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Anabolic Androgenic Steroids: No Longer on the Fringes of Endocrinology

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In this review, "Anabolic Androgenic Steroids," or "AAS" is used as the preferred term to indicate the use of exogenous androgens in supraphysiologic doses for the purpose of muscle growth or image enhancement. This definition does not include the use of physiologic doses of testosterone prescribed for the treatment of male hypogonadism. Although there is disagreement regarding the accuracy of this terminology, AAS continues to be one of the most common designations used in the literature today.

Background/Prevalence

Public awareness of AAS has historically focused on athletes seeking a competitive advantage over their peers.[1] More recently, this pattern has shifted, with the majority of AAS use occurring among recreational users whose motivations differ from those of athletes.[2,3] While these substances were once available only through black market sources, they are now widely available via social media, internet websites, and direct mailing of substances to consumers, despite laws prohibiting their use in many countries.[4] As recreational AAS use has increased, so too have referrals to endocrinologists for issues ranging from infertility and adverse drug effects to assistance with discontinuing AAS use altogether. Previously, some physicians have been reluctant

Drug name (Trade name)	Route
17β Ester Derivatives	
Testosterone propionate	IM
Testosterone enanthate	IM
Testosterone cypionate	IM
Testosterone undecanoate	IM/PO
Boldenone undecylenate (Equipoise®)	IM
17α Alkyl Derivatives	
Metandienone (Dianabol®)	PO
Methyltestosterone (Android®)	PO
Oxandrolone (Anavar®)	PO
Oxymetholone (Anadrol®)	PO
Stanozolol (Winstrol®)	PO/IM
19-Nortestosterone Derivatives	
Nandrolone decanoate (Deca-Durabolin®)	IM
Trenbolone acetate (Parabolan®)	PO, IM, pellets

Table 1. Commonly Used Anabolic Androgenic Steroids listed by name and route; *adapted from Grant et al., 2024*[4]*; Solanki et al., 2023.*[9]

Abbreviations: IM: intramuscular; **PO:** oral.

to accept such referrals due to a lack of expertise in this area or ethical concerns related to AAS use.[3,5] However, the emergence of commercial testosterone clinics, the ease of purchasing medications online, the growing availability of guidance from internet forums,[4,5] and artificial intelligence tools have led many physicians to realize that AAS management can no longer be exclusively confined to specialized clinics.

AAS use is increasing, with lifetime prevalence estimates ranging from 1% to 5% in Western countries,[6] and a 1.6% lifetime prevalence of AAS use among sampled adolescents and young adults in a Canadian cohort.[7]

Substances obtained through underground sources are notoriously variable in quality and purity and may be contaminated with other unlabelled substances, or in some cases, may not contain the intended product at all.[2] In the HAARLEM study, only 47% of sampled AAS products contained the substance listed on the product label; additionally, 68% of samples contained undeclared AAS, and 49% contained a higher number of AAS compounds than indicated.[8]

Pharmacology/Patterns of Use

AAS are not one specific compound, rather; they encompass a heterogeneous group of substances that share structural similarities to testosterone. These substances can be broadly divided into 17-beta-ester derivatives, 17α-alkylated derivatives, and 19-nortestosterone derivatives[4,9] (see **Table 1**). Differences in their effects on body tissues arise in part based on whether they undergo further conversion by aromatization or 5-alpha reduction.[10] AAS are frequently used in combination with other hormonal or non-hormonal substances, and are administered over variable timeframes, often in fluctuating doses throughout their cycles of use.[1] These multiple variables complicate efforts to attribute adverse events to any single androgenic agent.[1]

Specific terminology is employed to describe patterns of AAS use. “Cycles” refer to discrete periods during which AAS are used typically followed by a period of discontinuation until a new cycle begins. “Stacking” refers to the concurrent use of additional steroidal or non-steroidal agents on top of a testosterone ester “core”[8] regimen (see **Table 2**). “Pyramiding” involves the stepwise escalation of AAS doses to a peak level, which is then tapered back down.[4] Another commonly used regimen is “blasting and cruising,” in which AAS doses are increased and then tapered back down to a lower, but still supratherapeutic, maintenance dose. Note that this approach does not involve a recovery period during which AAS use is temporarily discontinued.[4]

