Article published online: 2021-10-11

Female Hyperandrogenism in Elite Sports and the Athletic Triad

Angelica Lindén Hirschberg, MD, PhD[1,2]

[1] Department of Women's and Children's Health, Karolinska Institutet, Stockholm, Sweden
[2] Department of Gynecology and Reproductive Medicine, Karolinska University Hospital, Stockholm, Sweden

Address for correspondence Angelica Lindén Hirschberg, MD, PhD, Department of Women's and Children's Health, Karolinska Institutet, SE 171 76 Stockholm, Sweden (e-mail: angelica.linden-hirschberg@sll.se).

Semin Reprod Med 2022;40:32–41

Abstract

Essential hyperandrogenism seems to be overrepresented in female elite athletes. This applies to mild forms such as polycystic ovary syndrome, as well as rare differences/disorders of sex development (DSD). The reason is likely a selection bias since there is increasing evidence that androgens are beneficial for athletic performance by potent anabolic effects on muscle mass and bone mass, and stimulation of erythropoiesis. XY DSD may cause a greatly increased production of testosterone in the male range, that is, 10 to 20 times higher than the normal female range. The established regulations concerning the eligibility of female athletes with severe hyperandrogenism to compete in the female classification remain controversial. The most common cause of menstrual disorders in female athletes, however, is probably an acquired functional hypothalamic disturbance due to energy deficiency in relation to energy expenditure, which could lead to low bone mineral density and increased risk of injury. This condition is particularly common in endurance and esthetic sports, where a lean body composition is considered an advantage for physical performance. It is important to carefully evaluate endocrine disturbances and menstrual disorders in athletes since the management should be specific according to the underlying cause.

Keywords
- differences of sex development
- female athlete triad
- functional hypothalamic amenorrhea
- hyperandrogenism
- polycystic ovary syndrome

Endocrine disturbances are common among female elite athletes often resulting in menstrual disorders. These hormonal disturbances can either be a consequence of intense physical exercise in combination with energy deficiency leading to functional hypothalamic amenorrhea (FHA) or the result of selection of individuals with certain endocrine characteristics, which can be beneficial for physical performance. The latter refers to essential hyperandrogenism, such as the polycystic ovary syndrome (PCOS). The management of menstrual disorders in these women is dependent on the underlying endocrine mechanism. This review is an update of the literature on hyperandrogenic disorders among female athletes, as well as energy deficiency–related amenorrhea and its long-term medical consequences.

Hyperandrogenism among Female Athletes

Essential hyperandrogenism in women can vary from mild to severe forms depending on the underlying cause. PCOS is the most common cause of mild hyperandrogenism, affecting approximately 10% of the female population,[1] whereas differences/disorders of sex development (DSD) are rare conditions that may cause severe hyperandrogenism in women. Both mild and severe forms of hyperandrogenism seem to be overrepresented in female athletes.

Polycystic Ovary Syndrome

PCOS is considered the most frequent endocrine disorder in women of fertile age and is characterized by increased

Issue Theme Adolescent Sexual and Reproductive Health: Two-Volume Series (Part 1); Guest Editors, Tania S. Burgert, MD and Kathleen M. Hoeger, MD, MPH

Thieme Medical Publishers, Inc., 333 Seventh Avenue, 18th Floor, New York, NY 10001, USA

DOI https://doi.org/10.1055/s-0041-1736337.
ISSN 1526-8004.

ovarian production of androgens, anovulation, and ultrasound findings of polycystic ovaries.[1] Circulating levels of testosterone in PCOS are usually within the upper normal female range, whereas sex hormone–binding globulin (SHBG) is low resulting in increased levels of free androgen index (FAI).[2,3] The clinical symptoms of PCOS, including oligomenorrhea or amenorrhea, hirsutism, and acne, typically start to develop at puberty. Over time, the development of symptoms can be aggravated by increasing body weight, as insulin resistance and abdominal obesity are common features in women with PCOS and are known to exacerbate all symptoms linked to the syndrome.[1] In the long run, women with PCOS have an increased risk of type 2 diabetes and the metabolic syndrome.[1] The etiology of PCOS is still largely unknown, but there is strong evidence for a genetic predisposition, although environmental factors also play a part.

