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ANABOLIC ANDROGENIC STEROIDS INTAKE AND ITS IMPACT ON MALE REPRODUCTIVE SYSTEM- SYSTEMATIC REVIEW

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ABSTRACT

Anabolic-androgenic steroids (AAS) are a significant lifestyle factor in infertility and are often used to enhance physical performance and appearance. Endogenous AAS regulate male sex organ development and secondary characteristics. Commonly used AAS include testosterone esters like undecanoate and enanthate, as well as nandrolone esters, testosterone cypionate, and testosterone propionate—along with nandrolone esters, such as nandrolone decanoate and nandrolone phenylpropionate, stanozolol and methandrostenolone (also known as methandienone). AAS bind to androgen receptors, promoting muscle growth by increasing protein synthesis and promoting the growth hormone-insulin-like growth factor 1 axis. AAS are commonly administered via intramuscular injection or oral ingestion. Injectable forms of AAS have extended half-life due to esterification, while oral AAS face reduced bioavailability because of first-pass metabolism. Adverse effects of AAS include cardiovascular, endocrine, neuropsychiatric and dermatological issues. AAS usage has a

huge impact on male fertility. Anabolic androgenic steroids intake can result in suppressing gonadotropins, leading to reduced sperm concentration, testicular dysfunction and hypogonadism, which can lead to decreased libido and erectile dysfunction. While some effects on fertility may be reversible, recovery timelines vary.

MATERIALS AND METHODS

An extensive examination of articles published in scientific journals was carried out through online research platforms PubMed and Google Scholar. We searched articles by entering keywords in appropriate configuration: “anabolic steroids”, “AAS”, “bodybuilding”, “doping in sports”, “erectile dysfunction”, “infertility”, “male reproductive health”.

KEYWORDS

anabolic steroids; AAS; bodybuilding; doping in sports; erectile dysfunction; infertility; male reproductive health;

CURRENT STATE OF KNOWLEDGE

Anabolic-androgenic steroids are a significant lifestyle factor in infertility.[1] Anabolic-androgenic steroids (AAS) are often used by men to improve physical performance and due to visual effects. The use of anabolic steroids impacts not only professional athletes but also the general population, including bodybuilders, gym-goers, and adolescents. For professional athletes, steroid use is banned and penalized by the World Anti-Doping Agency and Olympic committees. Among other groups, however, determining the prevalence of steroid use is challenging, as many acquire these substances through online sources. Motivations for use vary widely, and diverse usage patterns and user profiles have been reported. Among the side effects, hypogonadism is the most common reason for seeking endocrinological consultation.[2]

Mechanism of action

Endogenous AAS are responsible for the growth and development of the sex organs in men and maintaining secondary sex characteristics.[3] The anabolic-androgenic steroids (AAS)

most commonly used in medicine include testosterone and its various esters—primarily testosterone undecanoate, testosterone enanthate, testosterone cypionate, and testosterone propionate—along with nandrolone esters, such as nandrolone decanoate and nandrolone phenylpropionate. Other frequently used AAS include stanozolol and methandrostenolone (also known as methandienone).[4] Endogenous anabolic steroids exert their effects through binding and activation androgen receptors. In skeletal muscle, these steroids influence the transcription of specific target genes that regulate DNA synthesis, a crucial process for muscle growth.[5][6] The primary mechanism for increased muscle mass and performance is attributed to the anabolic effects on actin and myosin, via somatomedins. There is a trend toward increased myosin heavy chain, suggesting that testosterone improves skeletal muscle mass and stimulates protein synthesis, rather than decreasing muscle protein breakdown.[7] Anabolic-androgenic steroids (AAS) affect various central nervous system neurotransmitters, inhibition of glucocorticoid activity, and activation of the growth hormone-insulin-like growth factor 1 (GH-IGF-1) axis.[3]

Administration

AAS are most commonly administered by intramuscular (i.m.) injection or by oral ingestion. AAS formulations for intramuscular injection are based on vegetable oils because of the testosterone ester structure. T he AAS gradually diffuse out of the oil depot into the interstitial fluid. Esterification of the 17β-hydroxyl group enhances the lipophilicity of the compound. Unmodified testosterone has an estimated half-life of approximately 10 minutes.[8] In contrast, esterification of the 17β-hydroxyl group extends its half-life to approximately 4.2 days.[9][10]

