical and metabolic screening—encompassing total and free testosterone, SHBG, DHEA-S, a lipid profile, and fasting metabolic markers—should be initiated, ideally timed to the early follicular phase. Finally, given the high prevalence of supplement use in athletic populations, practitioners must rigorously audit all medications and formulations to ensure compliance with anti-doping regulations(4,14,17). By embedding these practical tools into standard clinical workflows, sports medicine practitioners can effectively pierce the masking effects of high-performance training, safeguard the athlete's long-term health, and ensure a safe, compliant competitive environment(2,18,21) (table 4).

Step	Action Item	Key Focus & Objectives
1. Targeted Screening	History & Questionnaires	Screen for menstrual irregularity, training volume, energy intake, and dermatological symptoms.
2. Physical Examination	Stigmata Assessment	Evaluate for hirsutism (mFG score), acne, pattern hair loss, and acanthosis nigricans.
3. Biochemical Workup	Tiered Laboratory Testing	Assess androgens (total/free testosterone, DHEA-S), SHBG, metabolic markers, and 17-OHP.
4. Anti-Doping Audit	Supplement & Drug Review	Verify all nutritional supplements,

		formulations, and medications against WADA regulations.
5. Multidisciplinary Care	Collaborative Management	Coordinate care across sports medicine, endocrinology, nutrition, and psychology.

Table 4. Proposed Clinical Checklist for Sports Medicine Practice

13. Adolescent and Young Female Athletes

The presentation and management of hyperandrogenism in adolescent and young female athletes introduce a distinct layer of clinical complexity, as these cases coincide with the critical physiological windows of puberty, skeletal maturation, and peak bone mass acquisition(15,20). During adolescence, the hypothalamic-pituitary-gonadal (HPG) axis is naturally maturing, and physiological irregularities in menstrual cycles or mild dermatological manifestations such as adolescent acne are common. Consequently, distinguishing between normal pubertal transition variants and early-stage endocrine pathology, such as Polycystic Ovary Syndrome (PCOS), presents a significant diagnostic hurdle for sports medicine clinicians and pediatric endocrinologists alike(20,21). In young athletes, the compounding pressures of competitive sports—ranging from aesthetic ideals in gymnastics and figure skating to weight-class requirements in combat sports—frequently interact with emerging endocrine dysfunctions. When an adolescent athlete experiences secondary amenorrhea or severe cutaneous hyperandrogenism, the differential diagnosis must carefully weigh intrinsic endocrine disorders against Relative Energy Deficiency in Sport (RED-S)(20-23). Misdiagnosing an energy availability deficit as simple PCOS in a developing teenager can lead to inappropriate prescription of hormonal contraceptives without correcting the underlying caloric shortfall, jeopardizing her final adult height, endocrine development, and, most critically, lifelong skeletal health. Peak bone mass is largely established during the second decade of life; therefore, prolonged hypoestrogenism driven by either untreated RED-S or severe endocrine disruption during these formative years can cause irreversible deficits in bone mineral density, elevating the long-term risk of stress fractures and early-onset osteoporosis(5,23). Furthermore, the psychological vulnerability of adolescent athletes navigating body image changes, peer comparison, and performance expectations requires a deeply empathetic and holistic clinical

approach(24). Management strategies must prioritize nutritional education, gentle lifestyle and training modifications, and family involvement over aggressive pharmacological suppression whenever possible(25,26). By instituting age-appropriate screening protocols and maintaining a high index of suspicion for both endocrine pathologies and energy deficits, clinicians can protect the physical and emotional trajectory of young female competitors during their most formative developmental years(27,28).

14. Clinical Implications

The clinical implications (figure 3) of hyperandrogenism in female athletes extend far beyond transient cosmetic concerns or temporary athletic setbacks, representing a complex crossroads of endocrinology, sports medicine, cardiology, and psychology (figure 3)(10,21,28,30). Recognizing and properly diagnosing these conditions requires clinicians to synthesize a broad spectrum of physiological systems, as the downstream effects of untreated or mismanaged endocrine imbalances can permanently alter an athlete's health span and career trajectory(30). The primary clinical implications span several critical domains:

Long-Term Metabolic and Cardiovascular Disease Risk: Underlying drivers of hyperandrogenism, such as Polycystic Ovary Syndrome (PCOS) and chronic hyperinsulinemia, introduce substantial long-term risks for metabolic syndrome, impaired glucose tolerance, type 2 diabetes mellitus, and premature cardiovascular disease. Even when high-level training masks these risks through enhanced glucose disposal and favorable body compositions, the underlying vascular and metabolic inflexibility persists. Furthermore, illicit or inadvertent exposure to exogenous performance-enhancing androgens drastically amplifies cardiovascular danger through severe atherogenic lipid shifts, myocardial stress, and left ventricular hypertrophy.

