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# TESTOSTERONE AND SPORT: CURRENT PERSPECTIVES

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## Abstract

Testosterone and other anabolic-androgenic steroids enhance athletic performance in men and women. As a result, exogenous androgen is banned from most competitive sports. However, due to variability in endogenous secretion, and similarities with exogenous testosterone, it has been challenging to establish allowable limits for testosterone in competition. Endogenous androgen production is dynamically regulated by both exercise and winning in competition. Furthermore, testosterone may promote athletic performance, not only through its long-term anabolic actions, but also through rapid effects on behavior. In women, excess production of endogenous testosterone due to inborn disorders of sexual development (DSD) may convey a competitive advantage. For many years, female competitors have been subject to tests of sexual genotype and phenotype known as gender verification. Although gender verification has not identified any normal man competing as a woman, this process has identified women athletes with DSD. As understanding of DSD has expanded in recent years, women with DSD are increasingly able to continue athletic competition.

### Keywords

anabolic agents; sex characteristics; testosterone; epitestosterone; competition

The appropriate role of testosterone in sports is a frequent topic of debate. Although anabolic agents are banned from competition by most international sports federations, numerous competitors test positive every year. The resulting challenge is to discriminate illicit exogenous testosterone use from natural variation in endogenous androgen production in men and women. This review summarizes current understanding of the dynamics of endogenous testosterone production in competition, the allowable limits for identification of exogenous testosterone use, and the existence of naturally-occurring disorders that may elevate androgen levels in women athletes. The science of testosterone production and detection continues to shape current policy towards testosterone in sports.

Testosterone is both an endogenous hormone and a drug of abuse. Testosterone is the principal androgenic steroid produced by the testes. Testosterone is also a precursor to



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estrogen synthesis by the ovary in women. Steroids are hormones derived from cholesterol, and androgens promote the development and maintenance of male characteristics (Jones and Lopez, 2006). Many androgen actions in the body are mediated by binding to the androgen receptor, a nuclear receptor that modulates transcription of responsive genes (Rommets, 2004). In addition to their androgenic (masculinizing) effects, testosterone, and indeed all androgens, also have anabolic (muscle-building) actions. Hence, they are known collectively as anabolicandrogenic steroids (AAS).

Whether of endogenous or exogenous origin in males and in females, excess testosterone creates an advantage in sports (ACSM, 2006). Until recently, this concept was a matter of debate within the clinical and scientific community. While the anabolic effects of testosterone in hypogonadal males were well-accepted, early studies testing the effects of testosterone supplementation to eugonadal men were not well-controlled (reviewed in Bhasin et al, 2004). More recent studies have shown that testosterone stimulates muscle mass (Storer et al, 2003) and reduces body fat (Bhasin et al, 2004). Androgens likely also act on specific substrates in the brain to increase aggression and motivation for competition (Gleason et al., 2009; Hermans et al., 2008). Athletes aim to maximize their performance through the anabolic effects of testosterone and AAS, while limiting androgenic actions.

Starting in the mid-1930's after the chemical structure was published, testosterone's potential as a performance-enhancing drug was recognized and exploited by athletes. Use of androgens in athletics expanded in the 1950s and 1960s (van Amsterdam et al, 2010), coinciding with the development of synthetic AAS. Exogenous testosterone has been banned from Olympic competition since 1976, and was classified in the United States as a controlled substance by the Anabolic Steroid Control Act of 1990.

Testosterone is normally present in the circulation of both men and women. Due to the dynamic regulation of endogenous testosterone production, including the acute effects of competition and exercise, testosterone concentrations may vary considerably within and among individuals. Accordingly, it has been difficult to establish a threshold separating endogenous testosterone from exogenous sources. Furthermore, disorders of sexual differentiation (DSD) can produce elevated concentrations of endogenous androgens, potentially creating a competitive advantage for female athletes with DSD. For women with DSD, the standards for inclusion in major athletic competition continue to evolve.

## DYNAMICS OF TESTOSTERONE SECRETION

Defining the upper limit for endogenous testosterone is complicated by the dynamic changes in testosterone across a number of temporal scales. On the shortest time-scale, testosterone production in the gonads follows the pulsatile release of luteinizing hormone. This introduces a level of unpredictability for estimating circulating androgen concentrations in any single biologic sample. Secondly, in both sexes, testosterone follows a diurnal rhythm with peak concentrations in the morning followed by progressive decline over the course of the day, rising again at night during sleep (Dabbs, 1990a). In women, there is evidence that testosterone concentrations also vary as a function of the menstrual cycle, with peak testosterone concentrations in the periovulatory window, and lower values in the early follicular and late luteal phases (Dabbs and La Rue, 1991). On a somewhat longer time-scale, testosterone concentrations exhibit circannual variation and peak in the fall (Dabbs, 1990b; Stanton et al., 2011; van Anders et al, 2006). Lastly, men's testosterone concentrations slowly decline over the lifespan (Dabbs, 1990b) while women face an abrupt decline in testosterone at menopause (Schwenkhagen, 2007).

