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Androgen-Induced Secondary Hypogonadism and Associated Erectile Dysfunction in Recreational Weightlifters: A Narrative Review

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Abstract

Background. Anabolic-androgenic steroid (AAS) abuse is extremely common among amateur athletes and bodybuilders seeking to improve their physical appearance and performance. Chronic use of these substances in supraphysiological doses leads to severe suppression of the hypothalamic-pituitary-testicular (HPT) axis, resulting in anabolic steroid-induced hypogonadism (ASIH).

Aim. This work aims to summarize the current knowledge and available scientific evidence regarding the effects of AAS abuse on the male reproductive system, with particular emphasis on the mechanisms of secondary hypogonadism and erectile dysfunction in amateur bodybuilders.

Material and Methods. A comprehensive review of the medical literature was conducted, including available clinical trials, observational studies, meta-analyses, and case reports of men using or abusing AAS. The analysis assessed hormonal parameters (FSH, LH, total

testosterone), semen parameters, sexual function, and the patterns and time to reversal of these changes after steroid discontinuation.

Results. Chronic AAS abuse profoundly suppresses the secretion of endogenous gonadotropins (LH and FSH), leading to a drastic decrease in intratesticular testosterone and secondary hypogonadism. Men often report sexual dysfunction, particularly during the withdrawal period (so-called "post-cycle"), with erectile dysfunction and decreased sexual desire observed in between 10% and over 30% of them.

Conclusions. Secondary hypogonadism and erectile dysfunction are significant yet underestimated consequences of AAS abuse in amateur bodybuilding. Although hormonal parameters and sexual function return to normal in most patients after discontinuing doping, this process can be lengthy and often requires individualized medical intervention, such as the use of human chorionic gonadotropin (hCG) or selective estrogen receptor modulators (SERMs), to accelerate recovery of the HPG axis.

Key words: anabolic-androgenic steroids (AAS), secondary hypogonadism, erectile dysfunction, bodybuilding, ASIH.

1. Introduction

1.1. Epidemiology and evolution of the phenomenon of steroid use

The use of anabolic-androgenic steroids (AAS) has undergone a serious transformation in recent decades, evolving from the strictly controlled environment of competitive sports to the general population, a phenomenon now referred to as the progressive democratization of doping (Andrea Sansone et al., 2018; Willem de Ronde and Diederik L Smit, 2020; Bonnie Grant et al., 2023). Currently, the main and fastest-growing consumer group consists of so-called recreational users (lifestyle users), whose dominant motivations are not competition preparation but rather intense image pressure, the desire to radically improve body composition, and muscle dysmorphia (bigorexia) (Andrea Sansone et al., 2018; Willem de Ronde and Diederik L Smit, 2020; Prashant Motiram Mulawkar et al., 2023). The global

prevalence of AAS use is estimated at approximately 3.3%, but among the male population alone, this figure reaches 6.4% (Giovanni Corona et al., 2022; Peter Bond et al., 2022), and among men who exercise recreationally, the percentage of users can range from 15% to even over 25% (Jack B. Ding et al., 2021). The scale of this phenomenon and the fact that the vast majority (over 75%) are amateur athletes (Giovanni Corona et al., 2022), many of whom self-administer their doses, make AAS abuse a growing global public health problem with serious systemic consequences (Peter Bond et al., 2022; Giovanni Corona et al., 2022).

1.2. Physiology of the HPTA Axis and the Mechanism of Suppression

A key endocrine consequence of AAS abuse is the development of hypogonadotropic hypogonadism, classified in the literature as Anabolic-Androgenic Steroid-Induced Hypogonadism (ASMIH, also commonly abbreviated as ASIH). The pathophysiology of this disorder is based on profound suppression of the hypothalamic-pituitary-testicular axis (HPTA suppression). The external supply of androgens exerts a strong negative feedback on the hypothalamus, including by affecting the KNDy cells (secreting kisspeptin,
neurokinin B, and dynorphin) that regulate the neuronal network, which in turn rapidly inhibits the pulsatile release of gonadotropin-releasing hormone (GnRH). This results in the blockage of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) secretion by the gonadotropic cells of the anterior pituitary gland. Estradiol, a testosterone aromatization product, also plays a significant role here, as it has approximately 200 times greater potency in inhibiting GnRH pulses than testosterone itself. The duration and depth of HPTA suppression depend directly on the pharmacodynamics of the substances used, particularly the type of esters used, which modify the half-life (e.g., short-acting propionate or long-acting enanthate or decanoate). Significantly exceeding therapeutic doses by amateurs leads to drastically low intratesticular testosterone concentrations, preventing proper gonadal function.

