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PSYCHIATRIC EFFECTS OF ANABOLIC-ANDROGENIC STEROIDS (AAS) ON ATHLETES - A COMPREHENSIVE LITERATURE REVIEW

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# PSYCHIATRIC EFFECTS OF ANABOLIC-ANDROGENIC STEROIDS (AAS) ON ATHLETES - A COMPREHENSIVE LITERATURE REVIEW

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## ABSTRACT

**Background and Objectives:** Anabolic-androgenic steroids (AAS) are synthetic derivatives of testosterone widely misused by athletes to enhance physical performance and appearance. Emerging evidence indicates substantial psychiatric morbidity associated with AAS use. This comprehensive literature review synthesizes empirical evidence from peer-reviewed research published between 2015 and 2025 examining psychiatric effects of AAS among athletic populations.

**Methods:** A systematic search of electronic databases (PubMed, Scopus, Web of Science, Google Scholar) was conducted using keywords related to AAS, psychiatric effects, mental health, and athletes. Studies published in English or Polish between January 2015 and December 2025 were included. Data extraction focused on prevalence, clinical manifestations, neurobiological mechanisms, and treatment implications.

**Results:** Recent epidemiological data indicate AAS prevalence of approximately 10–20% among elite athletes and over 25% among weightlifters, with rates exceeding 36% in some bodybuilding populations. Significant associations were identified between AAS use and depression (Beck Depression Inventory scores significantly elevated,  $p < 0.001$ ), anxiety disorders (Beck Anxiety Inventory scores  $p < 0.001$ ), aggression and violence (meta-analytic evidence of increased interpersonal violence risk), mania and psychosis (dose-dependent), muscle dysmorphia (prevalence 58% in some AAS-using samples vs. 2–6% general population), cognitive deficits (visuospatial memory  $p = 0.004$ , executive function impairment), and dependence syndromes (affecting approximately 30% of users). Neuroimaging studies demonstrated structural brain changes including cortical thinning, altered amygdala volume, and reduced frontal connectivity. Withdrawal syndrome characterized by prolonged hypogonadism and severe depression may persist for months to years. Risk factors include body image disorders ( $r = 0.24$  with obsessive-compulsive traits), competitive pressure, and polysubstance use.

**Conclusions:** AAS use among athletes is associated with substantial psychiatric morbidity affecting mood, cognition, behavior, and brain structure. Evidence from 2015–2025 employing advanced methodologies (neuroimaging, network analysis, large-scale epidemiology) has substantially enhanced understanding. Integrated treatment approaches targeting substance use, psychiatric symptoms, and underlying body image pathology are essential. Future research priorities include longitudinal studies, gender-specific investigations, neurobiological mechanism elucidation, and evidence-based treatment trials.

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## KEYWORDS

Anabolic-Androgenic Steroids, Athletes, Psychiatric Effects, Depression, Anxiety, Muscle Dysmorphia

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## **Introduction**

### **Background**

Anabolic-androgenic steroids (AAS) are synthetic derivatives of testosterone possessing both anabolic (muscle-building) and androgenic (masculinizing) properties. Originally developed for medical purposes including treatment of hypogonadism, delayed puberty, and muscle-wasting conditions, AAS have become widely misused by athletes and bodybuilders seeking enhanced physical performance and appearance[1][2]. The global landscape of AAS use has evolved substantially over the past decade, with increasing prevalence across diverse athletic populations and growing recognition of associated health risks.

Recent epidemiological data reveal concerning prevalence rates. Among elite athletes, AAS usage estimates range from 10% to 20%, increasing to over 25% among weightlifters[2]. In specific regions such as Iran, prevalence exceeds 36% in athletic populations, with bodybuilders demonstrating particularly elevated rates (36.3%) compared to other athletes (30.9%)[3]. Among gym-goers and recreational bodybuilders, prevalence reaches 24.3% and 43% respectively[4]. In this review, the term “athletic populations” therefore encompasses not only elite and competitive athletes but also recreational bodybuilders and regular gym-goers who engage in structured resistance training[4].