## Competition effects on testosterone

Exercise and competition also acutely alter testosterone concentrations, with obvious relevance to urine samples collected directly after athletic competition that are to be used for doping assessment. Generally, cardiovascular exercise and resistance training transiently increase testosterone concentrations in men (reviewed in Hackney [2001], Kraemer and Ratamess [2005]) and women (Enea et al., 2009), although a few studies report null effects. Testosterone concentrations also vary both before and after competition in a systematic and consistent manner.

**Pre-competition—**Wingfield and colleagues (1990) proposed the "challenge hypothesis", which posits that during mating seasons and times of resource scarcity testosterone concentrations rise to facilitate competition, particularly amongst males. The challenge hypothesis is relevant to human competition in the world of sports (Archer, 2006). An athletic match is a competition between individuals over a scarce and valuable resource (e.g. victory itself, prize money, fame, prestige). As predicted by the challenge hypothesis, pre-competition concentrations of testosterone rise in male and female athletes in anticipation of the impending competition (Bateup et al, 2002; Booth et al., 1989; Edwards and Kurlander, 2010; Mazur et al., 1997; Oliveira et al., 2009; Salvador et al, 2003; Suay et al, 1999). We speculate that the pre-competition increase in testosterone may facilitate competition by both increasing motivation to compete (Booth et al., 1989) and physical ability, but further studies of the mediating effects of pre-competition testosterone increases on changes in motivation and physical ability are needed.

**Post-competition—**In men, testosterone commonly increases following victory and decreases following loss (Booth et al., 1989; Elias, 1981; Gladue et al., 1989; Mazur et al., 1992; Mazur & Lamb, 1980; McCaul et al., 1992; Pound et al., 2009), including experiences of vicarious victory and defeat (Bernhardt et al., 1998; Stanton, Beehner, et al., 2009). Although, this main effect of winning or losing on changes in men's testosterone is not always observed (Gonzalez-Bono et al., 1999; Mazur et al., 1997; Serrano et al., 2000; Suay et al., 1999; van Anders & Watson, 2007). Several studies have shown that other factors like context (Carre, 2009), individual differences, e.g., power motivation (Schultheiss et al., 1999; Schultheiss & Rohde, 2002; Schultheiss et al., 2005; Stanton & Schultheiss, 2009), social anxiety (Maner et al., 2008), and motivation to win (Suay et al., 1999), as well as cognitive appraisal (reviewed in Salvador & Costa, 2009; Salvador et al., 2003; Serrano et al., 2000; Suay et al., 1999; van der Meij et al., 2010) can play an important role in predicting post-competition testosterone changes. Dominance-motivated individuals, who positively value interpersonal dominance and dislike submission, are those most likely to experience outcome-dependent changes in testosterone (Schultheiss, 2007; Stanton and Schultheiss, 2009). Competitors' level of engagement is also relevant to testosterone changes (van der Meij et al, 2010), such that men's testosterone increases are greatest when one's opponents feel more confident. An elite athlete in an international competition is likely to be more engaged and to value victory and defeat much more significantly than a participant in laboratory manipulations with cognitive games. Accordingly, testosterone changes in situations of high value and importance are likely to be of greater magnitude.

The "winner effect" and "loser effect" describe the post-competition changes in behavior as a function of winning and losing the contest, in which testosterone appears to be differentially involved. The winner effect describes the increases in dominance engagement and likelihood of winning future fights after recently winning dominance competitions, and the loser effect describes the withdrawal from and increased chances of losing further dominance interactions after losing recent fights (Dugatkin, 1997; Gleason et al., 2009). Both of these effects are argued to have adaptive benefits. A winner will likely benefit from

continued victory and increased access to resources, whereas a loser, who may be injured or still in the presence of the victory-primed winner, will likely benefit from disengagement (Mazur, 1985). Several animal studies have elucidated the mediating effects of testosterone in the winner and loser effect, which have been subsequently studied in humans. In castrated male rodents, experimentally-manipulated post-victory increases in testosterone facilitate increased aggression and victory in subsequent encounters, whereas the experience of victory alone does not (Trainor et al., 2004). However, when studying intact male mice, Oyegbile and Marler (2005) showed that the experience of victory increased a male's chance of winning future aggressive encounters, and in addition the experience of victory lead to endogenous increases in testosterone, i.e., post-victory endogenous testosterone increases have a reinforcing effect on aggressive behaviors that lead to victory. In addition, being in one's home cage (i.e., an effect of context) enhances the winner effect by facilitating greater testosterone change and increased incidence of winning future fights (Fuxjager et al., 2009), an effect that is at least partially corroborated by human research (Carre, 2009; Neave & Wolfson, 2003). As in other mammalian species, several studies of humans have documented an association between post-competition testosterone increases and increasing aggression, willingness to reengage in dominance behavior, and willingness to compete in another dominance contest, although those testosterone increases were not always associated with victory (Carre et al., 2010; Carre & McCormick, 2008; Carre et al., 2009; Mehta & Josephs, 2006). In contrast, the loser effect does not appear to be mediated by post-loss changes in testosterone (Oliveira et al., 2009), but this study was conducted in fish and the extent to which the result generalizes to humans is unknown. There are no human studies to our knowledge that show testosterone decrements mediate reductions in dominance or aggression post-loss in a dominance contest, in spite of such a hypothesis.