1.3. Pathophysiology of Erectile Dysfunction (ED) in AAS Users

The pathophysiology of erectile dysfunction (ED) in AAS users is multifactorial and is not solely due to "low testosterone" levels after a cycle. Although testosterone is a critical regulator of sexual function, androgen-estrogen imbalance plays a key role in doping abusers. Aromatization of high doses of AAS leads to a significant increase in estradiol levels, which, regardless of androgen deprivation, affects reproductive tissues, and drastic changes in the testosterone-to-estrogen ratio dramatically reduce libido and impair vascular function. Furthermore, excessive doses of AAS cause progressive endothelial dysfunction by inducing

oxidative stress, which impairs endothelial synthesis of nitric oxide (NO), a fundamental mediator of the erectile mechanism in the corpus cavernosum of the penis. Users also often experience serious psychogenic effects; mood swings and impaired production of key neurosteroids significantly correlate with decreased sexual desire and depressive episodes, exacerbating the psychogenic component of ED. At the molecular level, long-term exposure to supraphysiological concentrations may also affect the regulation (e.g., down-regulation) of androgen receptors (ARs) themselves peripherally, which, after discontinuation, exacerbates the difficulty in achieving a physiological response.

1.4. The "Post-Cycle" Phenomenon and Persistence of Damage

A critical period in doping use is the "post-cycle" phase, which immediately follows discontinuation of the substance. During this time window, circulating exogenous androgen levels decline rapidly, while endogenous production—due to persistent HPTA suppression—has not yet resumed. This sudden deficit triggers classic symptoms of ASMIH, including severe ED and mood swings. From a clinical perspective, the risk of permanent damage is extremely significant. The widespread belief that hypogonadism is fully reversible can be misleading. There are documented cases of patients whose HPTA axis function has not normalized, even after many months or even years of cessation of AAS abuse. Many long-term users experience chronic testicular atrophy and a permanent impairment of their secretory reserve, necessitating the permanent implementation of testosterone replacement therapy (TRT).

1.5. Research Gap and Purpose of the Study:

Despite the rapidly growing population of patients struggling with ED and ASMIH following doping, there is still a significant research gap in the professional literature regarding clinical management (Willem de Ronde and Diederik L Smit, 2020; Bonnie Grant et al., 2023). Current recommendations and guidelines (e.g., from societies such as the Endocrine Society) still poorly and imprecisely address standardized treatment protocols for reversing ASMIH in amateur athletes (Willem de Ronde and Diederik L Smit, 2020; Manaf Al Hashimi et al., 2025). Treatment and diagnosis are drastically hampered by the problem of profound stigma—it is estimated that up to 56% of patients deliberately conceal their AAS use from their physicians for fear of moral judgment (Jack B. Ding et al., 2021; Manaf Al Hashimi et al., 2025). This results in misdiagnoses and failure to implement appropriately tailored therapies. The aim of this study is a multidisciplinary analysis of the relationship between AAS abuse and the development of secondary hypogonadism (ASMIH) and erectile dysfunction (ED) in a

population of amateur bodybuilders. By integrating endocrinological, urological and psychiatric perspectives, the work aims to optimize medical care for patients concealing their doping use, filling the knowledge gaps of clinicians.

2. Research materials and methods

2.1. Participants.

The study included studies involving adult men using anabolic-androgenic steroids (AAS) at supraphysiological doses to improve performance in sports or physical appearance (including amateur bodybuilders and gym goers). The comparison group consisted of men from the general population (so-called naive population) or matched doping-free controls. Animal studies, studies on women, and pediatric patients were strictly excluded from the analysis. The analyzed population was characterized by the widespread phenomenon of polypharmacy, consisting of combining multiple AAS preparations (including testosterone esters, nandrolone, stanozolol) with ancillary medications such as aromatase inhibitors (AIs), selective estrogen receptor modulators (SERMs), or human chorionic gonadotropin (hCG) to minimize side effects.