There is support that PCOS is common in female athletes.[3–13] In adolescent athletes, the occurrence of PCOS was higher among elite swimmers than controls,[10] and in another study, one-third of 49 asymptomatic elite adolescent female athletes had elevated FAI.[9] Testosterone levels were also increased in young ballet dancers with menstrual disorders.[12] Moreover, Hagmar et al reported that PCOS was the most frequent cause of menstrual disorders among 90 female Olympic athletes (►**Fig. 1**).[7] Another study showed that 17% of 100 young exercising women with oligo/amenorrhea but no other clinical symptoms had biochemical hyperandrogenism.[13] Thus, mild forms of hyperandrogenism such as PCOS can occur in female athletes without overt clinical signs of hyperandrogenism. Hypothetically, athletes with PCOS have lower BMI than women with PCOS in general and regular physical exercise might counteract clinical symptoms of hyperandrogenism by increasing insulin sensitivity and SHBG, and thus reducing FAI.[14,15]

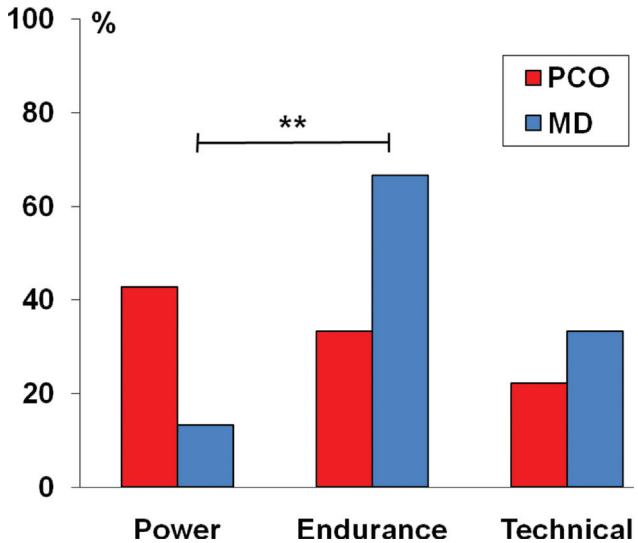

Fig. 1 The proportion of 90 Olympic athletes not using hormonal contraceptives who had menstrual dysfunction (MD) and polycystic ovaries (PCO). Values are presented as percentage of all athletes in each sport group. A significantly larger proportion of the endurance athletes than of those participating in power disciplines exhibited menstrual disturbances. Data from Hagmar et al.[7]

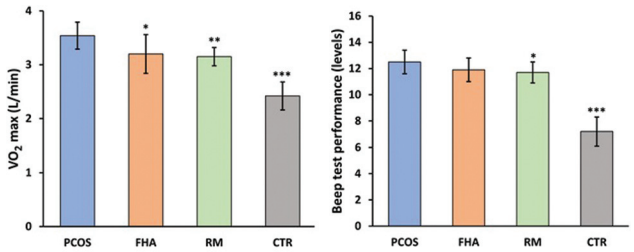

Fig. 2 Maximal oxygen uptake and endurance performance levels (the Beep test) in groups of 39 athletes with polycystic ovary syndrome (PCOS), functional hypothalamic amenorrhea (FHA), and regular menstruation (RM) and in a sedentary control group (CTR). Asterisk (*) indicates significant differences compared with the PCOS group. Data from Rickenlund et al.[5]

Athletes with PCOS have greater muscle mass and bone mass than other athletes,[5,11] and this condition appears to provide good protection from bone loss despite relative estrogen deficiency and oligomenorrhea/amenorrhea.[5,11] Furthermore, women athletes and nonathletes with PCOS have greater muscle strength related to levels of bioavailable testosterone and irrespective of body composition in comparison with control women.[5,16–19] Athletes with PCOS also exhibit higher aerobic performance levels than non-PCOS athletes (►**Fig. 2**).[5] It was therefore suggested that the higher levels of testosterone may be an advantage for physical performance and that PCOS could play a role in the recruitment of women to competitive sports.[5] Although some studies, but not all,[20–23] have shown that acute exercise may induce an increase in testosterone in women,[24–27] there is no support that long-term strenuous physical exercise induces PCOS. It is therefore likely that women with PCOS are selected to sports due to their performance.

Differences/Disorders of Sex Development

A few women have severe forms of hyperandrogenism due to DSD, in which the development of chromosomal, gonadal, and anatomic sex is atypical. Such a condition is usually diagnosed at birth because of ambiguous genitalia or at puberty due to the absence of menarche. There are several forms of DSD but in those involving a male karyotype and undescended but functioning testes (XY DSD), the individual can produce testosterone within the male range, that is, 10 to 20 times higher than the normal female range (0.1–1.8 vs. 8–29 nmol/L).[2,3] If the individual has normal androgen receptor sensitivity, her body will undergo virilization when the testes start to produce large amounts of androgens at puberty, resulting in muscle growth, increase in body hair, hair loss of male type, deepening of the voice, breast atrophy, and the enlargement of the clitoris. Examples of XY DSD are 5α reductase deficiency, implying failure to convert testosterone into dihydrotestosterone (DHT), and androgen insensitivity syndrome (AIS) caused by a mutation in the androgen receptor gene, and leading to various degrees of undervirilization (►**Table 1**).[2,3,28] Some of these disorders clearly increase the risk of germ cell cancer if the gonads are intra-abdominal, such as partial androgen insensitivity