There is a notable sexual dimorphism in testosterone responses to competition in humans. Testosterone responses to winning and losing appear to principally apply to men. Only a single study has reported an effect winning/losing on differential testosterone changes in women (Oliveira et al, 2009), whereas many more studies have failed to find an effect (Bateup et al., 2002; Edwards & Kurlander, 2010; Edwards & O'Neal, 2009; Edwards et al., 2006; Mazur et al., 1997; Schultheiss et al., 2005; Stanton, 2011; Stanton, Beehner, et al., 2009; Stanton & Schultheiss, 2007; van Anders & Watson, 2007). This is likely a function of the different source glands for testosterone between the sexes, which include the testes and adrenals in men and ovaries and adrenals in women.

## Testosterone and motivation to compete

It is possible that high-testosterone individuals have increased motivation to compete in sports. High-testosterone individuals may select into sports as a function of testosterone's positive influence on dominance striving, also known as power motivation (Stanton and Schultheiss, 2009). Basal testosterone is positively correlated with power motivation in men (Schultheiss et al, 2003; Schultheiss et al, 2005), whereas basal estradiol is positively correlated with power motivation in women (Stanton & Edelstein, 2009; Stanton & Schultheiss, 2007). High concentrations of testosterone are also positively associated with selection into power-laden careers, e.g., trial law and acting (Dabbs et al, 1990; 1998). Knowing that power-motivated individuals are motivated to pursue dominance and find dominance experiences rewarding, the positive association between testosterone and power motivation suggests that high testosterone individuals may be the individuals most motivated to pursue athletic competition (e.g., Dabbs et al, 1990).

In addition to motivating dominance striving, testosterone is positively associated with a number of traits and behaviors that we speculate might foster advantage in competitive sports. For example, testosterone is associated with reduced responses to startling stimuli (Hermans et al., 2007; Hermans, Putman, Baas, et al., 2006) which may lead to reduced

distraction from negatively-valenced and arousing stimuli in the sports environment. Testosterone is also associated with reduced empathy (Hermans, Putman, & van Honk, 2006), reduced perception of negative emotions (van Honk et al., 2005), enhanced attention to social threat (van Honk et al., 1999), and enhanced amygdala responses to social threat (Hermans et al., 2008; but see also Stanton, Wirth, et al., 2009), which may promote an increased willingness and interest in attaining dominance over one's competitors independent of the consequences for one's competitors. Additionally, testosterone has been linked to increased risk-taking in economic domains (Stanton et al., 2011; van Honk et al., 2004, but see also Stanton, Mullette-Gillman et al. 2011) and social domains (Mazur, 1995). Lastly, testosterone is associated with enhanced visuospatial ability (Aleman et al., 2004) which may provide greater abilities in the perceiving critical targets and navigating the physical sports environment, i.e., field, rink, or court. Thus, high endogenous concentrations of testosterone may confer both psychological and physiological advantage in sports.

The foregoing discussion highlights the challenges in setting limits on ‘normal’ levels of testosterone to distinguish use of exogenous androgens. A single sample collected at the peak of endogenous testosterone production has potential to produce a false positive result, when compared against a population-based average. Thus, as different androgen formulations have proliferated among athletes in recent decades, there has been a corresponding increase in sophisticated standards for detection of exogenous AAS.

## EXOGENOUS TESTOSTERONE AND AAS

In the 2011 Prohibited List of the World Anti-Doping Agency (WADA), testosterone is classified among prohibited substances that are “Endogenous AAS when administered exogenously” (WADA, 2011). All synthetic AAS are derived from testosterone (Figure 1, reviewed in Wood, 2008). They have a carbon skeleton with 4 fused rings; most have 19 carbons. Modifications include hydroxylation at the C10 position to increase receptor binding affinity (e.g. nandrolone [Saartok et al, 1984]), esterification to slow release into circulation (e.g. testosterone cypionate), or alkylation at the C17 position to permit oral delivery by reducing first-pass metabolism in the liver (e.g. oxymetholone). AAS can be converted to highly-androgenic or estrogenic metabolites. For testosterone, dihydrotestosterone is the principle androgenic product; estradiol is the major estrogenic metabolite. Non-aromatizable AAS (e.g. drostanolone) have fewer estrogenic side-effects such as gynecomastia. Non-reducible AAS (e.g. oxandrolone) have fewer androgenic side-effects such as acne, baldness, and prostatic hypertrophy because they have lower binding affinity for the androgen receptor (Saartok et al, 1984).