### **Psychiatric Consequences**

The misuse of supraphysiological doses of AAS carries significant health risks extending beyond well-documented cardiovascular, hepatic, and endocrine complications, increasingly encompassing mood and anxiety disorders, aggressive and violent behavior, psychotic symptoms, cognitive impairment, and structural and functional brain changes.

Emerging evidence from 2015–2025 has increasingly illuminated these profound psychiatric consequences, with studies documenting associations between AAS use and mood disorders, psychotic symptoms, aggressive behavior, cognitive deficits, and substance dependence[1][5][6]. These effects represent a critical public health concern, particularly given the high prevalence of AAS use among young athletes during vulnerable neurodevelopmental periods.

The psychiatric sequelae manifest across multiple domains: affective disturbances (depression, anxiety, mood lability), behavioral changes (aggression, violence, risk-taking), psychotic phenomena (mania, paranoia, hallucinations), cognitive deficits (memory impairment, executive dysfunction), and addiction syndromes characterized by withdrawal symptoms and compulsive use patterns[7]. Recent neuroimaging studies have revealed structural and functional brain alterations in long-term users, providing neurobiological substrates for observed psychiatric symptoms[8]. Withdrawal from AAS frequently precipitates severe complications including major depression and hypogonadal symptoms persisting for months, potentially driving relapse and dependence[9].

### **Sociocultural Context**

Despite growing recognition of risks, AAS use continues to increase globally, driven by sociocultural pressures emphasizing muscularity, competitive advantages in sports, and proliferation of image-focused social media[10]. The phenomenon of “bigorexia” or muscle dysmorphia—a body dysmorphic disorder characterized by pathological preoccupation with muscularity—has emerged as both driver and consequence of AAS misuse[11][12]. The intersection of AAS use with other psychiatric conditions, particularly obsessive-compulsive spectrum disorders and eating pathology, complicates clinical presentation and treatment approaches[13].

### **Rationale and Objectives**

Despite accumulating evidence, comprehensive reviews synthesizing research from 2015-2025 on psychiatric effects of AAS in athletes remain limited. Recent methodological advances including network analysis, neuroimaging techniques, and large-scale epidemiological studies have generated novel insights warranting systematic synthesis. This literature review aims to:

1. Comprehensively examine psychiatric effects of AAS use among athletes based on research published 2015-2025
2. Synthesize evidence regarding prevalence, risk factors, and clinical manifestations
3. Evaluate neurobiological mechanisms underlying psychiatric symptoms
4. Identify gaps in current knowledge and directions for future research
5. Discuss implications for prevention, screening, and treatment in athletic populations

## Methodology

### Search Strategy

A comprehensive literature search was conducted across multiple electronic databases including PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar. The search strategy employed keyword combinations: ("anabolic androgenic steroids" OR "AAS" OR "anabolic steroids" OR "performance enhancing drugs") AND ("psychiatric" OR "mental health" OR "depression" OR "anxiety" OR "psychosis" OR "aggression" OR "cognitive" OR "brain" OR "mood disorders") AND ("athletes" OR "bodybuilders" OR "weightlifters" OR "sports"). Additional searches included terms related to specific psychiatric conditions: "muscle dysmorphia," "body dysmorphic disorder," "dependence," "addiction," "withdrawal," and "violence."

### Inclusion and Exclusion Criteria

Studies were included if they: (1) were published between January 1, 2015 and December 31, 2025; (2) were original research articles, systematic reviews, or meta-analyses; (3) examined psychiatric effects, mental health outcomes, or neuropsychological consequences of AAS use; (4) included participants who were athletes, bodybuilders, weightlifters, or physically active individuals; (5) were published in English or Polish; (6) utilized validated psychiatric assessment instruments or standardized diagnostic criteria.

Exclusion criteria included: (1) studies published before 2015; (2) animal or in vitro studies without human relevance; (3) case reports of single individuals; (4) conference abstracts without full-text availability; (5) studies examining only non-psychiatric medical outcomes. Additionally, high-quality narrative reviews were consulted to contextualize the empirical findings but were not treated as primary data sources in the synthesis.

### Data Extraction and Synthesis

From each included study, data were extracted on: study design and sample characteristics (sample size, age, gender, sport type), AAS use patterns (duration, dosage, specific compounds), psychiatric assessment methods, primary outcomes, and key findings. Given heterogeneity of study designs and outcome measures, a narrative synthesis approach was employed, organizing findings by psychiatric domain: mood disorders, behavioral disturbances, psychotic symptoms, body image pathology, cognitive effects, and dependence patterns.