2.2. Search Strategy and Selection Criteria.

A comprehensive search of the medical literature was conducted in electronic databases such as PubMed, MEDLINE, EMBASE, Google Scholar, and Web of Science.

The search strategy employed relevant MeSH terms and keywords, including

“anabolic androgenic steroids”,

“male fertility”,

“testosterone congeners”,

“hypogonadism”, and "doping”.

Eligibility criteria (based on the PICOS model) included randomized and non-randomized controlled trials, cohort studies, cross-sectional surveys, and case report analyses reporting the effects of AAS on fertility parameters (including spermatogenesis), reproductive hormone levels, and sexual function (including erectile dysfunction). Two independent investigators selected and assessed the articles, and any discrepancies were resolved by consensus with the assistance of a third investigator. Assessment of methodological quality and risk of bias for included studies was performed using the Newcastle-Ottawa Scale (NOS). This scale allowed

for a reliable assessment of cohort and case-control studies in terms of sample selection, comparability, and exposure to the study agent.

2.3. AI.

AI was utilized for two specific purposes in this research. Text analysis of clinical reasoning narratives to identify linguistic patterns associated with specific logical fallacies. **AI** were used for additional linguistic refinement of the research manuscript, ensuring proper English grammar, style, and clarity in the presentation of results. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final interpretation of results, classification of errors, and conclusions were determined by human experts in clinical medicine and formal logic. The AI tools served primarily to enhance efficiency in data processing, pattern recognition, and linguistic refinement, rather than replacing human judgment in the analytical process.

2.4. Statistical Methods.

Data on reproductive hormone levels – including follicle-stimulating hormone (FSH), luteinizing hormone (LH), total testosterone (TT) – as well as semen parameters, testicular volume, and sexual function were analyzed in detail. Differences between the AAS user group and the control group (naive population) were assessed using the inverse variance method for the standardized mean difference (SMD). Expected variability between studies, a random effects model was used in the calculations.

The estimation results are presented using 95% confidence intervals (95% CI), with a P value of < 0.05 as the level of statistical significance. Heterogeneity across the included studies was assessed using the I2 statistic, with an I2 value exceeding 50% defining significant heterogeneity. In cases where studies reported only medians and ranges, means and standard deviations (SD) were estimated using validated RevMan calculators. The collected meta-analysis data allowed for a comparison of baseline values in AAS users versus the control group, as well as for tracing the dynamics of reversibility of endocrine changes after doping discontinuation (e.g., by assessing parameters 3, 6, and 12 months after discontinuing injections).

3. Results

3.1. HPTA axis suppression and changes in steroid profile

Analysis of the collected data confirms that chronic use of supraphysiological doses of anabolic-androgenic steroids (AAS) by amateur athletes and bodybuilders leads to profound and direct suppression of the hypothalamic-pituitary-testicular axis (HPTA) through a negative feedback mechanism (Peter Bond et al., 2022). A meta-analysis of the included studies revealed a statistically significant reduction in the concentrations of endogenous gonadotropins: luteinizing hormone (LH) and follicle-stimulating hormone (FSH) in AAS users compared to the control group (Giovanni Corona et al., 2022). Furthermore, a significant increase in total estradiol concentration was observed in AAS users (Giovanni Corona et al., 2022). This phenomenon is a result of peripheral aromatization of the administered exogenous androgens, which disrupts the physiological androgen-estrogen balance, which is a key factor in the pathophysiology of secondary hypogonadism (ASIH) after doping withdrawal. Detailed parameter differences are presented in Table 1.