For athletes subject to drug testing, a key drawback of synthetic AAS is that their use is easily detected, since their metabolites are not normally present (Ventura and Segura, 2010). An extended pre-competition wash-out period is necessary to avoid a positive test. This varies with the route of administration and the half-life of the individual AAS. However, long-acting AAS such as nandrolone can be detected for at least 6 months (Bagchus et al, 2005). By contrast, the urinary metabolites of exogenous and endogenous testosterone are virtually identical. Furthermore, testosterone is both relatively inexpensive and readily available (Summers, 2002). Many athletes take long-acting testosterone esters such as testosterone propionate (reviewed in Wood, 2008). Although esterification prolongs the half-life in circulation, the active steroid is still testosterone. In 2006, testosterone was the single most-common banned substance detected in urine tests at WADA-accredited laboratories, representing 26% of all “adverse analytical findings” (Figure 2, [WADA, 2006]). The AAS nandrolone was 4th (5.5% of positive tests). Likewise, testosterone accounted for the largest fraction (34%) of AAS-positive urine tests at the 2000 Sydney Olympic Games; nandrolone was detected in 32% (Van Eenoo and Delbeke, 2003).

## Testosterone and Epitestosterone

To overcome difficulties in setting a strict limit on allowable testosterone concentrations, without also identifying false positives among individuals with high endogenous testosterone, Manfred Donike proposed to compare the ratio of testosterone to its inactive 17a epimer, epitestosterone (see Catlin et al, 1996). Ordinarily, testosterone and epitestosterone are produced in equal amounts, and testosterone cannot be converted to epitestosterone. Accordingly, use of exogenous testosterone should increase the relative amount of testosterone vs epitestosterone (T/E ratio). The first tests at the 1983 Pan American Games used a 6:1 T/E ratio as the cut-off for a positive test (Yesalis, 2002). Fifteen athletes tested positive; additional athletes withdrew from competition and were not tested. Athletes soon recognized that taking epitestosterone along with testosterone could prevent a positive drug test. For this reason, epitestosterone is included on WADA's list of prohibited substances (WADA, 2011). In 2005, WADA reduced the upper limit for an allowable T/E ratio to 4:1.

**Competition effects on epitestosterone – what we (don't) know—**Epitestosterone changes, or more importantly, changes in the T/E ratio as a function of winning or losing a competition are unstudied to our knowledge. The mechanisms of epitestosterone production, synthesis, and binding are still not well characterized (Starka, 2003). Knowing that the T/E ratio is a critical metric of doping and that competition alters testosterone concentrations, should anti-doping agencies be concerned that the T/E ratio may be subject to systematic fluctuations as a function of exertion, competition, victory and defeat? Knowing that testosterone concentrations can change up to 100% post-competition (Oliveira et al, 2009), false positives in doping assessment based on the T/E ratio are plausible. This is especially true for winners, who tend to experience testosterone increases from both winning and exertion and are most likely to be tested post-competition. In this regard, Kicman and colleagues (1990) found that vigorous exercise did not significantly raise men's T/E ratio across all subjects , but their sample size was very small (n = 9). This study is one of very few to examine the effects of exertion on the T/E ratio -- more are needed and with larger sample sizes.

Most experimental studies of post-competition changes in testosterone have used serum or salivary sampling, and it is well established that serum and salivary testosterone levels are highly correlated (Ellison, 1988; Riad-Fahmy et al., 1982; Smith et al., 1979). Comparatively, few experimental studies have measured competition- or exercise-induced testosterone changes in urine, but at least one study of cyclists showed that urinary testosterone is increased as a function of competition (Maynar et al., 1994). Moving forward, athletes and testing authorities would each benefit from rigorous studies that examine variation in the T/E ratio in multiple bodily fluids (e.g., urine, serum, and saliva) as a function of pre- and post-competition changes in testosterone.

## Time-course of testosterone action

The classic model of steroid action is that steroid hormones have a relatively slow time-course of action by acting as transcription factors after binding to intracellular receptors (Rommets, 2004). Athletes exploit the persistence of AAS action. They know that the anabolic gains realized while on a pre-competition steroid “cycle” will persist for weeks after AAS use is discontinued (Summers, 2002).

However, research in animals has demonstrated behavioral effects of testosterone that occur within minutes (reviewed in Clark and Henderson, 2003). In the 1980's, East German scientists developed an androgen nasal spray to enhance aggression and competitiveness without systemic effects (Dickman, 1991). Similarly, intranasal 4,16-androstadien-3-one

induces an amphetamine-like “high” in human volunteers (Jacob et al, 2002). More recently, rapid actions of testosterone could have contributed to American cyclist Floyd Landis’ dramatic comeback in the mountainous 17th stage of the 2006 Tour de France. Although Landis was initially declared the winner of the 2006 Tour, his urine sample collected after the 17th stage revealed an elevated T/E ratio, consistent with use of exogenous testosterone (Walsh, 2007). Supplemental testosterone would not be expected to acutely enhance boost muscle function or exercise capacity, but could increase competitive drive.