## Results

### Prevalence of AAS Use Among Athletes (2015-2025)

#### *Global and Regional Estimates*

Recent epidemiological data from 2015-2025 indicate substantial AAS use prevalence among athletic populations. Amaral et al. (2022) conducted a comprehensive meta-analysis examining prevalence of AAS users seeking support from physicians[14]. The estimated overall prevalence was 37.12% (95% CI 29.71-44.52%), with considerable variation across populations. The smallest prevalence rate (1.76%; 95% CI 0.61-2.91%) was observed in gym users in Pakistan, while the highest rate (88.76%, 95% CI 77.46-100.06%) was reported in non-specific AAS users in Australia[14].

Among specific populations, Lin et al. (2025) reported that elite athletes demonstrate prevalence of approximately 10-20%, rising to over 25% among weightlifters, with rates exceeding 30% in certain regions[2].

#### *Sport-Specific and Demographic Patterns*

Noori et al. (2020) examined AAS prevalence in the Iranian athletic population[3]. The overall prevalence rate was 36.2% (95% CI 29-43) with significant heterogeneity between studies ( $I^2=99.0\%$ ,  $p<0.001$ ). Prevalence was higher among elite athletes, male athletes, and younger athletes ( $p<0.05$ ). Bodybuilding athletes showed particularly elevated rates (36.3%) compared to other sports (30.9%,  $p<0.001$ )[3].

Al Hamid (2025) reported that among gym-goers, 24.3% reported AAS use, while 43% of recreational bodybuilders reported AAS abuse[4]. Karagun and Altug (2024) examined 25 male AAS-using bodybuilders (mean age  $31.2\pm 8.9$  years) with mean AAS exposure duration of  $2.44\pm 2.04$  years[15].

Gender differences remain striking, with male-to-female ratios exceeding 50:1 in most studies[2]. However, emerging evidence suggests increasing AAS use among female athletes, particularly in physique sports, though research in this population remains limited.

## Depression and Mood Disorders

### *Clinical Evidence from Recent Studies*

Multiple studies from 2015-2025 have documented strong associations between AAS use and depressive symptoms using validated instruments. Karagun and Altug (2024) conducted a cross-sectional study comparing 25 male AAS-using bodybuilders with 25 matched controls[15]. Beck Depression Inventory (BDI) scores were significantly higher in the AAS user group compared to controls ( $6.4 \pm 6.7$  vs.  $0.8 \pm 1.4$ ,  $p < 0.001$ )[15]. In the AAS user group, seven individuals (28%) displayed depressive symptoms: four with mild depression (BDI 10-16, representing 16% of sample) and three with moderate depression (BDI 17-29, representing 12% of sample)[15]. No participants in the control group exhibited depression[15].

Lin et al. (2025) described psychiatric complications in powerlifters using AAS[2]. One case involved a 35-year-old powerlifter using testosterone (600mg/week) and drostanolone (100mg/week) for three months who presented with depressed mood, irritability, low self-esteem, fatigue, and insomnia absent of identifiable psychosocial stressors[2]. Despite AAS discontinuation, mood symptoms did not achieve full remission during three-month follow-up as measured by Beck Depression Inventory[2].

Windfeld-Mathiasen et al. (2022) examined psychiatric morbidity in a large cohort study of men using anabolic steroids[16]. They found significantly elevated rates of major depressive disorder among AAS users compared to non-users, with particular vulnerability during withdrawal periods. Individuals discontinuing AAS demonstrated notable risk for major depressive episodes within initial months after cessation[16].

### *Neurobiological Correlates*

Karagun and Altug (2024) identified significant correlations between depression scores and biochemical markers[15]. Regression analysis revealed that increases in serum creatinine were associated with significant increases in BDI scores ( $p < 0.001$ ), as were increases in estradiol levels ( $p = 0.028$ )[15]. The correlation between creatinine (marker of potential organ damage) and depression suggests physical health complications may contribute to psychological distress[15]. ROC curve analysis demonstrated that creatinine values  $\geq 1.08$  mg/dL could predict moderate-to-severe depression with 85.7% sensitivity and 87.8% specificity (AUC: 0.853,  $p = 0.007$ )[15].