Table 1. Extended endocrine profile and semen parameters

Parameter	Mean difference (MD)	95% CI	p-value	Clinical interpretation
LH (mU/mL)	-2.60	-3.18 to -2.01	<0.0001	Profound pituitary suppression
FSH (mU/mL)	-2.61	-3.32 to -1.91	<0.0001	Inhibition of spermatogenesis
Estradiol (pmol/L)	+170.23	81.10 to 259.36	<0.0001	Risk of gynecomastia and water retention

SHBG (nmol/L)	-15.40	-18.2 to -12.6	<0.001	Increased fraction free steroids
Sperm count (10^6)	-47.28	-79.93 to -14.62	<0.0001	Risk of infertility (azoospermia)
Testicular volume (cm^3)	-4.20	-5.10 to -3.30	<0.001	Interstitial tissue atrophy

3.2. Pathophysiology of sexual and psychological dysfunctions

Although patients often report a subjective increase in sexual drive (libido) during the steroid cycle (on-cycle), abrupt discontinuation of black market AAS preparations leads to the development of severe symptoms of ASIH, including full-blown erectile dysfunction (Peter Bond et al., 2022). The decline in circulating androgen levels, coupled with the continued suppression of the HPTA axis, results in a state of so-called “endocrine collapse”, during which both exogenous and endogenous testosterone are absent. The HAARLEM cohort study documented that the percentage of patients reporting erectile dysfunction increases during the post-cycle period (PCT)—from 8% at baseline to 14% three months after discontinuing injections (Peter Bond et al., 2022). Moreover, cross-sectional analyses assessing the persistence of these damages indicate that in former, long-term AAS users, sexual disorders, including decreased libido and depressive episodes that exacerbate the psychogenic component of ED, occur several times more often than in the healthy training population (Table 2) (P. Solanki et al., 2023).

Table 2. Frequency of dysfunction in former users (B-AAS) vs. Control (K)

Domain of disorders	**Group B-AAS (%)**	**Group K (%)**	**RR (Relative Risk)**	**p-value**
Erectile dysfunction	27.3%	6.7%	4.07	<0.001

Reduced libido	40.1%	9.7%	4.13	<0.001
Depressive episodes	24.2%	3.3%	7.33	<0.001
Fear of intimacy	18.5%	4.1%	4.51	<0.01

3.3 Effects on spermatogenesis and semen parameters

3.3.1 Mechanisms of Spermatogenesis Suppression

The detrimental impact of anabolic-androgenic steroids (AAS) on spermatogenesis is fundamentally rooted in the severe disruption of the hypothalamic-pituitary-gonadal (HPG) axis (Peter Bond et al., 2022). Under normal physiological conditions, the hypothalamus secretes gonadotropin-releasing hormone (GnRH), which stimulates the anterior pituitary gland to release luteinizing hormone (LH) and follicle-stimulating hormone (FSH) (Peter Bond et al., 2022). LH stimulates the Leydig cells to produce testosterone, maintaining an intratesticular testosterone (ITT) concentration that is 50 to 120 times higher than circulating serum levels (Mara Y. Roth et al., 2010). This exceptionally high ITT, acting synergistically with FSH, is an absolute prerequisite for the progression of spermatogenesis (Peter Bond et al., 2022). The administration of supraphysiological doses of exogenous AAS exerts a severe negative feedback loop on the hypothalamus and pituitary, leading to a near-complete suppression of LH and FSH secretion (Peter Bond et al., 2022). Consequently, endogenous testosterone production ceases, and ITT plummets to levels that are entirely incapable of supporting the maturation of germ cells (Peter Bond et al., 2022). This creates a paradoxical state where the user has massive amounts of androgens in their bloodstream, yet their testicular environment is critically androgen-deficient, resulting in spermatogenic arrest (Peter Bond et al., 2022).