Table 1 Examples of 46,XY differences/disorders of sex development

Condition	Incidence	Comment	Advantage in sports?
5α-Reductase deficiency type 2	Extremely rare with geographical regions of higher incidence	Individuals may be assigned female sex at birth. Virilization proceeds at puberty	Yes
Complete androgen insensitivity	1/50,000	Female phenotype without symptoms of hyperandrogenism. No effect of high testosterone levels	No
Partial androgen insensitivity	1/130,000	Ambiguous genitalia at birth. From puberty, high testosterone, but with varying effect of virilization depending on androgen receptor sensitivity	Possibly

(PAIS) increasing the risk as much as 30 to 40%.[3,28] In these cases, gonadectomy is recommended. Furthermore, XY DSD may result in changed gender identity, as in 5α reductase deficiency where most individuals are raised as girls, but more than half choose to become men after puberty.[3,28]

The incidence of XY DSD in the general population has been reported to be 1 in 20,000,[28] whereas the prevalence of XY DSD among elite female athletes is estimated to be increased approximately 140 times.[29] In the sports community and in the society as a whole, there is a great controversy, whether it is fair to let female athletes with DSD and testosterone in the male range to compete against women with normal levels of testosterone. This led the International Association of Athletics Federation (IAAF), now World Athletics, to establish regulations for the first time in 2011, concerning the eligibility of female athletes with hyperandrogenism to participate in the female classification. The rules were endorsed by the International Olympic Committee (IOC) prior to the Olympic Games in London in 2012. However, the regulations were criticized,[30–34] and in 2019 an update was made.[35,36] According to the current regulations, an athlete with XY DSD can compete in the female category if she has testosterone levels below 5 nmol/L. A potent suppression of testosterone can be obtained by combined hormonal contraceptives or similar treatment. An exception to the rules is complete AIS (CAIS), since in this case the individual cannot respond to androgens, although she has testosterone in the male range. In the latest International Federation of Sports Medicine (FIMS) 2021 Consensus Statement, it was concluded that although there is a lack of sports performance data in transwomen and DSD women, the current rules are based on the best available scientific evidence.[37]

The Role of Androgens for Physical Performance

Androgens are considered beneficial for athletic performance by potent anabolic effects on muscle mass and bone tissue.[2,38,39] Testosterone also stimulates erythropoiesis and promotes competitive behavior.[2,40] Although most studies have been performed in men, there is emerging evidence that androgens also are of importance for physical performance in women.

Endogenous Androgens and Physical Performance in Women

Few studies have investigated the influence of endogenous androgen levels on physical performance in exercising women, which may be explained by the challenges of hormonal variations during the menstrual cycle and usage of hormonal contraception. One earlier study reported a positive correlation between serum testosterone levels within the normal range and explosive performance in 22 female elite athletes.[41] However, in a study of four female Olympic weightlifters, there was no correlation between salivary testosterone and performance tests.[42] On the other hand, Bermon and Garnier showed in a large study involving more than 1,000 female elite athletes in track and field that athletes with the highest tertile of free testosterone performed significantly better than those in the lowest tertile.[43] Furthermore, we reported that female Olympic athletes had higher levels of endogenous precursor androgens including dehydroepiandrosterone (DHEA) and DHT than controls, and androgen levels correlated positively to lean mass and physical performance in the athletes (►**Fig. 3**).[44] These studies support that endogenous androgen levels in the normal range are related to physical performance in female athletes.

Less is known about the effect of pathologically increased endogenous testosterone on physical performance in women. As mentioned earlier, there are data supporting that athletes with PCOS perform better in strength and aerobic performance tests in relation to testosterone levels than non-PCOS athletes.[4,5,16–19] However, most women with PCOS have total testosterone levels within the normal upper range, although levels of free or bioavailable testosterone are increased. By obvious reasons, there are no studies on groups of female athletes with rare disorders of XY DSD and endogenous testosterone in the male range. However, in a case report of XY DSD, a reduction of testosterone by treatment resulted in 5 to 7% impairment in physical performance.[45]

Exogenous Androgens and Physical Performance in Women

It is well-known that the administration of high doses of exogenous androgens has been used for a long time to