Orally ingested AAS are rapidly absorbed in the gastrointestinal (GI) tract, with serum concentrations peaking 1–2 hours after ingestion of methyltestosterone.[11] The absorbed AAS reach the liver through the portal vein. Without structural modification significant fraction of the absorbed AAS will be metabolized before leaving the liver. This drastically decreases oral bioavailability. For example, after oral administration of 25 mg testosterone, less than 1 mg (4%) becomes systemically available. The oral bioavailability of AAS can be increased due to esterification process. This modification allows a larger fraction of the absorbed AAS to enter the lymphatic system and bypass first-pass metabolism. Even though bioavailability remains poor at the level of 6.8%.[12] Once in the systemic circulation, anabolic-androgenic steroids (AAS) are transported to target tissues by binding to plasma proteins, including albumin, sex hormone-binding globulin (SHBG), corticosteroid-binding

globulin (CBG), and orosomucoid. Under physiological conditions, testosterone is primarily bound to albumin and SHBG, with only 1% to 4% of circulating testosterone remaining unbound.[13]

As many as 90% of anabolic androgenic steroid (AAS) users combine or "stack" several androgens, a practice believed to enhance results while reducing unwanted side effects. Typically, a bodybuilding cycle involves stacking multiple AAS at a total dosage of 500–1,500 mg per week, lasting an average of 4 to 12 weeks.[14][15]

Indications

FDA-approved indications for the use of anabolic steroids are primary hypogonadism, delayed puberty in boys , hypogonadotropic hypogonadism, gonadotropin and luteinizing hormone- releasing hormone deficiency, pituitary-hypothalamic axis dysfunction. Other indications for the use of testosterone include primary testicular failure in patients with e.g. cryptorchidism, orchitis, Klinefelter syndrome, toxic damage from alcohol use, and heavy metals.

Off-label indications for androgenic steroids include bone marrow stimulation in leukemia, aplastic anemia, kidney failure, growth failure, stimulation of appetite, and muscle mass in malignancy and acquired immunodeficiency syndrome.[3]

Adverse effects

Anabolic androgenic steroids (AAS) can cause various adverse effects due to their interaction with androgen receptors and the disruption of the biosynthesis, transformation, and degradation of endogenous steroids. These effects are observed in both animals and humans, and the widespread presence of androgen receptors in the body contributes to these outcomes. It's important to distinguish between side effects from medical use under supervision and those resulting from abuse, which involves high doses and polydrug use, complicating the attribution of effects to AAS alone. Additionally, the effects of AAS depend on factors such as sex, dosage, and the duration of administration, with most adverse effects appearing after long-term use.[7]

System	Adverse Effects
Cardiovascular	Coronary heart disease, cardiomyopathy, hypertension (3% or less)
Endocrine and Metabolic	Decreased HDL cholesterol (6% or less), hyperlipidemia (6% or less), hypokalemia, increased serum triglycerides, increased thyroid-stimulating hormone level, increased plasma estradiol concentration, decreased libido (3% or less), gynecomastia (3% or less), hot flashes, weight gain
Gastrointestinal	Gingivitis (9% or less), mouth irritation (9% or less), increased serum bilirubin, abnormal hepatic function tests, decreased appetite, dysgeusia, gastroesophageal reflux disease, gastrointestinal hemorrhage
Genitourinary	Increase in prostate-specific antigen (topical 18% or less), benign prostatic hypertrophy (12%), testicular atrophy (6% or less), suppression of spermatogenesis, mastalgia, hypogonadism (following withdrawal), prostatitis, dysuria, hematuria, impotence, pelvic pain, urinary incontinence, urinary tract infection, testicular tenderness, ejaculatory disorder, erectile dysfunction (nandrolone)
Hematologic and Oncologic	Polycythemia (6%), prostate carcinoma (less than 3%)
Neuromuscular and Skeletal	Myalgia (6% or less), premature epiphyseal closure (when taken before completion of puberty), limb pain, tendon rupture, abnormal bone growth, hemarthrosis
Neuropsychiatric	Emotional lability, major mood disorders, anosmia, headache, depression, nervousness, body pain, violence, insomnia, aggressive behavior
Dermatologic	Skin blister (12%), acne vulgaris (8% or less), crusted skin, nasal excoriation (6% or less), contact dermatitis, bulla, skin rash, pruritus
Renal	Increase in serum creatinine, increased frequency of urination
Specific Adverse Effect of Nandrolone in Women	Hirsutism, deepening of voice (with extended use)