Skeletal Integrity and Endocrine Health: The delicate interplay between hyperandrogenism, menstrual dysfunction, and Relative Energy Deficiency in Sport (RED-S) carries profound implications for bone mineral density. Prolonged hypoestrogenism—whether driven by central energy deprivation or inappropriate pharmacological suppression—during critical developmental or active training years can cause irreversible bone mass deficits, predisposing athletes to recurrent stress fractures and early-onset osteopenia or osteoporosis.

Psychosocial and Career Longevity: The psychological toll of navigating visible stigmata like hirsutism, cystic acne, and hair loss—coupled with the stress of regulatory compliance, anti-doping scrutiny, and body image pressures—can severely compromise an athlete’s mental health, leading to anxiety, depression, burnout, or premature retirement from competitive sport.

Diagnostic and Therapeutic Precision: Perhaps the most critical clinical implication is the absolute necessity for diagnostic accuracy. Misattributing an energy deficit to simple PCOS or prescribing hormonal contraceptives without resolving underlying nutritional and training imbalances can lead to disastrous long-term health consequences.

Ultimately, navigating these clinical implications demands a holistic, multidisciplinary approach where sports physicians, endocrinologists, dietitians, and sports psychologists collaborate to safeguard both the immediate competitive safety and the lifelong somatic and psychological well-being of the female athlete.

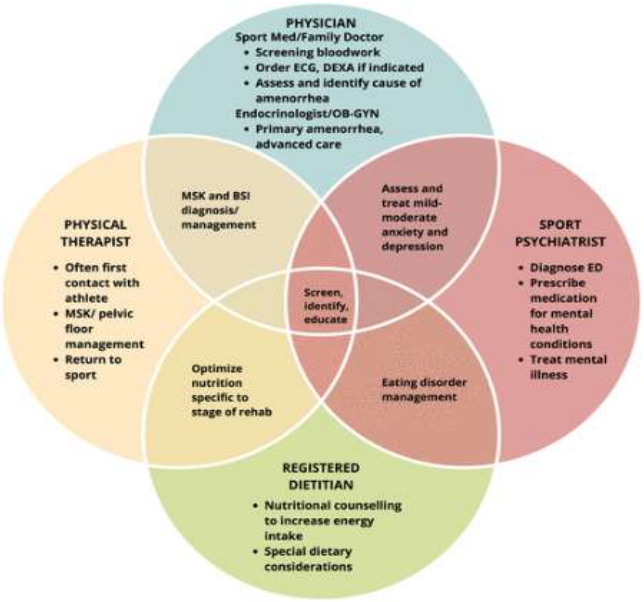

Figure 3. Multidisciplinary model of care for the hyperandrogenic female athlete.

15..Conclusion

Hyperandrogenism in female athletes represents a uniquely intricate convergence of endocrinology, sports medicine, dermatology, and metabolic science. Because the physical stigmata of androgen excess—ranging from persistent acne and hirsutism to female pattern hair loss and acanthosis nigricans—often overlap with or are masked by the physiological

adaptations of rigorous training, identifying the true underlying etiology demands exceptional clinical vigilance.

Throughout this diagnostic and therapeutic journey, distinguishing between intrinsic endocrine pathologies like Polycystic Ovary Syndrome (PCOS), exercise-induced energy deficits such as Relative Energy Deficiency in Sport (RED-S), and illicit exogenous androgen exposure is paramount. Missteps in this differential diagnosis—such as prescribing hormonal contraceptives for perceived PCOS without addressing a foundational caloric shortfall—can severely compromise an athlete's skeletal integrity, metabolic health, and long-term career longevity.

Ultimately, managing hyperandrogenism requires moving far beyond superficial symptom resolution. It necessitates a holistic, multidisciplinary framework where sports physicians, endocrinologists, sports dietitians, and mental health professionals collaborate seamlessly. By prioritizing targeted screening, individualized metabolic and cardiovascular risk stratification, and compassionate psychosocial support, clinicians can successfully safeguard the immediate competitive safety, regulatory compliance, and lifelong somatic and emotional well-being of the female athlete.