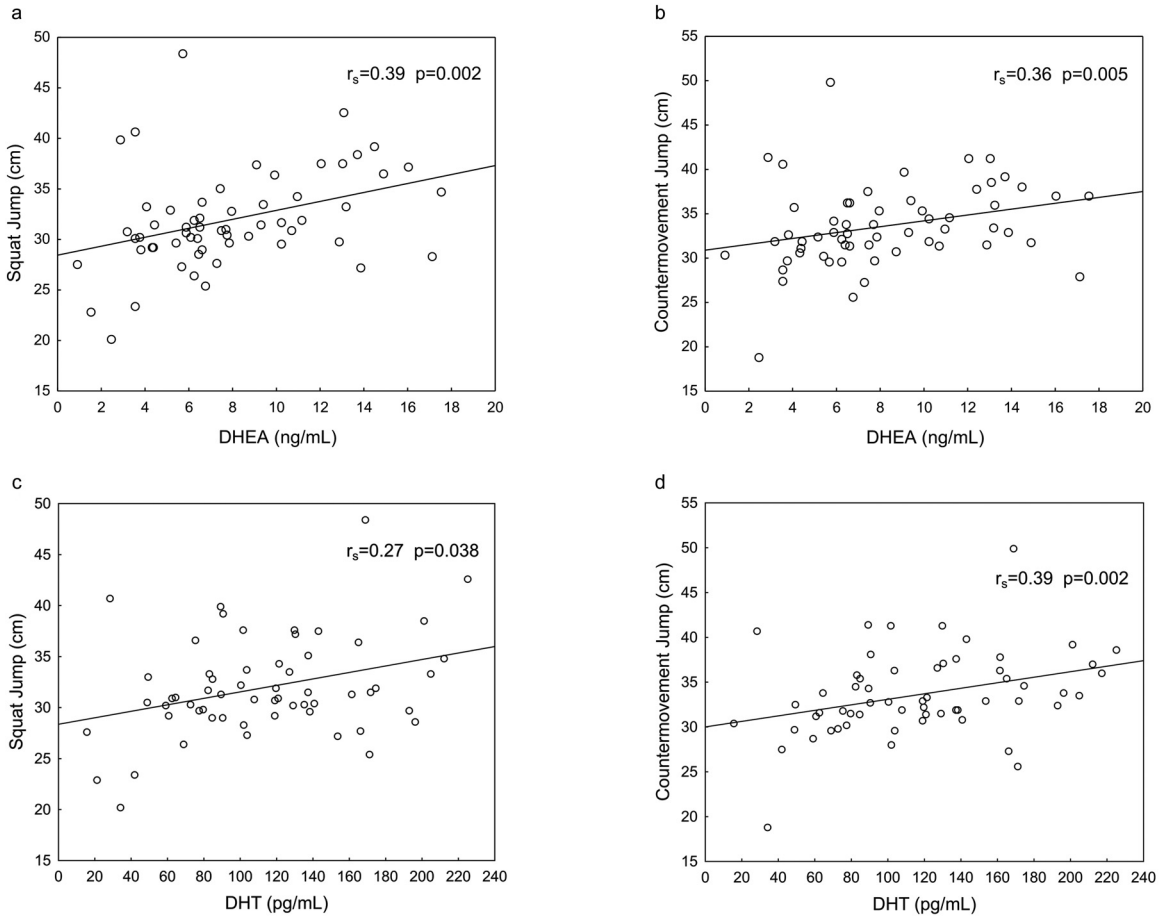

Fig. 3 (**a–d**) Significant positive correlations between the serum androgen precursor dehydroepiandrosterone (DHEA), dihydrotestosterone (DHT), and physical performance tests (squat jump and countermovement jump) in 106 female Olympic athletes. Used with permission from Eklund et al.[44]

increase power and athletic capacity in both men and women in sports, and these substances are therefore classified as doping agents and banned in sports.[46] However, there are few studies on the effects of exogenous testosterone on physical performance in women, probably due to the ethical concerns and risks of adverse effects on fertility and irreversible masculinizing effects.

One study in postmenopausal women showed a dose–response effect on muscle mass and muscle strength by increasing doses of testosterone to a maximal mean level of 7.3 nmol/L.[47] Recently, we published for the first time the results of a double-bind, randomized, placebo-controlled trial on the effect of testosterone supplementation (10 mg daily) on physical performance in young healthy, exercising women.[48] We found that testosterone, increased to a mean level of 4.3 nmol/L, enhanced muscle mass and aerobic performance by 8.5% (►**Fig. 4**). Furthermore, it has been reported that 1 year of gender-affirming treatment in transmen (biological female) increased muscle mass by 15% and muscle strength by 12 to 25%, whereas transwomen (biological male) decreased muscle volume by 5% but maintained muscle strength.[49]

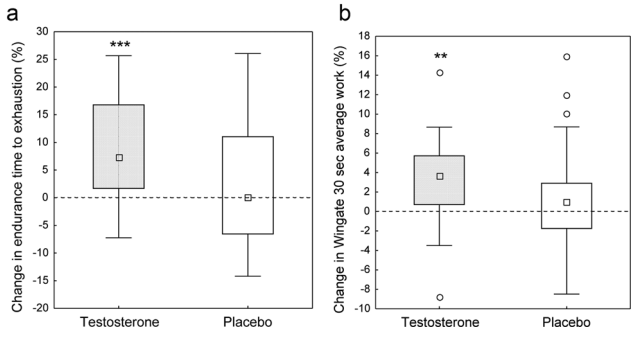

Fig. 4 Percentage change in aerobic performance measured as endurance time to exhaustion (**a**) and anaerobic performance measured as Wingate 30-second average work (**b**), before and after 10 weeks of treatment with testosterone or placebo in young, physically active, healthy women ($n = 24$ in each group). Values are median and total range. Endurance time to exhaustion increased significantly within the testosterone group and compared with the placebo group, whereas the change in Wingate power was significant only within the testosterone group. Used with permission from reference Hirschberg et al.[48]