Tab. 1. Adverse effects of AAS intake. [3]

Neural system

The neurotoxic action of AAS is associated with both membrane AR and G-protein coupled receptors.[16] Furthermore, several studies highlighted the role of apoptosis in determining brain damage . Indeed, it was demonstrated that high concentrations of methandienone and 17-a-methyltestosterone provoke detrimental effects on neuron cell cultures expressing AR,

inhibiting neurite network maintenance, leading to cell death through apoptosis and cleavage of protective chaperone proteins, such as Hsp90.[17]

A recent study highlights the role of microRNA (miRNA) dysregulation in anabolic androgen steroid (AAS)-related brain damage, specifically noting elevated levels of miR-34 and miR-132 in AAS users compared to cocaine abusers and aging individuals.[18] The research suggests that long-term nandrolone treatment in rats may induce apoptosis in brain regions, linking oxidative stress with NF-kB signaling and resulting in brain injury, particularly in the hippocampus, striatum, and frontal cortex.[19] Additionally, daily stanozolol injections for 28 days caused histopathological changes in the hippocampus by activating apoptotic and pre-apoptotic cells.[20] Another study found that supraphysiological doses of AAS could negate the positive effects of physical activity on hippocampal cell proliferation and apoptotic signaling.[21] Conversely, endurance exercise appears to enhance redox balance and stabilize mitochondrial membranes, thereby reducing apoptotic effects associated with nandrolone in neural cells.[22]

Cardiovascular system

Notwithstanding the elevated morbidity and mortality, cardiac and metabolic consequences of AAS abuse are still unclear.[23] Cardiac injury is the most frequent consequence of the administration of exogenous steroids, due to its susceptibility to oxidative stress and its important metabolic activity, compared with the remaining body tissues and organs.[24] Chronic administration of high doses of AAS is responsible for the dysfunction in tonic cardiac autonomic regulation. Indeed, an experimental study demonstrated that rats treated with AAS were characterized by the impairment of parasympathetic cardiac modulation, decreased high frequency power and heart rate variability.[25] Furthermore, the inflammatory process may play a role in triggering cardiac injury in AAS abusers. In fact, in a mouse model a strong cytokine reaction was observed in mice treated with anabolic steroids compared to the control group, suggesting a role of TNF-α in determining myocardial injury.[26]

Sport and common use

In the United States, between 1 million and 3 million people (1% of the population) are thought to have used AAS.[27] Studies in the United States have shown that AAS users tend to be mostly middle-class men with a median age of about 25 who are noncompetitive

bodybuilders and non-athletes and use the drugs for cosmetic purposes.[28] According to a recent survey, 78.4% of steroid users were noncompetitive bodybuilders and non-athletes, while about 13% reported unsafe injection practices such as reusing needles, sharing needles, and sharing multidose vials.[29]

Anabolic steroids are frequently utilized by athletes as a form of illegal chemical doping. Initially, particularly during the 1970s and 1980s, their use was not formally prohibited, as these substances were emerging on the market at a pace that outstripped changes in sports regulations.

Anabolic steroids are particularly prevalent in strength sports, where success is primarily determined by muscle strength and endurance. AAS have been used by men and women in many different kinds of professional sports to attain a competitive edge or to assist in recovery from injury. These sports include bodybuilding, weightlifting, shot put and other track and field, cycling, baseball, wrestling, mixed martial arts, boxing, football, and cricket. AAS use occurs among adolescents, especially by those participating in competitive sports. It has been suggested that the prevalence of use among high-school students in the U.S. may be as high as 2.7%.[30]

In the mid-1990s, the International Olympic Committee imposed a complete ban on the use of anabolic steroids in official sports, which led to similar prohibitions in most countries that send athletes to the Olympic Games.