Aromatization of testosterone to estradiol may contribute to mood disturbances, as evidenced by elevated estradiol levels in symptomatic users[2].

## Anxiety Disorders

### *Prevalence and Clinical Presentation*

Anxiety disorders represent another prominent psychiatric complication documented in recent research. Karagun and Altug (2024) demonstrated significantly higher Beck Anxiety Inventory (BAI) scores in AAS users compared to controls ( $5.8 \pm 3.7$  vs.  $1.2 \pm 1.2$ ,  $p < 0.001$ )[15]. While the control group showed no instances of anxiety, seven individuals (28%) in the AAS user group reported mild anxiety symptoms (BAI 8-15)[15]. No participants exhibited moderate or severe anxiety in this sample[15].

Lin et al. (2025) presented a case of a 28-year-old powerlifter who, following a 2-month regimen of multiple AAS compounds (testosterone 600mg/week, drostanolone 200mg/week, trenbolone 100mg/week, nandrolone 200mg/week), reported anxiety, irritability, and reduced libido absent of identifiable psychosocial triggers[2]. Serum analysis revealed elevated estradiol and testosterone levels[2]. Notably, emotional disturbances showed improvement after discontinuation for one week without psychotropic medication, as indicated by Beck Anxiety Inventory scores[2].

### *Biological Mechanisms*

Anxiety symptoms in AAS users correlate with specific biomarkers. Karagun and Altug (2024) identified moderate positive correlations between lactate dehydrogenase (LDH) levels and BAI scores ( $r = 0.495$ ,  $p = 0.012$ ), as well as between creatinine and BAI scores ( $r = 0.595$ ,  $p = 0.002$ )[15]. Linear regression analysis demonstrated that increases in both creatinine ( $p < 0.001$ ) and LDH ( $p = 0.039$ ) were associated with statistically significant increases in anxiety scores[15]. These findings suggest anxiety may be exacerbated by physiological stress markers related to organ damage from supraphysiological AAS doses[15].

AAS may alter affinity or density of serotonin (5-HT) receptors, decreasing serotonergic tone associated with anxiety, aggression, and altered behaviors. The relationship between elevated estradiol (resulting from aromatization) and anxiety symptoms further implicates hormonal mechanisms in pathogenesis[2].

## **Aggression, Violence, and Behavioral Disturbances**

### *Clinical Studies and Case Evidence*

Lin et al. (2025) reported increased aggression in powerlifters using high-dose, multi-compound AAS cycles, with numerous conflicts with family members emerging in the weeks preceding psychiatric assessment. These conflicts were characterized by episodes of explosive anger, irritability, and confrontational behavior, often in the absence of clear external stressors and temporally associated with periods of intensive AAS use[2].

In addition to such clinical case reports, a recent meta-analysis of 14 studies including 16 samples and 137,055 participants found a statistically significant, albeit small, association between nonmedical AAS use and interpersonal violence ( $r=0.21$ , 95% CI 0.15–0.27,  $p<.00001$ ). The authors concluded that AAS users, as a group, show elevated rates of violent behavior, but emphasized that the direction of causality remains unclear and that the relationship is likely embedded in a broader context of personality traits, lifestyle factors, and polysubstance use rather than reflecting a simple, uniform effect of AAS[20].

## **Mania and Psychotic Symptoms**

### *Clinical Presentations*

Manic and psychotic symptoms represent severe psychiatric complications documented in recent literature. Lin et al. (2025) estimated that high-dose AAS users may develop mood disorders, anxiety disorders, and in extreme cases, mania or psychosis[2]. Case descriptions included sleep-wake cycle disruption followed by increasing anxiety and persecutory ideation, progressing to increased aggression and conflicts with family members[2].

### *Dose-Dependent Effects*

Trenbolone has been particularly associated with higher rates of psychiatric symptoms including paranoia and mood lability[2]. The relationship between dose, duration, and psychiatric symptoms appears complex, with individual vulnerability factors (genetic predisposition, concurrent substance use, premorbid psychiatric conditions) influencing risk.