## ENDOGENOUS ANDROGENS AND DISORDERS OF SEXUAL DIFFERENTIATION

In addition to the challenges of detecting illicit use of exogenous testosterone, the International Olympic Committee has struggled to define the role of endogenous testosterone in women's athletics. A variety of DSD may result in elevated androgen production in women. Given testosterone's role as a performance-enhancing substance, it is not surprising that this could convey a competitive advantage in athletics. Accordingly, DSDs have attracted attention in the context of ‘gender verification’ testing. Mandatory testing of all female athletes has been eliminated from Olympic competition. Nonetheless, individual athletes may be required by their sport federation to undergo testing for sexual genotype and phenotype based on a challenge by another athlete or a suspicion raised during sample provision for doping control (IAAF, 2006). Gender verification received widespread attention in 2009 with the South African middle distance runner, Caster Semenya. The International Association of Athletics Federations (IAAF) ordered Semenya to undergo gender verification following her win in the women's 800 m race at the IAAF World Championships in Athletics in Berlin. Based on the rapid improvement in her race times (almost 9 seconds from 2008 to 2009), initial suspicions focused on the possibility that Semenya was using performance-enhancing drugs. Urine tests after her race in Berlin cleared Semenya from the suspicion of exogenous androgen use. However, her appearance prompted speculation about possible endogenous sources of androgens related to DSD. It is appropriate that the results of her gender verification test have not been made public. In 2010, Semenya was permitted to resume women's track competition. This is consistent with current policy for athletes with DSD, which allows participation by women with “conditions that may accord some advantages”, including congenital adrenal hyperplasia, androgen-secreting tumors, and polycystic ovarian syndrome (IAAF, 2006). Nonetheless, gender verification is a somewhat subjective process, as demonstrated by Indian runner Santhi Soundarajan, who was stripped of her silver medal in the women's 800 m at the 2006 Asian Games following gender verification, despite previously being cleared for competition at the Asian Championships in 2005 (Terry, 2010).

At birth, an infant's sex is typically assigned on the basis of the external genitalia. DSD is often confused with “intersex”, referring to conditions where the external genitalia do not conform to the standards of either male or female (Ritchie et al, 2008). Individuals with DSD may present with a normal masculine or feminine phenotype, even if that does not accord with the genotypic sex. However, the external genitalia represent only one facet of masculine or feminine development, and we do not yet understand how differentiation of each sexually-dimorphic trait throughout the body and brain is coordinated.

### Gender vs sex

Unfortunately, ‘gender verification’ is a misnomer that confuses the distinction between sex and gender. Sex is a biologic definition that distinguishes male and female; gender is the sense of one's own self as a man or woman (see Wilson, 2000). As such, gender represents a combination of biologic and cultural factors. And ultimately, each of us defines our own

gender. Accordingly, the distinction of male or female sex applies to all animals, but only humans can be said to have gender. Nonetheless, we will continue to refer to gender verification, since this is the term most widely in use.

## Disorders of sexual development and athletic performance

DSD may include alterations in the complement of sex chromosomes, as well as defects in the production, metabolism or binding capacity of sex steroid hormones produced by the gonads. Athletes have been blocked from competition after testing, presumably due to DSD. Because the results of these tests are not made public, definite evidence is often not available.

**Abnormalities in sex chromosomes—**Individuals may have only one sex chromosome (Turner's syndrome), or sometimes three or four [Klinefelter's syndrome (XXY) is the most common example; Bojesen and Gravholt (2007)]. This results from non-disjunction of sex chromosomes during meiosis in the developing spermatozoan or oocyte (Jones and Lopez, 2006). Typically, individuals with Turner's or Klinefelter's syndrome are hypogonadal, which is not a competitive advantage in sport. Nonetheless, IAAF policy specifically permits women with Turner's syndrome to compete (IAAF, 2006).

**XX/XY genotype (True hermaphrodite, mixed gonadal dysgenesis):** True hermaphrodites may have an ovary opposite a testis, or they may develop with ovarian and testicular tissue in the same gonad (ovotestis; Kim et al, 2002). Their sexual phenotype is typically ambiguous at birth, depending on the amount of testosterone produced by the gonadal tissue. In 1950, sprinter Foekje Dillema was expelled 1950 from the Dutch national team and banned from international competition following a gynecologic exam. A posthumous investigation revealed her to be a 46:XX/46:XY mosaic (Dohle, 2007).

Mixed gonadal dysgenesis differs from true hermaphroditism in that individuals have unilateral gonadal development, with a streak gonad contralaterally (Davidoff and Federman, 1973). The genotype is often 45:XO/46:XY. As with true hermaphrodites, the genital phenotype at birth is variable, perhaps reflecting delayed testicular maturation. Although many affected individuals are initially reared as females, further masculinization may develop at puberty, and some will subsequently switch to a masculine gender identity (Reiner, 2005). Currently, gonadal dysgenesis is permissable in IAAF competition, with the proviso that the gonads are removed to prevent malignancy (IAAF, 2006).

**Abnormalities in androgen production and androgen responsiveness—**Many DSDs are caused not by abnormalities in the sex chromosomes, but by autosomal disorders that cause alterations in androgen production or androgen responsiveness (Jones and Lopez, 2006).