Among people who use AAS, there is also widespread use of supplements, nutraceuticals, and “testosterone boosters” that have not undergone proper safety or quality testing.[1] Although these products are often perceived as safe and accurately labelled, research shows that many contain doses inconsistent with what is stated on the label or include undeclared ingredients.[11] In one study, undeclared doping substances were identified in 25 of the 66 sports nutrition supplements that were analyzed.[11]

Type of Substance	Examples
Non-steroidal drugs with anabolic effects	IGF-1, GH, GHRH, insulin
Drugs that counteract the effects of AAS	Aromatase inhibitors, SERMs, gonadotropins
Drugs for weight loss, fat reduction, or water loss	Diuretics, L-thyroxine, GLP-1 agonists, beta-2 adrenergic agonists
Fertility preservation drugs	Gonadotropins, clomiphene citrate
Drugs to prevent AAS detection	Diuretics, estrogens, probenecid

Table 2. Substances Often "Stacked" with AAS; *adapted from Pope et al., 2014.*[1]

Abbreviations: AAS: anabolic androgenic steroids; **GLP-1:** glucagon-like peptide-1; **GH:** growth hormone; **GHRH:** growth hormone releasing hormone; **hCG:** human chorionic gonadotropin; **IGF-1:** insulin-like growth factor-1; **LH:** luteinizing hormone; **SERMs:** selective estrogen receptor modulators

Clinical Recognition/Diagnosis

Although the prevalence of AAS use has increased, clinical presentations likely represent only the "tip of the iceberg." Many individuals who use AAS avoid physician involvement for several reasons, such as a perceived lack of medical expertise regarding these substances, a desire to avoid being chastised for their use, and a belief that their use does not pose significant health risks.[3] Improving physician education in this area may therefore improve patient confidence in disclosing their AAS use.

Patients who seek medical attention while using AAS may do so for health monitoring or because of concerns about adverse events.[5] However, presentations are often subtle. Some patients may report concerns of inadequate muscle development despite having a muscular physique, suggesting a subtype of body dysmorphic disorder termed "muscle dysmorphia".[12] Others may report no longer "seeing the same gains" at the gym, or prolonged recovery times following exercise. Physical features that may also suggest AAS use can include testicular atrophy, gynecomastia (or related surgical scars), truncal acne, or truncal striae.[2,6]

Measurement of testosterone, luteinizing hormone (LH) and follicle-stimulating hormone (FSH) levels is commonly used in in the evaluation of AAS use and can produce different laboratory patterns based on the substances that are being used[13] (see **Table 3**). Elevated testosterone levels are not always observed on laboratory testing, as some androgens are not detected by testosterone assays, resulting in paradoxically low testosterone levels despite physical findings that suggest the opposite to be true.[13] LH and FSH levels can be low/suppressed; however, in patients using aromatase inhibitors or clomiphene, levels of LH and FSH may instead be elevated, accompanied by high testosterone levels.[6] Additional laboratory findings that may suggest AAS use include low sex hormone-binding globulin levels, elevated hemoglobin/hematocrit, reduced thyroxine-binding globulin, and low high-density lipoprotein (HDL) levels.[4,6]

Potential Adverse Effects

Adverse effects from AAS use are commonly reported by users. In a Canadian study of 2,774 young men, 75% reported experiencing at least one AAS-related adverse event.[7] Most concerning, however, is the evidence suggesting a threefold increase in mortality among AAS users.[4] The adverse events outlined below represent a selection of potential adverse effects, and are not intended to be an exhaustive list.