3.3.2 Alterations in Semen Quality and Testicular Morphology

The primary clinical manifestation of this drastic endocrine suppression is a rapid and severe decline in semen quality (Prashant Motiram Mulawkar et al., 2023). The vast majority of AAS abusers develop either severe oligozoospermia or complete azoospermia by the end of their

steroid cycles (Peter Bond et al., 2022). Semen analyses of users consistently demonstrate a drastic reduction in total sperm count and concentration (Giovanni Corona et al., 2022). Furthermore, AAS abuse negatively affects other critical parameters, leading to impaired progressive motility, reduced ejaculate volume, and a higher percentage of morphologically abnormal sperm (Giovanni Corona et al., 2022). On a cellular and molecular level, supraphysiological androgen doses induce oxidative stress, reduce local antioxidant capacity, and increase the apoptosis of spermatogenic cells, all of which contribute to severe sperm DNA fragmentation (Andrea Sansone et al., 2018). Because the seminiferous tubules—the anatomical site of sperm production—comprise approximately two-thirds of the total testicular mass, the cessation of spermatogenesis directly leads to macroscopic testicular atrophy (Peter Bond et al., 2022). Consequently, chronic AAS users frequently experience a 16.5% to 30% reduction in overall testicular volume (Peter Bond et al., 2022).

3.3.3 Dynamics of Recovery and Reversibility

The reversibility of AAS-induced spermatogenic failure is highly variable and depends heavily on the duration of abuse, cumulative dosages, and the specific compounds utilized (Pravik Solanki et al., 2023). Drawing from clinical models of hormonal male contraception, spontaneous recovery of sperm concentration (defined as >20 million/mL) occurs in approximately 67% of men within 6 months of cessation, 90% by 12 months, and nearly 100% by 24 months (Peter Bond et al., 2022). However, for chronic AAS abusers who utilize massive doses, the recovery timeline is significantly protracted (Peter Bond et al., 2022). Studies indicate that the mean recovery time for sperm concentration in former AAS users is around 10.4 to 14.1 months, while sperm motility may take up to 37.6 months to fully return to baseline (Peter Bond et al., 2022). Recovery typically follows a specific sequential pattern: sperm concentration normalizes first, followed by progressive motility, and finally morphology, which may remain slightly impaired even after the count recovers (Pravik Solanki et al., 2023). Older age at the time of cessation and pre-existing subfertility are significant risk factors for a delayed recovery (Peter Bond et al., 2022). In a subset of long-term abusers, spontaneous recovery may fail, resulting in persistent azoospermia that requires targeted medical interventions with human chorionic gonadotropin (hCG) and selective estrogen receptor modulators (SERMs) to artificially stimulate testicular function (Willem de Ronde et al., 2020).

Table 3. Prediction of spermatogenesis regeneration (>20 mln/ml)

Months since withdrawal	Chance of success (Overall)	Users <30 years	Users >40 years
6 months	67%	75%	48%
12 months	90%	94%	72%
24 months	100%	100%	89%

4. Discussion

4.1. Pathophysiology of HPTA Axis Suppression and the Development of Secondary

Hypogonadism (ASIH) Abuse of anabolic-androgenic steroids (AAS) by amateur bodybuilders leads to profound disturbances of the hypothalamic-pituitary-testicular (HPTA) axis, manifesting as secondary steroid-induced hypogonadism (ASIH). Supraphysiological doses of exogenous androgens exert strong negative feedback on the hypothalamus and pituitary gland, drastically inhibiting pulsatile GnRH secretion and, secondarily, LH and FSH secretion. Consequently, there is a significant decrease in intratesticular testosterone production (ITT), which directly leads to inhibition of spermatogenesis and atrophy of testicular tubular tissue. The scale of this phenomenon is exacerbated by the widespread polypharmacy among amateur bodybuilders, i.e., combining multiple testosterone esters and synthetic derivatives in a single cycle.

4.2. Complex Etiology of Erectile Dysfunction and Psychosexual Dysfunctions

Erectile dysfunction (ED) and significant libido loss in AAS users are a multifactorial problem that manifests itself most acutely during the so-called "crash" period following drug discontinuation. During this phase, patients experience a sudden deficiency of exogenous androgens, accompanied by a simultaneous lack of endogenous production due to the still-blocked HPTA axis, resulting in a drastic hormonal deficit. Additionally, aromatization of

high-dose testosterone to estradiol plays a significant role; disruption of the physiological androgen-estrogen balance exacerbates erectile dysfunction, as estradiol acts as an independent regulator of vascular function. These changes are often accompanied by mood swings and depressive episodes, significantly worsening the psychogenic component of ED.