**Congenital adrenal hyperplasia (CAH):** CAH results from one of several mutations in the steroidogenic enzymes synthesizing cortisol in the adrenals. The most common is a deficiency in the 21-hydroxylase enzyme that converts 17a-hydroxyprogesterone into 11-deoxycortisol, the precursor to cortisol. Lacking the ability to generate cortisol or aldosterone, their precursors accumulate in the adrenal and are converted to androgens. Typically, the adrenals begin to synthesize excess androgens at the start of the 2nd trimester, when sexual differentiation normally takes place. With diminished cortisol negative feedback, the levels of adrenocorticotrophic hormone are elevated (Dauber et al, 2010). This further drives androgen production. Virilization of affected females is variable at birth. By promoting masculine development, adrenal androgens in CAH would be expected to convey a competitive advantage in sports for women.

**Androgen insensitivity syndrome (AIS):** XY individuals with AIS have a defect in the androgen receptor, rendering them insensitive to their own testicular androgens (see Hiort and Zitzmann, 2004). At birth, affected XY infants have intraabdominal testes, and a female phenotype. The condition is often unrecognized in childhood: they are raised as girls, and their gender identity is female. Women with AIS are often tall because puberty begins later. While complete AIS would be unlikely to improve athletic performance, incomplete forms of AIS can produce intermediate masculinization of affected women. In 1985, the Spanish hurdler Maria Jose Martinez-Patino was blocked from competition at the World University Games due to AIS (Martinez-Patino, 2005). As was customary at the time, she was advised to claim injury. When she continued to race, her condition was revealed publicly. Dr. Martinez-Patino's efforts to contest chromosome-based gender verification increased acceptance of women athletes with DSD. Her eligibility was reinstated three years later, but she failed to qualify for the 1992 Olympics in Barcelona.

**5a-reductase deficiency (guevedoces):** Individuals with 5a-reductase deficiency lack the ability to convert testosterone to DHT (Houk et al, 2005). DHT has a high affinity for AR binding, and plays a particularly important role in the prenatal masculinization of the external genitalia. Lacking DHT, the external genitalia of XY males are feminized at birth, although the testes descend into the labia majora under the influence of testosterone (Wilson et al, 1993). During puberty, elevated levels of testosterone induce virilization and male secondary sexual characteristics. At this point, some affected individuals will switch gender identity to live as men. Due to high androgens in circulation, 5a-reductase deficiency in an XY athlete competing as a woman would provide a competitive advantage.

## History of Gender Verification

In the 1930's, concerns were raised about the masculine appearance of several female athletes, most notably two competitors in the 100 meter event at the 1936 Olympic games in Berlin (Ritchie et al, 2008). The American sprinter Helen Stephens won the gold medal, and was subject to an impromptu genital examination by the Olympic committee, which judged her to be female. Ironically, the accusations against Stephens were brought by Polish runner Stella Walsh, herself a somewhat masculinized athlete (dubbed “Stella the Fella” in the press). At her death in 1980, an autopsy revealed that Walsh possessed ambiguous genitalia.

Obligatory testing of all female competitors began in the 1960's and continued until 1999 (Figure 3). Initial tests in the 1960's were based on phenotypic characteristics by direct physical and/or gynecologic examination. Such tests would have potential to identify as males both XX athletes with masculinization due to CAH, and XY competitors with incomplete AIS or partial genital masculinization due to 5a-reductase deficiency. Chromosomal screening was instituted at the 1968 Olympics. At first, chromosomal tests verified the presence of a Barr body in buccal smears (Simpson et al, 2000). By Barr body screening, male competitors with Klinefelter's syndrome or female athletes with CAH are identified as female, but women with AIS are identified as male. Later tests screened female athletes for the presence of a Y chromosome, and subsequently for the SRY gene on the Y chromosome (Simpson et al, 2000). As with the Barr body, testing for SRY identifies AIS women as male. Using this method, 8 of 3387 women competing in the 1996 Atlanta Summer Olympics were SRY-positive (Genel, 2000). 7 had AIS; 1 had been gonadectomized subsequent to diagnosis with 5a-reductase deficiency. All were permitted to compete.

## Gender verification in 2011

Gender verification for all female competitors was finally dropped from Olympic competition in 1999 (Dickinson et al, 2002). The purported goal of gender verification was

to prevent males from posing as females in athletic competition. The requirement that top finishers produce a urine sample under direct observation would seem likely to catch any male competitors posing as women (Women's Sports Foundation, 2010). Indeed, there has been no recorded instance of a normal man competing as a woman (Simpson et al, 2000). Instead, gender verification has identified competitors with DSD.

Ultimately, world-class athletes have a variety of physical and psychologic attributes that enable their success in competition. Furthermore, there is increasing recognition that gender does not conform to a strict binary division. Indeed, transsexual athletes are now allowed to compete in the Olympics in their adopted gender, providing they have undergone gonadectomy with appropriate hormonal replacement for at least 2 years (Reeser, 2005). This would appear to represent the logical extension of the concept that an individual who identifies as a woman, and lives as a woman, should be allowed to compete as a woman.