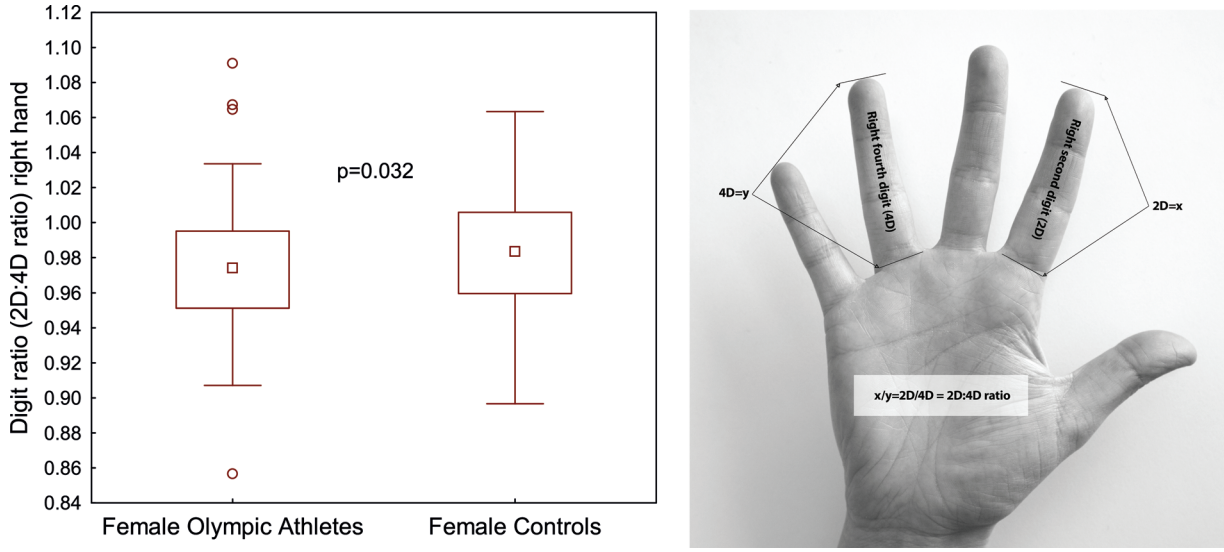

Fig. 5 Significant difference in digit ratio (2D:4D ratio) right hand expressed in millimeters (mm) between female Olympic athletes ($n = 104$) and controls ($n = 117$). Used with permission from Eklund et al[55]

Marker of Prenatal Androgen Exposure

The second to fourth digit ratio (2D:4D) is suggested to be a negative correlate of prenatal androgen exposure, showing an association between a low 2D:4D ratio and high environmental levels of androgens during fetal life.[50] Although this concept is controversial and has been debated,[51] the 2D:4D has been related to athletic ability.[52] Thus, previous studies mainly in male athletes have shown a lower digit ratio than in nonathletes, indicating higher exposure to androgens during fetal life in the athletes.[52–54] In female Olympic athletes, we recently reported lower digit ratio than in controls (►**Fig. 5**) and an association between a lower 2D:4D ratio and better physical performance levels.[55] Furthermore, a lower 2D:4D ratio correlated to higher urinary androgen metabolites but not to serum androgen levels. It was suggested that genetic variation of the androgen metabolism and thereby androgen activity could influence the development of the 2D:4D ratio and the predisposition for physical performance.

Functional Hypothalamic Amenorrhea

Relative Energy Deficiency

Although hyperandrogenic disorders like PCOS seem to be frequent among female athletes, the most common cause of amenorrhea in exercising women is probably an acquired functional hypothalamic disturbance due to energy deficiency in relation to energy expenditure. The intense physical training necessary for top athletic achievement requires a high energy output that many athletes do not match with a corresponding caloric intake. Recent advances have therefore led to the understanding that many sportswomen develop relative energy deficiency.[3,56] However, it is also common with a conscious pursuit of leanness, that is, low amount of body fat in relation to muscle mass, as this is an important factor for sports performance in several disciplines including endurance sports, weight class sports, and esthetic sports.[3,56,57] Furthermore, female athletes are particularly at risk for developing eating disorders known to be related to menstrual disturbances and amenorrhea.[58]

Endocrine Disturbances

FHA in athletes, also called athletic amenorrhea, is attributed to inhibition of the hypothalamic–pituitary–gonadal (HPG) axis.[3,59] This leads to a disruption of the pulsatile release of gonadotropin-releasing hormone (GnRH), which in turn causes a reduced secretion of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) from the pituitary resulting in attenuated ovarian production of estradiol, progesterone, and testosterone and subsequent anovulation and amenorrhea (►**Fig. 6**).[3,6,59]