## **Muscle Dysmorphia and Body Image Disorders**

### *Prevalence and Characteristics*

Muscle dysmorphia (MD), termed "bigorexia" or "reverse anorexia," is a body dysmorphic disorder characterized by pathological preoccupation with muscularity and persistent belief that one's body is insufficiently muscular despite normal or exceptional musculature. MD represents both risk factor for and consequence of AAS use, creating bidirectional relationships perpetuating substance use and body image pathology.

Çınaroğlu and Yılmaz (2025) conducted systematic review and meta-analysis of muscle dysmorphia, obsessive-compulsive traits, and anabolic steroid use, examining studies from 2015-2025[17]. Meta-analytic findings revealed significantly higher MD symptoms among AAS users compared to non-users (Cohen's  $d\approx 0.45$ ,  $p<0.001$ )[17]. Odds of MD were markedly higher in steroid-using populations, with odds ratios ranging from 25 to 30[17].

### *Relationship with Obsessive-Compulsive Traits*

MD shares phenomenological features with obsessive-compulsive disorder (OCD), including intrusive thoughts about appearance, compulsive behaviors (mirror checking, excessive exercise), and ritualistic patterns. Çınaroğlu and Yılmaz (2025) found moderate positive correlation between MD symptom severity and obsessive-compulsive traits ( $r\approx 0.24$ , 95% CI: 0.20-0.28,  $p<0.01$ )[17]. AAS users showed higher OCD symptom scores compared to non-users (Cohen's  $d\approx 0.3$ ,  $p<0.001$ )[17].

## **Cognitive Deficits and Neuropsychological Impairment**

### *Evidence from Neuropsychological Studies*

Lin et al. (2025) assessed cognitive functioning using Wechsler Adult Intelligence Scale (WAIS-IV) and Conners Continuous Performance Test (CPT-3) in two AAS-using powerlifters[2]. One subject demonstrated below-average cognitive functioning (FSIQ=87, 95% CI=83-91) along with impulsivity and inattention traits[2]. Another showed above-average cognitive functioning (FSIQ=108, 95% CI=101-109) but also exhibited impulsivity and inattention traits[2]. These findings suggest heterogeneous cognitive effects, with impulsivity and attention deficits being common features even in higher-functioning individuals[2].

### *Domain-Specific Impairments*

Research has identified specific cognitive domains particularly vulnerable to AAS effects. Executive functions including planning, cognitive flexibility, and inhibitory control are frequently affected. Attention and concentration deficits, documented by continuous performance tests, suggest disruption of prefrontal cortical systems[2].

Recent research examined cognitive deficits in relation to AAS dose and duration, finding that higher cumulative doses and longer durations were associated with greater cognitive impairment. The relationship appeared partially reversible, with some cognitive recovery observed following prolonged abstinence, though residual deficits often persisted.

## **Structural and Functional Brain Changes**

### *Neuroimaging Findings*

Advanced neuroimaging techniques have revealed structural brain alterations in long-term AAS users. Bjørnebekk et al. (2017) conducted structural brain imaging of long-term AAS users using magnetic resonance imaging (MRI)[8]. The study compared 82 male weightlifters (41 long-term AAS users with mean lifetime use of 9.1 years, 41 non-using controls)[8]. AAS users showed significantly reduced cortical thickness in several brain regions, particularly in frontal and temporal cortices[8]. Additionally, AAS users demonstrated altered volumes in subcortical structures including amygdala (involved in emotion processing) and hippocampus (critical for memory)[8].

The magnitude of structural changes correlated with duration and dose of AAS use, suggesting dose-dependent neurotoxic effects[8]. Regions showing greatest structural alterations (prefrontal cortex, amygdala) correspond to areas implicated in impulse control, emotion regulation, and aggression—aligning with observed behavioral changes in AAS users[8].

## **Neurotoxicity Mechanisms**

Multiple mechanisms underlie AAS-induced neurotoxicity. Supraphysiological doses induce oxidative stress, mitochondrial dysfunction, and apoptotic processes in neural tissue. AAS overwhelm neuroprotective mechanisms, resulting in cellular damage particularly in regions with high androgen receptor density. Additionally, AAS disrupt neurosteroid production, affecting GABAergic neurotransmission and neuronal excitability.

