

Review

Behavioral and Psychological Changes Associated with Anabolic Steroid Use: A Comprehensive Review

Ridham Agnihotri¹, Rajat Koundal², Hurmandeep Kaur³, Shubham Garg⁴, Anurag Chaudhary⁵

^{1,5} Student, College of Pharmacy, RIMT University, Mandi Gobindgarh, Punjab, India-147301

² Associate Professor, College of Pharmacy, RIMT University, Mandi Gobindgarh, Punjab, India-147301

^{3,4} Assistant Professor, College of Pharmacy, RIMT University, Mandi Gobindgarh, Punjab, India-147301

Corresponding Author:

Ridham Agnihotri

Email:

agnihotriridham@gmail.com

DOI: 10.62896/cplr.3.2.03

Conflict of interest: NIL

Article History

Received: 12/03/2026

Accepted: 27/04/2026

Published: 28/04/2026

Abstract:

Anabolic-androgenic steroids (AAS) are synthetic derivatives of testosterone widely used for medical purposes but increasingly misused for performance enhancement and body image improvement. The rising prevalence of AAS abuse, particularly among athletes, bodybuilders, and young adults, has become a significant public health concern. This review aims to comprehensively evaluate the behavioral and psychological changes associated with AAS use, focusing on underlying neurobiological mechanisms, patterns of abuse, and clinical implications. Evidence indicates that AAS misuse is associated with a broad spectrum of behavioral alterations, including increased aggression, impulsivity, risk-taking behavior, and, in severe cases, violent or criminal tendencies. In addition, users frequently experience psychological disturbances such as mood disorders (depression, mania, and anxiety), dependence, withdrawal symptoms, and cognitive impairments, including memory deficits and poor judgment. These effects are primarily mediated through alterations in central neurotransmitter systems, including dopamine, serotonin, and gamma-aminobutyric acid, as well as structural and functional changes in brain regions such as the amygdala and hippocampus. Both short-term and long-term use of AAS contribute to adverse mental health outcomes, with chronic exposure leading to persistent psychiatric disorders. Risk factors such as high dosage, prolonged use, early initiation, and environmental influences further increase susceptibility. Despite growing evidence, challenges such as underreporting and limited long-term human studies persist. Overall, AAS misuse is strongly linked to significant neuropsychiatric disturbances, highlighting the need for improved awareness, preventive strategies, and multidisciplinary clinical management.

Keywords: Anabolic-androgenic steroids; Behavioral changes; Psychological effects; Aggression; Mood disorders; Substance dependence; Neurobiology

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

1. Introduction: Anabolic-androgenic steroids (AAS) are synthetic derivatives of the endogenous male

hormone testosterone, developed to enhance anabolic effects such as muscle growth while retaining

androgenic properties related to male sexual characteristics. These compounds were initially introduced for legitimate medical purposes, including the treatment of hypogonadism, delayed puberty, muscle wasting conditions, and certain forms of anemia [1,2]. Despite their clinical utility, AAS are widely misused for non-medical purposes, particularly among athletes, bodybuilders, and recreational fitness enthusiasts aiming to improve physical performance and appearance. Such misuse typically involves the administration of supraphysiological doses, stacking of multiple steroid compounds, and prolonged usage cycles, all of which significantly increase the risk of adverse effects [3,4].

The easy availability of these substances through illicit markets and online platforms has further contributed to their widespread abuse. Over the past few decades, the prevalence of AAS misuse has increased substantially across different populations, including adolescents and young adults. Factors such as body image dissatisfaction, competitive pressure, and sociocultural influences play a critical role in driving this trend [5]. AAS use is frequently associated with the concurrent use of other substances, including stimulants and performance-enhancing drugs, which may intensify their harmful consequences [6]. A major concern associated with AAS misuse is its significant impact on behavioral and psychological health. Evidence suggests that AAS use is linked to a wide range of psychiatric manifestations, including increased aggression, mood instability, depression, anxiety, impulsivity, and, in some cases, psychotic symptoms [7,8]. These effects are believed to arise from alterations in central nervous system function, particularly involving neurotransmitter systems such as dopamine, serotonin, and gamma-aminobutyric acid (GABA) [9].