Several mechanisms are involved in such inhibition of the HPG axis, including an exercise-induced activation of the hypothalamic–pituitary–adrenal axis and a consequent increase in hypothalamic corticotropin-releasing hormone (CRH) and cortisol from the adrenal glands (►**Fig. 6**).[6,59,60] Cortisol increases in situations of acute physical and psychological distress to mobilize glucose for energy production. However, chronic elevation of cortisol levels at rest in female athletes indicates catabolic metabolism and adaptation to relative energy deficiency. CRH and cortisol together with the endorphins, also released in response to physical activity, inhibit GnRH secretion in the hypothalamus.[59,60]

Insulin-like growth factor I (IGF-I), which is secreted from the liver, is an anabolic hormone of importance for muscle and skeletal growth and a peripheral marker of nutritional status. Secondary to chronic energy deficiency, circulating levels of insulin and IGF-I are reduced and levels of growth hormone and IGF-binding protein-1 are increased (►**Fig. 6**).[59–62] Because IGF-I also stimulates the release of both GnRH and LH, a decline in IGF-I activity may, at least in part, explain the reduction in LH secretion.

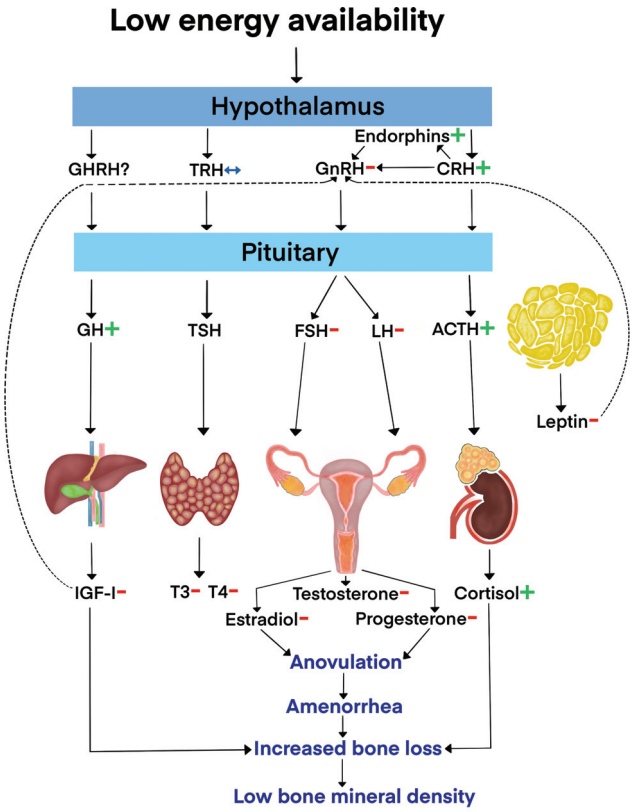

Fig. 6 A summary of endocrine disturbances associated with functional hypothalamic amenorrhea related to low energy availability. + indicates increased levels, − indicates reduced levels. ACTH, adrenocorticotropic hormone; CRH, corticotrophin-releasing hormone; FSH, follicle-stimulating hormone; GH, growth hormone; GHRH, growth hormone–releasing hormone; GnRH, gonadotropin-releasing hormone; IGF-I, insulin-like growth factor-I; LH, luteinizing hormone; T3, triiodothyronine; T4, thyroxine; TRH, thyrotropin-releasing hormone; TSH, thyroid-stimulating hormone.

Leptin, produced in adipocytes, is also a marker of nutritional status and involved in the pulsatile secretion of GnRH. This hormone is markedly reduced in amenorrheic athletes (►**Fig. 6**).[59,63] Furthermore, thyroid hormones, and particularly triiodothyronine (T3), are reduced in response to a hypometabolic state, whereas thyroid-stimulating hormone (TSH) is usually in the normal range (►**Fig. 6**).[59,64] This condition should not be treated by levothyroxine as thyroid hormone status will be normalized together with the other endocrine disturbances by improved energy balance.

Taken together, amenorrhea related to energy deficiency in athletes can be explained by a functional hypothalamic inhibition of the reproductive system by stress hormones and endorphins, together with reduced stimulation of GnRH due to low levels of IGF-I and leptin.