Zoob Carter et al. (2024) demonstrated associations between neuronal degeneration markers and AAS exposure[19]. Elevated levels of neurofilament light chain (biomarker of axonal damage) were observed in AAS users compared to controls, suggesting ongoing neurodegeneration even in relatively young individuals[19].

## **AAS Dependence and Addiction**

### *Diagnostic Criteria and Prevalence*

AAS dependence is characterized by continued use despite negative health consequences, withdrawal symptoms, tolerance, loss of control over use, and substantial time devoted to obtaining and using substances. Approximately one-third of individuals who use AAS develop clinically significant dependence[19].

### *Development and Validation of New Measures*

Zoob Carter et al. (2024) developed and validated dependence and craving measures specific to athletes using AAS[19]. The study involved two samples (n=206 and n=224) of male and female strength athletes. The AAS Dependence Scale (AASDS) consists of 15 items across five first-order factors (Effectiveness, Withdrawal, Physical Effects, Psychological Effects, Social Effects) represented by one second-order factor[19]. The AAS Craving Scale (AASCS) consists of 16 items across four first-order factors (Expectation, Environment, Positive Mood, Negative Mood) represented by one second-order factor[19].

Both scales demonstrated excellent psychometric properties. Confirmatory factor analyses confirmed factorial structures. Internal consistency was good to excellent (Cronbach's  $\alpha \geq 0.80$ )[19]. Evidence for concurrent, convergent, and discriminant validity was provided through associations with theoretically related variables including doping moral disengagement, self-regulatory efficacy, and existing dependence measures[19].

## **Gender Differences**

### *Female Athletes and AAS Use*

While AAS use is far more common in males, female athletes also misuse these substances, particularly in bodybuilding and physique sports. Female AAS users face unique challenges including masculinization effects (voice deepening, clitoral enlargement, facial hair, male-pattern baldness) that may be irreversible. Psychiatric effects in females include depression, anxiety, increased aggression, and body image disturbances related to loss of femininity.

Limited research from 2015-2025 has examined gender differences in psychiatric outcomes. Windfeldt-Mathiasen et al. (2022) found no significant gender differences in depression prevalence among AAS users, though women reported higher rates of body dissatisfaction[16]. Qualitative research has documented that female AAS users experience identity conflicts, oscillating between desires for increased muscularity and concerns about appearing "too masculine."

## **Risk Factors and Predictors**

### *Individual Vulnerability Factors*

Multiple factors documented in recent research predict initiation of AAS use and development of psychiatric complications. Body image disorders, particularly muscle dysmorphia, represent primary risk factors[17][18]. Personality characteristics including impulsivity, sensation-seeking, and narcissism have been associated with AAS use and dependence.

### *Sociocultural Influences*

Contemporary sociocultural factors documented in recent research contribute to AAS use. Exposure to muscularity-oriented social media content is significantly associated with heightened MD symptoms among boys and men. Social media platforms proliferate unrealistic body images, often depicting physiques unattainable without AAS use. Online communities may normalize AAS use and provide access to substances and information about use patterns.

Competitive pressure in elite sports, despite anti-doping regulations, continues to drive AAS use. Athletes perceive that competitors use performance-enhancing substances, creating pressure to "level the playing field." Economic incentives associated with elite performance (sponsorships, prize money, professional contracts) further motivate use despite known risks.

## **Discussion**

### **Summary of Key Findings**

This comprehensive literature review, synthesizing research from 2015-2025, demonstrates that AAS use among athletes is associated with substantial psychiatric morbidity across multiple domains. Depression and anxiety are consistently elevated in AAS users, with effect sizes ranging from moderate to large (Cohen's  $d=0.4-0.8$ )[15].

Muscle dysmorphia emerges as both driver and consequence of AAS use. The bidirectional relationship between MD and AAS use creates self-perpetuating cycles resistant to intervention[17][18].

Neuroimaging studies reveal structural brain changes including cortical thinning and subcortical volume alterations, providing neurobiological substrates for psychiatric symptoms[8]. These changes correlate with dose and duration, suggesting neurotoxic effects that may be partially irreversible[8][19].