2. Overview of Anabolic Steroids

2.1 Definition and Classification

Anabolic-androgenic steroids (AAS) are synthetic derivatives of testosterone designed to enhance anabolic effects, such as protein synthesis and muscle growth, while maintaining varying degrees of androgenic activity related to male sexual characteristics. Structurally, these compounds are modifications of the testosterone molecule, allowing alterations in pharmacokinetic properties, potency, and tissue selectivity [10,11].

AAS can be broadly classified based on their chemical structure and route of administration:

- Testosterone esters (injectable forms): e.g., testosterone enanthate, testosterone cypionate
- 17 α -alkylated steroids (oral forms): e.g., stanozolol, oxandrolone
- 19-nortestosterone derivatives: e.g., nandrolone

These structural modifications influence their metabolic stability, bioavailability, and duration of action. For example, 17 α -alkylated steroids are resistant to hepatic metabolism, making them orally active but also increasing the risk of hepatotoxicity [12].

Table 1: Classification and Examples of Anabolic-Androgenic Steroids

| Class | Examples | Route of Administration | Key Features |
|---------------------------------|--|-------------------------|-------------------------------|
| Testosterone esters | Testosterone enanthate, testosterone cypionate | Injectable | Long-acting, used in HRT |
| 17 α -alkylated steroids | Stanozolol, Oxandrolone | Oral | Hepatotoxic, orally active |
| 19-nortestosterone derivatives | Nandrolone | Injectable | High anabolic, low androgenic |

2.2 Commonly Used Steroids

Several AAS are commonly used both in clinical practice and illicitly for performance enhancement. Among the most frequently encountered compounds are:

- Testosterone: The primary endogenous androgen, often used therapeutically for hormone replacement but also widely abused in higher doses for anabolic effects [13].
- Nandrolone: A synthetic derivative with relatively stronger anabolic and weaker androgenic effects, commonly used to promote muscle growth and recovery [14].

- **Stanozolol:** A 17 α -alkylated oral steroid known for enhancing strength and lean muscle mass, frequently used in athletic settings [15].

Other commonly misused AAS include oxandrolone, methandrostenolone, and boldenone. These substances are often used in combination (“stacking”) or in cycles to maximize anabolic outcomes while attempting to minimize adverse effects, although such practices significantly increase health risks [16].

2.3 Mechanism of Action

The biological effects of AAS are primarily mediated through their interaction with androgen receptors and their influence on central nervous system (CNS) neurotransmitters.

Table 2: Neurotransmitter Systems Affected by AAS

| Neurotransmitter | Normal Function | Effect of AAS | Clinical Outcome |
|------------------|--------------------|---------------------|------------------------|
| Dopamine | Reward, motivation | Increased activity | Addiction, dependence |
| Serotonin | Mood regulation | Decreased activity | Depression, aggression |
| GABA | Inhibitory control | Disrupted signaling | Anxiety, impulsivity |

Androgen Receptor Binding

AAS exert their anabolic and androgenic effects by binding to intracellular androgen receptors located in various tissues, including skeletal muscle, liver, and brain. Upon binding, the steroid-receptor complex translocates to the nucleus, where it interacts with specific DNA sequences known as androgen response elements (AREs). This interaction leads to the regulation of gene expression, promoting increased protein synthesis, nitrogen retention, and muscle hypertrophy [17].

Effects on CNS Neurotransmitters

Beyond their peripheral effects, AAS significantly influence central nervous system function. They modulate key neurotransmitter systems, including:

- **Dopamine:** Associated with reward, motivation, and reinforcement, contributing to dependence and addictive behavior
- **Serotonin:** Regulates mood and emotional stability; alterations may lead to depression or aggression
- **Gamma-aminobutyric acid (GABA):** Involved in inhibitory signaling; disruption may increase impulsivity and anxiety

AAS-induced alterations in these neurotransmitter systems are believed to underlie many of the behavioral and psychological effects observed in users, including mood swings, aggression, and psychiatric disturbances [18,19]. Anabolic steroids are structurally modified testosterone derivatives with diverse pharmacological profiles. Their ability to enhance muscle growth through androgen receptor activation, combined with their influence on central neurotransmitter systems, explains both their therapeutic utility and their potential to induce significant behavioral and psychological changes when misused [20].