Hormone/Substance	Testosterone	LH	FSH
Testosterone	↑	↓	↓
Non-testosterone Androgen	↓	↓	↓
Testosterone Precursor (e.g. androstenedione)	↑	↓	↓
hCG	↑	↓	↓
Aromatase Inhibitor	↑	↑	↑
Clomiphene	↑	↑	↑

Table 3. Hormones/Substances and Their Effects on Testosterone, LH and FSH serum levels; *adapted from Anawalt, 2024.*[13]

Abbreviations: FSH: follicle-stimulating hormone; **hCG:** human chorionic gonadotropin; **LH:** luteinizing hormone

Gynecomastia

Gynecomastia, or breast tissue growth, has been reported in men during both AAS use and in the recovery phases following AAS use, with prevalence rates estimated as high as 52%.[4] It is most commonly attributed to conversion of supraphysiologic androgen doses to estrogens and is therefore more commonly observed with aromatizable substances.[14] In an effort to self-manage the acute phase of breast tissue growth, men often use selective estrogen receptor modulators (SERMs) such as tamoxifen or aromatase inhibitors.[4,10] Others use cabergoline or bromocriptine to treat gynecomastia, under a misguided belief that prolactin elevation is the sole cause of their breast growth.[10] However, once breast growth has progressed to the fibrotic/chronic stage of growth, surgical intervention is often required.[14]

Acne Vulgaris

Acne is a common manifestation of AAS use, with prevalence rates of 50% or higher.[4,8] It results from androgen-dependent sebum production, and suggests AAS use when located in a truncal pattern in adult men.[2] Patients may experience improvement with targeted therapies such as isotretinoin, and a subset of AAS users report incorporating low doses of this medication into their AAS cycles.[10]

Androgenic Alopecia

Androgenic alopecia is another reported adverse effect of AAS use and is often linked to increased production of dihydrotestosterone, via the 5-alpha reductase pathway.[10] In the HAARLEM study, 12% of participants reported alopecia by the end of their AAS cycles. To counteract this effect, some users add 5-alpha reductase inhibitors, such as finasteride and dutasteride, to their AAS cycles.[14] However, the effectiveness of these medications on high dose androgen users remains unclear, especially when substances are used that cannot undergo 5-alpha reduction.[10]

Hepatotoxicity

Hepatotoxicity, contrary to popular belief, is not a common complication associated with standard testosterone replacement therapy. The risk, however, is well-recognized with 17α-alkylated oral androgens and selective androgen receptor modulators.[2] Hepatic injury associated with 17α-alkylated androgens is thought to result from their increased oral bioavailability and diminished hepatic degradation.[4] These agents can cause elevations in aspartate aminotransferase, alanine aminotransferase, lactate dehydrogenase and gamma-glutamyl transferase, and can present with jaundice or pruritus.[10] In rare cases, their use has been linked to serious hepatic complications, including peliosis hepatis, hepatocellular carcinoma, and liver adenomas.[10]

Cardiovascular Effects

AAS use is associated with a range of adverse effects on the cardiovascular system, with a 2-3-fold increase in cardiovascular disease among users.[4] AAS use has been linked to left ventricular hypertrophy, hypertension, dyslipidemia, abnormal cardiac remodelling, fibrosis, prothrombotic states, premature atherosclerosis, and arrhythmias.[4,6,15]

Cumulative androgen exposure–defined as the number of years that a user has been exposed to supraphysiologic doses of androgens and

conceptually akin to pack-years in smokers—has been associated with an increased risk of cardiovascular disease.[5] Clinically, dyslipidemia manifests as elevated low-density lipoprotein (LDL) levels and reduced HDL levels,[6] with more pronounced lipid disturbances observed with 17α-alkylated androgens.[10] Sustained stimulation of androgen receptors (AR) in multiple cardiac tissues is thought to lead to the diverse adverse effects. Left ventricular hypertrophy results from chronic stimulation of AR in cardiac myocytes, while overstimulation of AR in vascular endothelial and smooth muscle cells results in vasoconstriction.[15] Accordingly, the presence of unexplained premature coronary artery disease, left ventricular hypertrophy, or arrhythmias in young patients should prompt physicians to consider possible AAS use as the underlying etiology.[15]