### **Neurobiological Mechanisms**

Aromatization of AAS to estrogens may exacerbate mood symptoms[2]. Direct effects on neurotransmitter systems, particularly serotonin and dopamine, affect mood regulation, impulse control, and reward processing.

AAS induce oxidative stress and neuroinflammation, resulting in neurotoxicity particularly in regions with high androgen receptor density such as hippocampus, amygdala, and prefrontal cortex[19]. These structural changes correlate with observed cognitive and behavioral symptoms[8]. Additionally, AAS disrupt neurosteroid production, affecting GABAergic neurotransmission and potentially contributing to anxiety and mood instability.

Neuroimaging findings from 2015-2025 have provided crucial insights. Bjørnebekk et al. (2017) demonstrated reduced cortical thickness and altered subcortical volumes in AAS users[8]. These structural and functional alterations provide biological substrates for observed psychiatric symptoms.

## Clinical Implications

### *Screening and Prevention*

Findings from 2015-2025 have critical implications for screening and prevention in athletic populations. Healthcare providers, coaches, and sports medicine professionals should routinely screen athletes for AAS use, particularly in high-risk sports (bodybuilding, weightlifting, strength sports). Screening should include assessment of body image concerns, MD symptoms, mood disturbances, and behavioral changes.

Prevention programs should address sociocultural factors driving AAS use, including unrealistic body ideals promoted through social media and competitive pressures in sports. Education about psychiatric risks of AAS should be integrated into athletic training, emphasizing that cognitive impairments may undermine performance goals motivating use. Early intervention for body image disorders may prevent progression to AAS use.

### *Treatment Approaches*

Treatment of AAS-related psychiatric complications requires integrated approaches addressing both substance use and underlying psychopathology. For AAS dependence, management of withdrawal symptoms is critical. Hormonal therapies (human chorionic gonadotropin, selective estrogen receptor modulators) may accelerate recovery of HPG axis, though evidence for efficacy remains limited.

Psychiatric medications (antidepressants, anxiolytics) may be necessary for mood symptoms, though must be used cautiously given complex hormonal milieu. Psychological interventions should target body image distortions characteristic of MD. Cognitive-behavioral therapy (CBT) adapted for body dysmorphic disorder shows promise, though evidence specifically in AAS-using populations remains limited. Exposure and response prevention (ERP), an evidence-based treatment for OCD, may address compulsive behaviors (mirror checking, excessive exercise) associated with MD.

Harm reduction approaches, acknowledging that some athletes may not be willing to cease AAS use immediately, should emphasize safer use practices, medical monitoring, and gradual tapering rather than abrupt cessation.

## Limitations and Methodological Considerations

Several limitations of literature from 2015-2025 warrant discussion. Most studies employ cross-sectional designs, limiting causal inferences about directionality of associations between AAS use and psychiatric symptoms[15]. It remains unclear whether psychiatric symptoms predate and motivate AAS use versus emerge as consequences of use. Longitudinal studies with pre-use baseline assessments are needed to clarify temporal relationships.

Sample sizes in many studies are relatively small, particularly for neuroimaging and neuropsychological investigations, limiting statistical power and generalizability[8].

Assessment of AAS use relies primarily on self-report, which may be subject to recall bias and underreporting[15]. Verification through biological samples (urine, serum analysis) enhances validity but is not always feasible. Heterogeneity in AAS compounds, doses, and patterns of use (stacking, cycling) complicates comparisons across studies[2].

Publication bias may inflate effect sizes, as studies with null findings may be less likely published. Additionally, most research is conducted in Western countries (Europe, North America), limiting generalizability to other cultural contexts where AAS use patterns and psychiatric presentations may differ.

## Directions for Future Research

Multiple research priorities emerge from synthesis of 2015-2025 literature:

1. Longitudinal Studies: Prospective studies following athletes before, during, and after AAS use are needed to establish causal relationships, identify vulnerability factors predicting psychiatric complications, and characterize recovery trajectories.

2. Gender-Specific Research: Expanded investigation of female AAS users is critical, examining gender differences in psychiatric symptoms, motivations for use, and treatment responses.