3. Prevalence and Patterns of Abuse

The misuse of anabolic-androgenic steroids (AAS) has become a significant public health concern worldwide. Over the past few decades, the prevalence of AAS use has expanded beyond elite athletes to include recreational gym users, adolescents, and the general population. This growing trend reflects changing societal attitudes toward body image, performance enhancement, and physical appearance [21,22].

3.1 Global and Regional Data

Epidemiological studies indicate that AAS use is a global phenomenon, with varying prevalence across regions and populations. It is estimated that approximately 1–5% of the general population has used AAS at least once in their lifetime, with higher rates observed among males compared to females [23]. In North America and Europe, AAS use is particularly prevalent among recreational athletes and gym users, with reported lifetime prevalence rates ranging from 3% to 6% in certain subgroups. In contrast, emerging data from developing countries, including regions of Asia and the Middle East, suggest a rising trend in AAS misuse, especially among young adults and fitness enthusiasts [24,25]. Among adolescents, the use of AAS is especially concerning,

with studies reporting prevalence rates of 1–3% in school-aged populations. Early exposure increases the risk of long-term psychological and behavioral complications [26].

3.2 Use in Bodybuilding, Sports, and Youth

AAS misuse is most associated with bodybuilding and competitive sports, where individuals seek enhanced muscle mass, strength, and physical performance. Bodybuilders often use AAS to achieve rapid increases in lean body mass and reduced body fat, while athletes may use them to gain a competitive advantage [27]. In recent years, AAS use has also become widespread among recreational gym users who are not involved in professional sports. The desire for an idealized body image, influenced by social media and cultural standards, has significantly contributed to this trend [28,29].

Adolescents and young adults represent a particularly vulnerable group. Peer pressure, low self-esteem, and body dissatisfaction are key factors driving AAS use in this population. Early initiation is associated with a higher likelihood of continued use and the development of substance dependence [30].

4. Neurobiological Basis of Psychological Effects

The behavioral and psychological effects associated with anabolic-androgenic steroid (AAS) use are largely mediated through complex neurobiological mechanisms involving alterations in neurotransmitter systems, brain structure and function, and hormonal balance [31]. AAS can cross the blood–brain barrier and exert direct effects on the central nervous system (CNS), leading to significant changes in mood, cognition, and behavior [32,33].

4.1 Dopamine, Serotonin, and GABA Pathways

AAS influence several key neurotransmitter systems that regulate emotional and behavioral responses:

- **Dopamine:**
Dopamine plays a central role in reward, motivation, and reinforcement pathways. AAS use has been shown to enhance dopaminergic activity in brain regions such as the mesolimbic pathway, particularly the nucleus accumbens. This overstimulation contributes to feelings of euphoria, increased motivation, and, in some cases, dependence and addictive behaviors [34].
- **Serotonin:**
Serotonin is crucial for mood regulation,

emotional stability, and impulse control. AAS exposure can reduce serotonergic activity, which is associated with increased aggression, irritability, and depressive symptoms. Reduced serotonin levels are also linked to impaired emotional regulation and heightened risk of mood disorders [35].

- **Gamma-aminobutyric acid (GABA):**
GABA is the primary inhibitory neurotransmitter in the CNS and plays a key role in controlling anxiety and impulsivity. AAS may alter GABAergic signaling, leading to decreased inhibitory control, increased excitability, and heightened aggressive or impulsive behavior [36].

The combined disruption of these neurotransmitter systems creates a neurochemical imbalance that underlies many of the psychological disturbances observed in AAS users.

4.2 Brain Regions Affected (Amygdala, Hippocampus)

AAS use has been associated with structural and functional changes in several brain regions involved in emotion, memory, and behavior:

- **Amygdala:**
The amygdala is a key structure involved in processing emotions such as fear, aggression, and threat perception. AAS exposure has been linked to increased amygdala reactivity, which may contribute to heightened aggression, irritability, and emotional instability commonly observed in users [37].
- **Hippocampus:**
The hippocampus plays an essential role in memory formation and emotional regulation. Chronic AAS use may impair hippocampal function, leading to memory deficits and increased susceptibility to anxiety and depressive disorders [38].