Authors' Contributions:

Conceptualization: **D.S.**

Formal analysis: **D.S J.P. A.G.S. N.G. M.F. A.N. A.B. R.B. A.S. L.K.**

Investigation: **D.S J.P. A.G.S. N.G**

Writing – original draft: **D.S J.P. A.G.S. N.G. M.F**

Writing – review and editing: **D.S J.P. A.G.S. N.G. M.F.**

Visualization: **D.S J.P. A.G.S. N.G. M.F. A.N. A.B. R.B. A.S. L.K.**

All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Avability Statement: Not applicable

Conflicts of Interest: The authors declare no conflict of interest.

AI Declaration: In preparing this work, the authors used Google Gemini to enhance linguistic clarity, improve readability, and assist with text formatting and basic data organization. Following the use of this service, the authors reviewed and edited the text and accept full responsibility for the substantive content of the publication

Ethical review and approval were not required for this study because it is a narrative review based exclusively on previously published literature and did not involve human participants, animals, or the collection of primary data.

References

1. Cruz-Jentoft AJ, Bahat G, Bauer J, Boirie Y, Bruyère O, Cederholm T, et al. Sarcopenia: revised European consensus on definition and diagnosis. Age Ageing. 2019;48(1):16-31. https://doi.org/10.1093/ageing/afy169
2. Hirschberg AL. Female hyperandrogenism and elite sport. J Clin Endocrinol Metab. 2020;105(3):dgaa068. https://doi.org/10.1210/clinem/dgaa068
3. Mountjoy M, Sundgot-Borgen J, Burke L, Ackerman KE, Blauwet C, Constantini N, et al. IOC consensus statement on relative energy deficiency in sport (RED-S): 2018 update. Br J Sports Med. 2018;52(11):687-697. https://doi.org/10.1136/bjsports-2018-099196
4. Teede HJ, Misso ML, Costello MF, Dokras A, Laven J, Moran L, et al. International evidence-based guideline for the assessment and management of polycystic ovary syndrome 2018. Fertil Steril. 2018;110(3):364-380. https://doi.org/10.1016/j.fertnstert.2018.06.002
5. Legro RS, Arslanian SA, Ehrmann DA, Hoeger KM, Murad MH, Pasquali R, et al. Diagnosis and treatment of polycystic ovary syndrome: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. 2013;98(12):4565-4592. https://doi.org/10.1210/jc.2013-2350
6. Ackerman KE, Holtzman B, Cooper AB, Flynn EF, Bruinvels G, Tenforde AS, et al. Low energy availability surrogates correlate with health and performance consequences of Relative Energy Deficiency in Sport. Sports Med. 2019;49(1):1-12. https://doi.org/10.1007/s40279-018-0995-8
7. De Souza MJ, Nattiv A, Joy E, Misso M, Williams NI, Mallinson RJ, et al. 2014 Female Athlete Triad Coalition consensus statement on treatment and return to play of the female athlete triad. Br J Sports Med. 2014;48(4):289. https://doi.org/10.1136/bjsports-2013-093218
8. Hirschberg AL. Female Hyperandrogenism in Elite Sports and the Athletic Triad. Semin Reprod Med. 2022;40(1-2):32-41. https://doi.org/10.1055/s-0041-1736337
9. Bermon S, Garnier PY, Hirschberg AL, Robinson N, Giraud S, Piccoli LJ, et al. Serum androgen levels and their relation to performance in elite female athletes: a cross-sectional study. Br J Sports Med. 2014;48(1):130-135. https://doi.org/10.1136/bjsports-2013-092843
10. Speiser PW, Arlt W, Auchus RJ, Baskin LS, Conway GS, Merke DP, et al. Congenital adrenal hyperplasia due to steroid 21-hydroxylase deficiency: an Endocrine Society

clinical practice guideline. J Clin Endocrinol Metab. 2018;103(11):4043-4088. https://doi.org/10.1210/jc.2018-01865