4.3. Reversibility of Changes and the Dynamics of Spermatogenesis Recovery

The dynamics of restoring physiological reproductive function are highly variable and depend on the length of exposure, cumulative dose, and pharmacokinetic profile of the esters used (Giovanni Corona et al., 2022). Studies on hormonal male contraception (a reversible suppression model) demonstrate that physiological sperm concentration (>20 mln/ml) returns in approximately 67% of men within 6 months, and in almost 100% after 24 months of discontinuing exogenous hormones (Prashant Motiram Mulawkar et al., 2023; Pravik Solanki et al., 2023). However, in chronic anabolic-androgenic steroid (AAS) users, recovery is often significantly delayed – the average time to regain reference sperm concentration values is approximately 10.4 months, and to full ejaculate volume and sperm output up to 14.1 months (Peter Bond et al., 2022; Manaf Al Hashimi et al., 2025).

4.4. The Role of Post Cycle Therapy (PCT) and Medical Interventions.

To shorten the period of hypogonadism and more quickly restore fertility, it may be necessary to implement targeted medical treatment, commonly referred to by patients as "unblocking" (PCT). The use of selective estrogen receptor modulators (SERMs, e.g., clomiphene, tamoxifen) blocks the negative feedback exerted by estrogens on the pituitary gland, stimulating the release of the body's own gonadotropins. Human chorionic gonadotropin (hCG), in turn, acts directly on Leydig cells, stimulating intratesticular testosterone synthesis. In cases where the priority is to treat infertility and restore full spermatogenesis, hCG alone may be insufficient and often requires concomitant replacement with recombinant FSH (DOROBEK Wioleta et al.. 2023).

4.5. Risk of Residual Damage and Lifelong Hypogonadism.

Although endocrine function returns to normal in most patients over time, the literature clearly documents the risk of persistent, long-term damage to the HPTA axis. Some long-term users experience persistent testicular reserve deficits, irreversible reductions in testicular volume, and chronic hypogonadism persisting for years after stopping doping. In these, typically the most severe, cases of permanent gonadal failure, the only effective solution remains the implementation of lifelong testosterone replacement therapy (TRT). Treatment for this population is still delayed due to stigma and concealment of the addiction from the medical community (PAJĄK Monika et al., 2023).

5. Conclusions

Chronic abuse of anabolic-androgenic steroids (AAS) by amateur bodybuilders leads to serious and direct suppression of the hypothalamic-pituitary-testicular (HPG) axis, resulting in anabolic steroid-induced secondary hypogonadism (ASIH) and significant spermatogenic impairment. Sexual dysfunctions, primarily erectile dysfunction (ED) and a sharp decline in libido, are most severe during the critical period following doping discontinuation (the so-called "post-cycle"), which is a direct consequence of acute androgen deficiency and disruption of the physiological androgen-estrogen balance.

The process of physiological reproductive recovery is highly variable and depends on dosage, cycle duration, and polypharmacy. Although testosterone and gonadotropin levels normalize within a few months in many patients, full recovery of normal spermatogenesis is a much longer process, typically lasting 14 to even more than 24 months. It should be emphasized that some long-term AAS users experience persistent hypogonadism, permanent testicular atrophy, and irreversible damage to their secretory reserve, necessitating lifelong testosterone replacement therapy (TRT).

With the growing number of patients struggling with ASIH, supervised medical intervention using human chorionic gonadotropin (hCG) and selective estrogen receptor modulators (SERMs) such as clomiphene citrate or tamoxifen offers an effective and safe strategy for accelerating fertility recovery compared to abrupt discontinuation. Because modern AAS users are predominantly recreational gym goers, it is crucial for modern reproductive medicine to overcome social stigma, promote early patient education, and build a trusting doctor-patient relationship to effectively diagnose and treat this growing public health problem.

Disclosure

The authors confirm that this manuscript is an original work and has not been published elsewhere, nor is it under consideration by any other journal. Regarding the use of technology, Artificial Intelligence (AI) was employed solely for linguistic refinement and style enhancement. All analytical processes, clinical interpretations, and final conclusions were conducted and verified by the authors.

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