4.3 Hormonal Imbalance: AAS misuse disrupts the normal endocrine balance by suppressing the hypothalamic–pituitary–gonadal (HPG) axis. Exogenous steroid administration leads to reduced endogenous testosterone production, resulting in hormonal dysregulation [39]. This imbalance affects not only physical health but also psychological well-being. Fluctuations in hormone levels are associated

with mood swings, irritability, depression, and anxiety. During withdrawal phases, individuals may experience severe depressive symptoms due to decreased endogenous hormone levels [40,41].

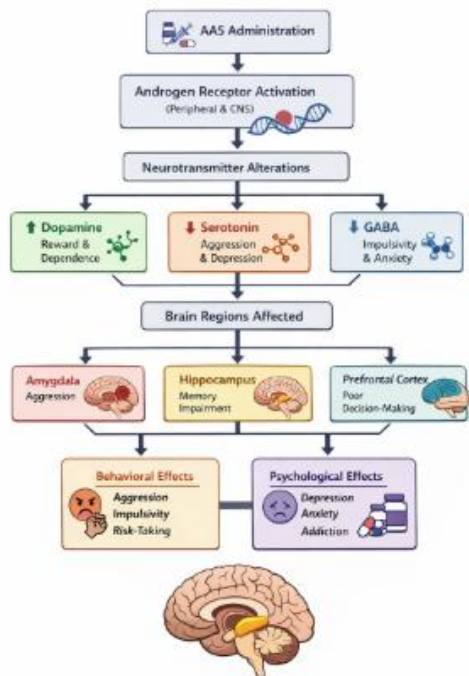


Figure 1: Mechanistic Pathway of Anabolic Steroid-Induced Behavioral and Psychological Effects

5. Behavioral Changes Associated with AAS Use

Anabolic-androgenic steroid (AAS) misuse has been strongly associated with a range of behavioral alterations that can significantly impact individual functioning and social interactions. These behavioral changes are primarily linked to neurochemical imbalances, hormonal fluctuations, and structural changes in brain regions responsible for emotional regulation and impulse control. The severity of these effects often depends on the dose, duration of use, and individual susceptibility [42,43].

5.1 Aggression (“Roid Rage”)

One of the most widely reported behavioral effects of AAS use is increased aggression, commonly referred to as “roid rage.” This phenomenon is characterized by sudden, intense episodes of anger, irritability, and hostility that may be disproportionate to the triggering stimulus. AAS-induced aggression is believed to result from alterations in serotonin and dopamine pathways, along with increased activity in the amygdala, which enhances emotional reactivity. High doses of AAS have been particularly associated with heightened

aggressive responses and reduced ability to control anger [44,45].

5.2 Impulsivity

Impulsivity is another significant behavioral consequence of AAS misuse. It involves a reduced ability to control immediate reactions and a tendency to act without considering potential consequences. This behavior is linked to dysfunction in the prefrontal cortex, which is responsible for executive functions such as decision-making and self-control. AAS-induced alterations in neurotransmitter systems, particularly decreased serotonergic activity, contribute to impaired impulse regulation [46].

5.3 Risk-Taking Behavior

AAS users often demonstrate an increased tendency toward risk-taking behaviors, which may include unsafe sexual practices, substance abuse, reckless driving, and engagement in dangerous activities. This behavior is associated with enhanced dopaminergic activity in reward pathways, which increases the pursuit of pleasurable or stimulating experiences despite potential negative consequences. The combination of heightened reward sensitivity and reduced inhibitory control contributes to this pattern [47].

5.4 Violence and Criminal Behavior

In severe cases, AAS misuse has been linked to violent and criminal behavior, including assault, domestic violence, and other forms of aggression. Studies have reported a higher incidence of violent offenses among AAS users compared to non-users, particularly in individuals using high doses or combining AAS with other substances [48].

The association between AAS use and criminal behavior is complex and may involve multiple contributing factors, including personality traits, environmental influences, and concurrent substance abuse. Nonetheless, the role of AAS in exacerbating aggressive and antisocial tendencies is well documented [49,50].