3. Neuroimaging and Biomarkers: Advanced neuroimaging techniques (functional MRI, diffusion tensor imaging, positron emission tomography) can elucidate brain mechanisms underlying psychiatric symptoms. Identification of biomarkers predicting vulnerability to neurotoxic effects would enhance risk stratification.

4. Treatment Trials: Randomized controlled trials are urgently needed to establish evidence-based treatments for AAS dependence and associated psychiatric complications. Trials should examine

pharmacological interventions (hormonal therapies, psychiatric medications) and psychological interventions (CBT, ERP, harm reduction counseling).

5. Mechanism Studies: Research elucidating specific neurobiological mechanisms (neurotransmitter alterations, oxidative stress, neuroinflammation) can identify novel therapeutic targets.

6. Prevention Research: Evaluation of prevention programs targeting body image, media literacy, and alternatives to AAS use is needed. Studies should assess effectiveness in reducing AAS initiation rates and associated psychiatric morbidity.

7. Network and Systems Approaches: Continued application of network analysis to understand symptom structures and identify intervention targets. Systems-level approaches examining interactions between biological, psychological, and social factors.

### **Public Health Implications**

AAS use among athletes represents significant public health concern documented extensively in research from 2015-2025. Prevalence rates of 10-36% in athletic populations, combined with substantial psychiatric morbidity, indicate widespread impact[2][3].

Public health approaches should address multiple levels: individual (screening, treatment access), community (education in gyms, schools, sports organizations), and policy (regulation of AAS availability, anti-doping enforcement, insurance coverage for treatment). Destigmatization of AAS use disorder is essential to promote treatment-seeking, as current stigma and legal concerns create barriers to care.

Collaboration between mental health providers, endocrinologists, sports medicine physicians, and addiction specialists is necessary for comprehensive care. Specialized clinics for AAS users, offering integrated medical and psychiatric services in nonjudgmental settings, may improve engagement and outcomes.

### **Conclusion**

This comprehensive literature review demonstrates that anabolic-androgenic steroid (AAS) use among athletes is associated with substantial psychiatric morbidity spanning affective, behavioral, cognitive, and neurobiological domains. Across studies published between 2015 and 2025, AAS use has been consistently linked to elevated rates of depression, anxiety, aggression, mania, psychosis, muscle dysmorphia, cognitive impairment, and clinically significant dependence. Neuroimaging and biomarker findings indicate dose-dependent neurotoxicity, with cortical thinning, subcortical volume alterations, and markers of axonal damage providing biological substrates for the observed symptoms. Quantitative evidence from recent meta-analyses further corroborates increased prevalence of muscle dysmorphia and interpersonal violence among AAS users compared to non-using peers.

Muscle dysmorphia emerges as a central factor in both the initiation and maintenance of AAS use, creating bidirectional relationships between body image pathology and substance use that are resistant to intervention. Approximately one-third of users develop AAS dependence, characterized by withdrawal syndromes with prolonged hypogonadism and severe mood disturbance, which may persist for months and contribute to relapse. Epidemiological data indicate AAS prevalence of roughly 10–20% in elite athletes, over 25% in weightlifters, and up to 36% or more in bodybuilding populations, underscoring the scope of the problem in strength and physique sports.

Research from 2015–2025 employing advanced methodologies—including structural and functional neuroimaging, network analysis, large-scale epidemiology, and validated psychiatric assessment instruments—has substantially advanced understanding of these psychiatric consequences. Nonetheless, important gaps remain regarding longitudinal trajectories, gender-specific pathways, causal mechanisms, and evidence-based treatments for AAS-related disorders. Clinical practice should routinely incorporate screening for AAS use and associated psychiatric complications in high-risk athletic populations and adopt multidisciplinary interventions that address substance use, co-occurring psychopathology, and underlying body image pathology. Prevention efforts must target sociocultural pressures promoting unrealistic muscular ideals and the normalization of AAS use within athletic and fitness communities.

Overall, the psychiatric effects of AAS reflect a complex interplay of biological vulnerability, psychological factors, and sociocultural influences, necessitating integration of neuroscience, psychiatry, endocrinology, sports medicine, and social science perspectives. Continued research and clinical attention are essential to mitigate the mental health burden associated with AAS use and to protect the wellbeing of athletes and physically active individuals.

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