The Female Athlete Triad

Relative energy deficiency and amenorrhea are associated with rapid loss of bone mass particularly of trabecular bone

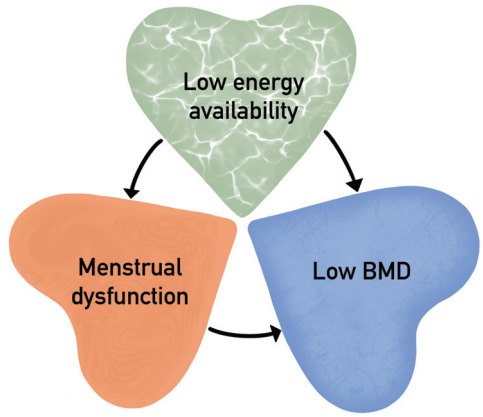

Fig. 7 The three components of the female athlete triad. BMD, bone mineral density.

such as the lumbar spine and pelvis.[5] Because physical activity usually promotes bone formation, it was initially considered paradoxical that elite athletes could develop reduced bone mass (i.e., osteopenia or even osteoporosis). However, this phenomenon is now known to arise from energy deficiency and its endocrine consequences including low levels of estradiol and IGF-I and increased levels of cortisol.[3,59]

The concept of "the female athlete triad" was first described at a consensus conference 1992 organized by the American College of Sports Medicine (ACSM) and defined as the three associated conditions: disordered eating, FHA, and osteoporosis.[65] A revised position stand was published in 2007 when the triad was redefined as a syndrome linking low energy availability (LEA) (with or without disordered eating), menstrual dysfunction, and low bone mineral density (BMD; ►**Fig. 7**).[66] A recent systematic review based on four studies showed that the triad, as well as LEA and menstrual disorders alone, was more prevalent in elite athletes than in controls, whereas low BMD (Z-score less than or equal to -2.0 adjusted for age and gender) demonstrated high heterogeneity among the studies.[67]

In 2014, the IOC published a consensus statement: "Beyond the Female Athlete Triad: Relative Energy Deficiency in Sport (RED-S).[56] The term "RED-S" broadened the concept of the female athlete triad to also include men and it refers to impaired physiological function including, but not limited to, metabolic rate, menstrual function, bone health, immunity, protein synthesis, and cardiovascular health caused by relative energy deficiency. In 2018, an update of this IOC consensus statement on RED-S was published.[68] It was concluded that there are still important gaps of knowledge regarding screening instruments to identify athletes at risk, prevention programs, knowledge of RED-S in male athletes, long-term health consequences, and the need of a practical guideline for the treatment and safe return to play.

Evaluation and Management of Menstrual Disorders in Female Athletes

Menstrual disorders are symptoms of hormonal imbalance and should always be investigated by physical and gynecological examination (unless the individual is adolescent or has not made her sexual debut) and endocrine evaluation. Amenorrhea can be either primary (spontaneous menstruation has never occurred) or secondary (absence of menstruation for at least 3 consecutive months), and oligomenorrhea meaning menstruation at increased intervals (>6 weeks, 5–9 periods during the past year).

Evaluation

The two most common underlying mechanisms to oligomenorrhea/amenorrhea in athletes seem to be FHA and PCOS. The typical clinical appearance of FHA is a lean woman with low body weight and/or body fat percentage. FHA is an acquired condition that can be normalized once the balance between energy intake and energy expenditure has been restored. A careful review of nutrition intake and eating habits in relation to training is important. On suspicion of eating disorders, the individual must be referred to a specialist clinic. The characteristic of an athlete with PCOS is instead a woman with anabolic body composition often presenting with oligomenorrhea. However, body weight is usually normal and symptoms of hyperandrogenism like acne and hirsutism may be less pronounced than PCOS in general.

Gynecological examination includes assessment of vaginal mucosa and ultrasound characteristics of the endometrium and ovaries. In FHA, there are often signs of vaginal dryness and the endometrium is thin due to estrogen deficiency, while PCOS may be associated with thickened endometrium. The ultrasound image of the ovaries in FHA can vary depending on when during the course of the functional disturbance the examination is performed. During periods of pronounced inhibition of the HPG axis, the ovaries are small with few visible follicles, while in connection with recovery, the ovaries become active again and often show a multifollicular appearance.[69] The most important difference between multifollicular ovaries and polycystic ovaries is that polycystic ovaries have increased stroma and many small follicles, whereas multifollicular ovaries do not have increased stroma and follicles are of larger size than in PCOS.

The endocrine evaluation is the most important for the determination of the underlying cause of menstrual disorders. FHA is characterized by clearly suppressed levels of LH, low levels of FSH, estradiol, testosterone, and T4/T3 but high levels of SHBG, whereas PCOS is associated with increased LH/FSH ratio, high levels of testosterone, and low levels of SHBG (►**Table 2**).[3,6,70] In most cases, there is an obvious difference in the hormonal profile between FHA and PCOS; however, in some athletes with amenorrhea, it can be a mixed underlying cause of both FHA and PCOS. When evaluating FHA, laboratory tests should also include measurement of complete blood count, electrolytes and liver panel, and other relevant nutritional factors.[70]

Table 2 Typical endocrine findings in FHA and PCOS

Hormone or binding protein	FHA	PCOS
FSH	↓	↔
LH	↓↓	↑
Estradiol	↓	↔
Testosterone	↓	↑
SHBG	↑	↓
Prolactin	↓	↔
TSH	↔	↔↑
Free T4/T3	↓	↔↓

Abbreviations: FHA, functional hypothalamic amenorrhea; FSH, follicle-stimulating hormone; LH, luteinizing hormone; PCOS, polycystic ovary syndrome; SHBG, sex hormone–binding globulin; T3, triiodothyronine; T4, thyroxine; TSH, thyroid-stimulating hormone.