**Testosterone in sport—**For much of the 20th century, a detailed understanding of testosterone dynamics lagged behind the empirical application of testosterone by athletes in competition. However, the expanding scientific expertise in measurement of testosterone and other AAS has potential both to apprehend athletes who misuse androgens in competition, and to defend those whose endogenous androgen production may fall outside the ‘normal’ range.

For the athlete who tests positive, their knowledge of other competitors who have used banned substances without detection must be bitter. When it comes to doping in international competition, it is very likely that almost “everyone's doing it” (Walsh, 2007). Ongoing advances in androgen detection will be offset by those athletes with better research, better access, and better monitoring to avoid an “adverse analytical finding”. Indeed, some have argued that we should stop trying to catch the cheats, and allow athletes to use whatever substances they like (discussed in Murray, 2010). According to their position, the athletes are only harming themselves. However, this view reflects a common misconception that steroid use is restricted to elite athletes. Instead, steroid use has infiltrated high school sports and neighborhood fitness centers (Johnston et al, 2003; Eaton et al, 2006). Thus, before we accept unregulated steroid use, it is important to acknowledge how widespread the problem is. The argument that “everyone's doing it”, does not take into account who “everyone” is.

For the women with DSD, the increased understanding of endogenous conditions that result in elevated androgen production has increased acceptance of affected individuals in women's athletics. Early testing for gender verification was humiliating and insensitive. Women cleared for competition were issued a “Certificate of Femininity”; those who ‘failed’ gender verification were officially barred from competition. Others simply withdrew from athletic contests. In the ensuing decades, further information about individual tests and allowable endocrine limits has been revealed. This has fostered continued debate in the athletic, scientific and medical community. Even so, findings from individual competitors remain guarded to protect the athlete's privacy. Competitors such as Caster Semenya have endured intense public scrutiny, but also have ultimately achieved acceptance in their sport.

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## Highlights

- Endogenous and exogenous androgens convey a competitive advantage in sports.
- Androgens can have rapid effects in brain to enhance competition.
- Winning a competition stimulates endogenous androgen secretion.
- Disorders of sexual differentiation may increase androgens among female athletes.

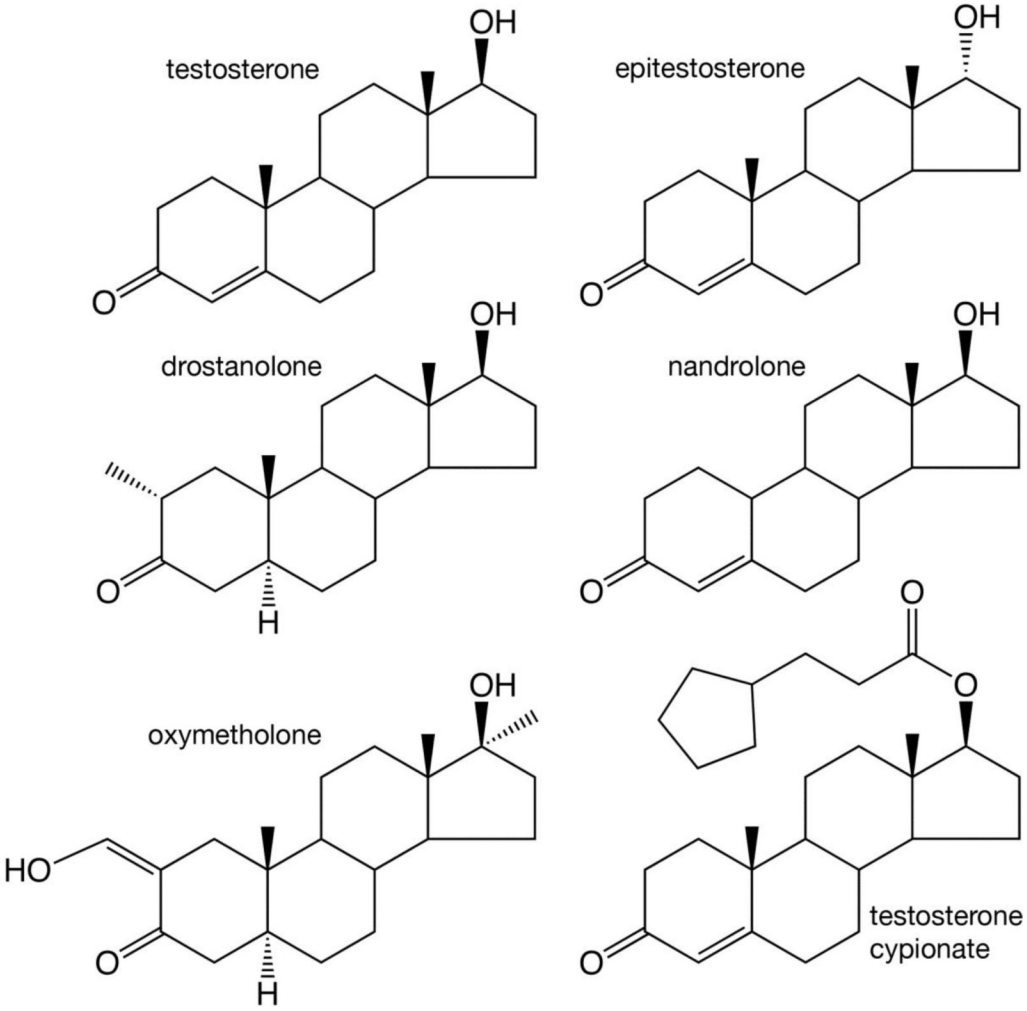


**Figure 1.**
Molecular structure of testosterone, epitestosterone, and examples of popular anabolic-androgenic steroids.