Mental Health

The relationship between mental health and AAS use is complex and difficult to unravel. It remains unclear whether mental health conditions predispose individuals to AAS use, or whether AAS use contributes to mental health conditions. Most likely, this relationship is bidirectional.[4] Muscle dysmorphia, a subtype of body dysmorphic disorder, is associated with a perceived inability to gain muscle or achieve one's desired physique, despite physical evidence to the contrary.[12] This perception may lead individuals to either seek medical evaluation for consideration of testosterone therapy (to treat presumed hypogonadism), or to self-treat with AAS.

AAS use has been implicated in the development of de novo mental health conditions, including mood disorders, anxiety, manic episodes, psychosis, and increased aggression.[1,6] AAS users are also at risk of developing drug dependence and addiction, with studies showing AAS dependence rates of approximately 30%.[1] A Canadian study has shown that one in five adolescents and young adults who used AAS experienced moderate-severe dependence.[17] As a result, androgen dependence is now recognized in the 5th edition of the Diagnostic and Statistical Manual of Mental Disorders.[2] Of greatest concern, however, is the increased risk of suicidal ideation and suicide-related death, both of which occur more often in AAS users compared to non-users.[4]

Infertility

Infertility is one of the most common reasons for AAS users to seek medical attention.[6] Exogenous androgens and estrogens suppress gonadotropin-releasing hormone, thereby reducing production of the gonadotropins, LH, and FSH. This suppression results in decreased production of intratesticular testosterone by Leydig cells, impaired Sertoli cell function, and disruption of spermatogenesis.[9,16] Long-term suppression of spermatogenesis results in testicular atrophy, as two-thirds of testicular volume is comprised of the seminiferous tubules.[4]

Contraception studies show an average time span of 6–24 months for sperm concentrations to normalize after testosterone discontinuation.[16] In AAS users, however, recovery can take over 24 months, owing to higher androgen doses and the concurrent "stacking" of other substances that can further inhibit spermatogenesis.[16] It has therefore been proposed that, in the absence of a recovery in spermatogenesis by 24 months following AAS cessation, initiation of gonadotropin therapy may be reasonable.[14,16]

Management

Although several studies have assessed the prevalence of AAS use and associated adverse events, considerably less research has focused on the management of patients using AAS, or on the associated health concerns that can arise following discontinuation of these substances.[17]

Approaches to caring for patients who use AAS vary widely, with some clinicians declining to engage with such patients altogether, while others advocate for a harm-reduction approach in addition to supported cessation attempts.[5,6,18] While some state that prescribing androgens to people who use AAS "colludes with and perpetuates the androgen use,"[2] others are more accepting of a harm-reduction approach that "acknowledges the reality of continued use and seeks to promote health and patient engagement."[5] **Table 4** illustrates components of a harm-reduction approach to AAS use.[5,18]

At present, there are no established medical guidelines for the management of AAS use or recovery. Consequently, there remains a lack of consensus among physicians. In one endocrine conference survey conducted at an endocrinology conference, 84% of respondents reported engaging in watchful waiting after AAS cessation, while the remaining participants actively

Components of a Harm-reduction Approach to AAS Abuse
Reducing the dose of AAS and associated drugs/supplements
Reducing the absolute number of substances overall
Preferential use of AAS with lower known toxicity (e.g., avoidance of 17α-alkylated androgens)
Shortening the duration of cycles, and discouraging "blast and cruise" cycles that eliminate drug-free intervals
Avoidance of needle sharing
Ensuring that cardiovascular risk factors are being monitored/managed
Ongoing encouragement/assistance with AAS cessation
Offering psychiatric/psychologic support to manage withdrawal symptoms and reduce relapse risk

Table 4. Components of a harm-reduction approach to AAS abuse; *adapted from Smit et al., 2026*[5]*; Bonnecaze et al., 2021.*[18]