Contemporary detection methods for anabolic steroid use, such as gas chromatography of hair samples, allow for the identification of their consumption even two to three years after the fact.[29]

Impact on male fertility

According to a meta-analysis from 2023 the use of anabolic-androgenic steroids (AAS) for performance enhancement is a well-documented phenomenon associated with significant adverse effects on male fertility. AAS use has been shown to suppress gonadotropin secretion, leading to reduced sperm concentration and motility, as well as testicular atrophy. [31]

Spermatogenesis, like testicular testosterone production, is regulated by the hypothalamic-pituitary-gonadal axis (HPGA). The coordinated actions of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) are essential for stimulating spermatogenesis, while suppression of these hormones inhibits the process. FSH directly promotes spermatogenesis by binding to FSH receptors on Sertoli cells. LH, on the other hand, acts indirectly by

stimulating testosterone production through the activation of luteinizing hormone/choriogonadotropin receptors (LHCGR) on Leydig cells. The testosterone produced then binds to androgen receptors (AR) on Sertoli cells to further support spermatogenesis.[30] Although testosterone is the primary mediator of luteinizing hormone (LH) effects on spermatogenesis, exogenous testosterone administration cannot sustain this process. Intratesticular testosterone (ITT) levels are naturally 50 to 100 times higher than circulating levels, and exogenous testosterone significantly suppresses ITT to concentrations insufficient to support spermatogenesis.[33] Hormonal male contraception involves the administration of exogenous androgens, which suppress gonadotropin production through negative feedback on the hypothalamic-pituitary-gonadal axis. This suppression reduces intratesticular testosterone production, thereby inhibiting sperm maturation.[34] In the HAARLEM study, nearly all participants exhibited undetectable levels of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) during anabolic-androgenic steroid (AAS) use. However, at the end of their cycles, only two-thirds of the participants were azoospermic or oligozoospermic.[35] The duration of suppression and the resulting symptomatic anabolic steroid–induced hypogonadism (ASIH) can vary significantly due to several factors, including the type of drugs used, the quantities, and the duration of use. There may also be differences in how individuals respond to the suppression of the HPG axis.[36] The arrest of spermatogenesis leads to testicular atrophy. Suppression of spermatogenesis by sex steroids reduces testicular volume by 16.5–30.0%. [35]

A cross-sectional observational study compared current AAS users with past users and nonusers to assess the mean time for recovery of sperm output and concentration. The authors estimated recovery time by calculating how long it took for AAS users to reach the mean values observed in the nonuser group, based on a linear regression of the variable over time since AAS cessation. The mean recovery times for sperm output and concentration were 14.1 months and 10.4 months, respectively. [37] HAARLEM research indicated that mean sperm concentration decreased from 46.8 million/mL to 11.7 million/mL during AAS use, with 68% of subjects meeting the criteria for oligozoospermia or azoospermia (<15 million/mL). Notably, 28% of subjects already met this criteria at baseline, indicating potential incomplete recovery of spermatogenesis from prior AAS use. Three months after cessation, mean sperm concentration remained significantly lower than baseline. [35]

Erectile dysfunction (ED) is a relatively common issue among anabolic-androgenic steroid (AAS) users. In the HAARLEM study, 8% of participants reported ED at baseline, increasing to 12% during AAS use. ED often develops following an AAS cycle due to the transient

hypogonadal state, where testosterone deficiency typically results in reduced libido and impaired erectile function. During this period, dissatisfaction with sexual performance, frustration in relationships with partners, and diminished self-confidence may contribute to the persistence of ED even after testosterone levels normalize. [35] ED is also occasionally reported during active AAS use, though the underlying mechanisms remain unclear. One hypothesis involves an imbalance between androgenic and estrogenic activity, as estradiol, independent of testosterone, plays a role in regulating erectile function. Psychological factors may also contribute; the heightened libido experienced by some AAS users during cycles can sometimes disrupt healthy and mutual sexual relationships, potentially exacerbating ED. [5]

Summary

Anabolic-androgenic steroids (AAS) are widely used to enhance physical performance and appearance, but their use has significant health implications, especially for male fertility. AAS can suppress gonadotropin secretion, reduce sperm concentration and motility, and cause testicular atrophy. These effects are linked to the disruption of the hypothalamic-pituitary-gonadal axis, which regulates sperm production. Additionally, AAS use can lead to hypogonadism, erectile dysfunction (ED), and a variety of other adverse effects, including cardiovascular, endocrine, gastrointestinal, and neuropsychiatric issues. While AAS are approved for certain medical conditions, their abuse, particularly in bodybuilding and sports, poses serious health risks. Recovery of sperm concentration after cessation of AAS use can take months, with some users experiencing long-term fertility issues. The prevalence of AAS use is particularly high among non-competitive bodybuilders and adolescents, often driven by cosmetic goals rather than athletic performance.

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