Since longstanding FHA and LEA are associated with a loss of bone mass and increased risk of musculoskeletal injuries,[71] bone mass should be assessed preferably by dual-energy X-ray absorptiometry (DXA),[70] which is the golden standard method for the assessment of bone mass and body composition including body fat. In PCOS, hyperandrogenism appears to provide good protection from bone loss despite oligomenorrhea/amenorrhea and relative estrogen deficiency.

Management of Functional Hypothalamic Amenorrhea

Adequate nutrition in relation to energy expenditure should always be the first-line strategy of intervention in athletes with FHA, and counseling by a dietitian or nutritionist is recommended.[68,70] Optimized energy intake and increased body weight/fat mass have documented effect on restoration of menstrual function in athletes with FHA.[68,72] A gradual increase of 200 to 600 kcal/day have been recommended.[73] In case of low bone mass, supplementation of calcium and vitamin D may also be beneficial. Sometimes a reduction of exercise or prohibition to compete is necessary at the time of trying to regain energy balance.

One year without resumption of menses should lead to consideration of pharmacological treatment particularly in athletes with severe bone loss (osteoporosis) or fracture history. Bisphosphonates are not recommended as this treatment is not approved for use in premenopausal women. Transdermal estrogen in combination with cyclic progesterone/progestogen could be considered. This treatment has been demonstrated to improve bone mass in adolescent girls with anorexia nervosa.[74] However, oral estrogen like in oral contraceptives should be avoided as oral estrogen has a suppressive effect on hepatic IGF-I, which is a bone trophic factor.[70] It should be noted that low BMD is not the only indication for estrogen substitution in athletes with FHA but also because of other symptoms of estrogen deficiency such as endothelial dysfunction, adverse lipid profile, urogenital symptoms, dyspareunia, and sexual dysfunction.[73,75]

Management of Polycystic Ovary Syndrome

PCOS is a highly heterogeneous disorder, but according to international evidence-based guidelines, the syndrome should be managed depending on reproductive, metabolic, and psychological symptoms and the focus should be on healthy lifestyle behaviors.[76] There is evidence that regular physical exercise of at least moderate intensity can reduce androgen levels and body weight and possibly improve ovulatory function in women with PCOS.[15,77] The main mechanism for improvement is probably increased insulin sensitivity, resulting in reduced compensatory hypersecretion of insulin, which in turn leads to increased SHBG and thus lower levels of free testosterone. Athletes with PCOS could still be anovulatory, and in this case treatment with oral contraceptives or cyclic progestogens should be considered. Furthermore, combined oral contraceptives can improve symptoms of hirsutism and acne by antiandrogenic effects. There is no evidence that oral contraceptives impair physical performance, as long as body weight/body composition is stable.[78,79]

Management of Severe Hyperandrogenism and DSD

In a few cases, an athlete will be diagnosed with a rare disorder of severe hyperandrogenism or DSD. Many of these individuals have already been diagnosed and treated in early childhood due to ambiguous genitalia, but in some cases, they might enter puberty undiagnosed. During adolescence, individuals with DSD can present with primary amenorrhea or progressive virilization (hirsutism, deepening of the voice, absence of breast development, enlarged clitoris) in a phenotypic girl. It is consensus that clinical evaluation and care of women with DSD should be managed by a multidisciplinary team at specialist clinics.[80] The evaluation includes a thorough medical history focusing on hereditary factors, a comprehensive physical examination, ultrasound or magnetic resonance tomography of internal genital organs, karyotyping and specific genetic testing, as well as endocrine evaluation. The investigation includes assessment of the potential risk of germ cell cancer development. Treatment is dependent on the specific diagnosis and gender identity.

Conclusion

Endocrine disturbances and menstrual disorders are common among female elite athletes. The underlying cause could be a consequence of relative energy deficiency leading to FHA or essential hyperandrogenism. There is increasing evidence that hyperandrogenic conditions can be beneficial for athletic performance and could therefore play a role in the recruitment of women to sports activities. However, only XY DSD with testosterone levels in the male range, and not PCOS, is affected by eligibility regulations for the female classification in sports. It is of great importance to carefully evaluate menstrual disorders and hyperandrogenism in female athletes as the management and treatment are dependent on the underlying cause. Severe hyperandrogenism, that is, DSD, should be managed by a multidisciplinary team according to clinical consensus.

Conflict of Interest
The author is a medical and scientific consultant to the Swedish Olympic Committee and a member of the working groups on hyperandrogenic female and transgender athletes set up by World Athletics (WA) and the International Olympic Committee (IOC).

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