Abbreviations: AAS: anabolic androgenic steroids

prescribed hormonal therapies for symptom management.[19] When asked about their confidence in managing hypogonadism following AAS use, only 20% of respondents stated that they felt confident or extremely confident in doing so.[19]

Patients often ask for post-cycle treatment (PCT), which may include use of human chorionic gonadotropin, SERMs and/or aromatase inhibitors.[13] Several recovery algorithms have been proposed, including an algorithm described by Rahnema et al.,[14] and another termed the "Scally Protocol", which is a popular discussion topic on online forums.[4] Although PCT is often requested, there is no high quality medical evidence demonstrating that it either hastens recovery of testicular function or produces better long-term outcomes.[4,10] In fact, participants in the HAARLEM study had similar serum testosterone levels 3 months after AAS cessation regardless of whether PCT was employed.[10]

There is also a lack of consensus regarding which laboratory tests and imaging studies should be completed in current and former AAS users. One review characterized physicians' overall diagnostic evaluations of these patients to be "inconsistent and at times inadequate."[3] A reasonable approach could include measuring total testosterone along with either free or bioavailable testosterone levels (given the suppressive effects of AAS on sex hormone-binding globulin) as well as LH and FSH levels to observe for recovery of the hypothalamic-pituitary-gonadal axis. Additional testing should include hemoglobin and hematocrit, especially if the patient has a history of erythrocytosis associated with their AAS use. In patients who have used 17α-alkylated AAS, assessment of liver enzymes and liver function tests are advised,[5] although the evidence supporting routine hepatic ultrasounds remains uncertain. Some authors also advise evaluation of a lipid profile, electrolytes, calcium, glucose, creatinine, and blood urea nitrogen.[18] If there is suspicion that a patient is employing a "cutting phase" in their AAS cycles, during which they are stacking multiple substances to lose weight, measurement of insulin-like growth factor-1 and thyroid testing should also be considered.[13] Finally, prostate-specific antigen testing should be considered on an individualized basis.[3,18]

Recommendations for cardiac monitoring vary widely across studies, with one author suggesting echocardiograms be ordered on an individualized basis.[10]

Many AAS users experience difficulty with prolonged cessation of AAS. One study reported unsuccessful cessation attempts in 60% of respondents.[3] This highlights the need for a multidisciplinary approach that includes addiction services and behavioural health specialists to support recovery and reduce the risk of relapse.[3]

Conclusions/Future Goals

Despite recent progress, this field remains fragmented by barriers to both knowledge and trust. These challenges range from clinical discomfort and a lack of public health recognition to the more profound erosion of the physician-patient relationship. Without mutual trust, patients are less likely to disclose AAS use, making it difficult to establish the shared goals that are central to an effective therapeutic partnership.

Addressing the growing concerns surrounding the use of AAS will require a coordinated strategy that includes further research, improved education for both physicians and the public, and the development of public health policies aimed at mitigating harm and supporting patient care.

Public education campaigns should be directed toward populations at highest risk for AAS use, alongside more stringent restrictions on online sales and improved enforcement and quality control within the supplement industry.

There is also a need for more effective strategies to address the role of internet-based misinformation and the growing influence of large language model artificial intelligence programs in this field. Further research is needed to better understand the links between social media and body image issues in men, including the development of body dysmorphia.

Research that focuses on the management of AAS use and on effective strategies to support patients during their recovery is essential. Equally important is recognition of the need for close collaboration between physicians and mental health care providers throughout this process. Improving physician education about AAS use and addressing physician discomfort in this area will improve the likelihood of patients being willing to disclose use, thereby leading to better outcomes in the long-term.

Physicians need to be educated and willing to engage with patients navigating AAS use and recovery. Although these discussions may be uncomfortable, AAS use remains a growing public health concern that is unlikely to resolve in the immediate future and requires thoughtful, sustained clinical engagement.

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