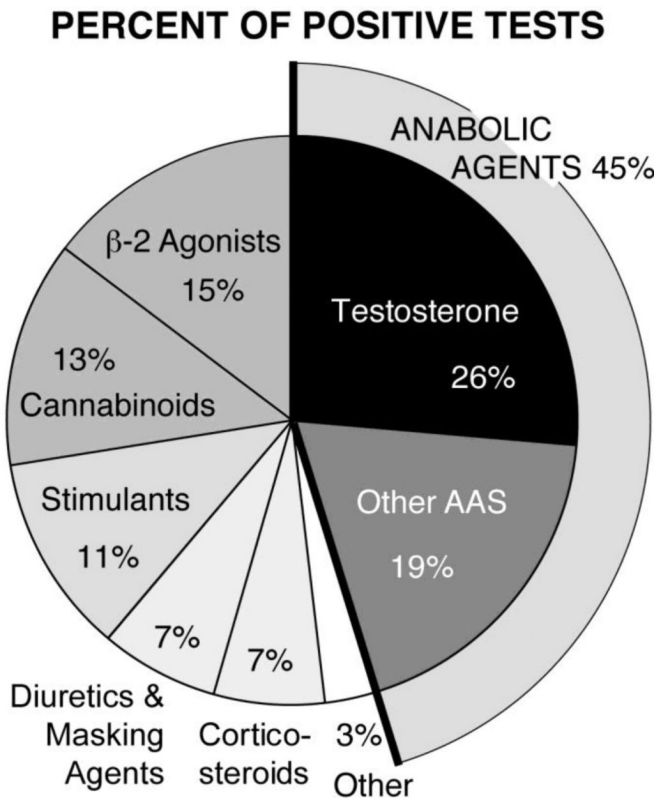


**Figure 2.**
Percent of adverse analytic findings reported by the World Anti-doping Agency in 2006. Abbreviation: AAS, anabolic-androgenic steroids. From [34].

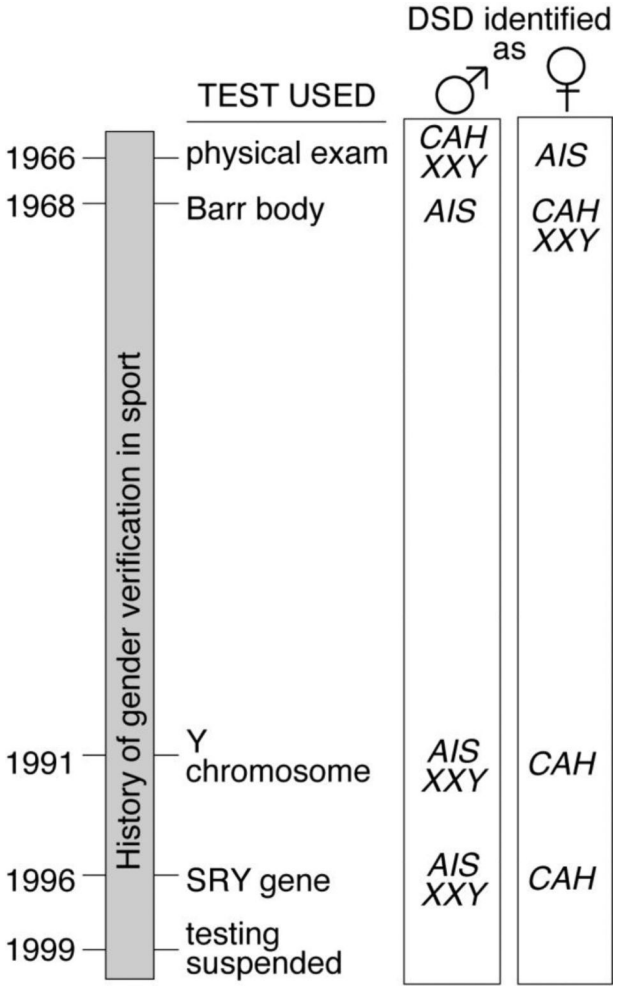


**Figure 3.**

Time-line of gender verification testing, showing the method used and the resulting gender for individuals with selected disorders of sexual differentiation (DSD). Abbreviations: AIS, androgen insensitivity syndrome; CAH, congenital adrenal hyperplasia; SRY, sex-determining region of the Y chromosome; XXY, Klinefelter's syndrome.