11. Auchus RJ, Arlt W. Approach to the patient: the congenital adrenal hyperplasias due to 21-hydroxylase deficiency. J Clin Endocrinol Metab. 2013;98(3):901-907. https://doi.org/10.1210/jc.2012-2661
12. Futterman R, Handelsman DJ. Androgens and athletic performance in women. Curr Opin Endocrinol Diabetes Obes. 2016;23(3):236-241. https://doi.org/10.1097/MED.0000000000000250
13. Handelsman DJ, Hirschberg AL, Bermon S. Circulating testosterone as the hormonal basis of sex differences in athletic performance. Endocr Rev. 2018;39(5):803-829. https://doi.org/10.1210/er.2018-00020
14. Gleeson M. Immune function in sport and exercise. J Appl Physiol (1985). 2007;103(2):693-699. https://doi.org/10.1152/japplphysiol.00008.2007
15. Hackney AC. Stress and the neuroendocrine system: the relationship with physical exercise. Expert Rev Endocrinol Metab. 2006;1(6):783-792. https://doi.org/10.1586/17446651.1.6.783
16. Melin AK, Tornberg ÅB, Skolnick S, Faber J, Sjödin A. The LEAF questionnaire: a screening tool for the identification of female athletes at risk for the female athlete triad. Br J Sports Med. 2014;48(7):540-545. https://doi.org/10.1136/bjsports-2013-093240
17. Loucks AB, Kiens B, Wright HH. Energy availability in athletes. J Sports Sci. 2011;29(sup1):S7-S15. https://doi.org/10.1080/02640414.2011.588958
18. Hagmar M, Berglund L, Brismar K, Hirschberg AL. Body composition and endocrine profile in male and female distance runners. Scand J Med Sci Sports. 2013;23(4):e197-e204. https://doi.org/10.1111/sms.12047
19. Nattiv A, Loucks AB, Manore MM, Sanborn CF, Sundgot-Borgen J, Warren MP. American College of Sports Medicine position stand. The female athlete triad. Med Sci Sports Exerc. 2007;39(10):1867-1882. https://doi.org/10.1249/mss.0b013e318149f111
20. Gordon CM, Ackerman KE, Berga SL, Kaplan JR, Mastorakos G, Misra M, et al. Functional hypothalamic amenorrhea: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. 2017;102(5):1413-1439. https://doi.org/10.1210/jc.2017-00131
21. Azziz R, Carmina E, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Futterweit W, et al. The Androgen Excess and PCOS Society criteria for the polycystic

ovary syndrome: the complete task force report. Fertil Steril. 2009;91(2):456-488. https://doi.org/10.1016/j.fertnstert.2008.06.035

22. Carmina E, Stanczyk FZ, Lobo RA. The diagnosis of polycystic ovary syndrome: is it time to change the criteria? Nat Rev Endocrinol. 2010;6(10):580-584. https://doi.org/10.1038/nrendo.2010.127
23. Goodarzi MO, Dumesic DA, Chazenbalk G, Azziz R. Polycystic ovary syndrome: aetiology, pathogenesis and diagnosis. Nat Rev Endocrinol. 2011;7(4):219-231. https://doi.org/10.1038/nrendo.2010.217
24. Escobar-Morreale HF. Polycystic ovary syndrome: definition, aetiology, diagnosis and treatment. Nat Rev Endocrinol. 2018;14(5):270-284. https://doi.org/10.1038/nrendo.2018.24
25. Sattar N, Preiss D. The metabolic syndrome: practical concepts and scientific challenges. BMJ. 2012;344:e469. https://doi.org/10.1136/bmj.e469
26. Wild RA, Carmina E, Diamanti-Kandarakis E, Dokras A, Escobar-Morreale HF, Futterweit W, et al. Assessment of cardiovascular risk and prevention of cardiovascular disease in women with the polycystic ovary syndrome: a consensus statement by the Androgen Excess and PCOS Society and the American Society for Reproductive Medicine. Fertil Steril. 2010;94(6):1986-1993. https://doi.org/10.1016/j.fertnstert.2010.04.053
27. World Anti-Doping Agency (WADA). The World Anti-Doping Code: International Standard Prohibited List 2026. Montreal: WADA; 2026.
28. Mottram DR, Chester N, editors. Drugs in Sport. 7th ed. London: Routledge; 2022.
29. Sonksen PH, Holt RI. Sarcopenia: revised European consensus on definition and diagnosis. Br J Sports Med. 2015;49(1):12-13. https://doi.org/10.1136/bjsports-2014-094459
30. Thompson B, Mauger AR, Warren A. Mental health and body image in elite female athletes: psychosocial considerations. Sports Med Open. 2021;7(1):45. https://doi.org/10.1186/s40798-021-00334-y