6. Psychological Effects

Anabolic-androgenic steroid (AAS) use is associated with a wide spectrum of psychological disturbances that can significantly affect mental health and quality of life. These effects range from mood disorders and dependence to cognitive impairments. The severity and manifestation of these psychological changes depend on factors such as dosage, duration of use,

individual susceptibility, and concurrent substance use [51].

6.1 Mood Disorders

AAS misuse has been strongly linked to the development of various mood disorders, including depression, mania, and anxiety. [52].

Depression

Depression is commonly observed, particularly during withdrawal phases when endogenous testosterone levels are suppressed. Users may experience persistent sadness, loss of interest in activities, fatigue, and, in severe cases, suicidal ideation [53].

Mania

During active AAS use, especially at high doses, individuals may exhibit symptoms of mania, including elevated mood, increased energy, irritability, grandiosity, and reduced need for sleep. These manic episodes can impair judgment and lead to risky or impulsive behavior [54].

Anxiety

Anxiety disorders are also frequently reported among AAS users. Symptoms may include restlessness, nervousness, and heightened stress responses. Alterations in GABAergic and serotonergic pathways are believed to play a key role in the development of anxiety-related symptoms [55].

6.2 Dependence and Addiction

Chronic use of AAS can lead to psychological dependence and addiction, characterized by compulsive use despite awareness of harmful consequences.

Withdrawal Symptoms

Discontinuation of AAS often results in withdrawal symptoms such as depression, fatigue, irritability, sleep disturbances, and decreased libido. These symptoms are largely due to suppression of the hypothalamic–pituitary–gonadal axis and reduced endogenous testosterone production [56].

6.3 Cognitive Effects

In addition to mood and behavioral changes, AAS use has been associated with impairments in cognitive function [57].

Poor Judgment

AAS users often exhibit impaired decision-making and poor judgment, which may result from dysfunction in the prefrontal cortex [58]. This can lead to risky behaviors, inability to assess consequences, and difficulty in maintaining social and occupational

responsibilities [59]. The psychological effects of anabolic steroid use encompass mood disorders, dependence, and cognitive impairments. These effects are driven by complex interactions between neurochemical changes, hormonal imbalance, and structural brain alterations [60].

7. Short-term vs Long-term Effects

The psychological impact of anabolic-androgenic steroid (AAS) use can be categorized into short-term (acute) and long-term (chronic) effects [61]. These effects vary in severity depending on dosage, duration of use, and individual susceptibility. Understanding this distinction is important for assessing both immediate and persistent risks associated with AAS misuse [62].

7.1 Acute Psychological Changes (Short-term Effects)

Short-term psychological effects typically occur during active AAS use, particularly when high or supraphysiological doses are administered.

Common acute effects include:

- Euphoria and increased confidence: Users may experience elevated mood, heightened self-esteem, and increased motivation.
- Irritability and mood swings: Rapid fluctuations in mood are frequently reported.
- Increased aggression: Episodes of anger and hostility may occur, especially at higher doses.
- Impulsivity and risk-taking behavior: Reduced inhibition may lead to reckless decisions.

These effects are primarily linked to alterations in neurotransmitter systems, including increased dopaminergic activity and decreased serotonergic regulation [63].

7.2 Chronic Psychiatric Disorders (Long-term Effects)

Long-term use of AAS is associated with more and persistent psychological consequences, which may continue even after discontinuation.

Common chronic effects include:

- Major depressive disorder: Often observed during withdrawal due to hormonal suppression.
- Anxiety disorders: Persistent anxiety and stress-related symptoms.

- Cognitive impairments: Memory deficits and reduced executive function.
- Psychotic symptoms: In rare cases, long-term use may lead to paranoia, hallucinations, or delusional thinking.

Chronic exposure to AAS can result in lasting changes in brain structure and function, particularly in regions such as the hippocampus and prefrontal cortex. These alterations contribute to the persistence of psychiatric symptoms [64,65].

8. Risk Factors

The development and severity of behavioral and psychological effects associated with AAS use are influenced by multiple risk factors. These factors determine individual susceptibility and the likelihood of adverse outcomes [66].

8.1 Dose and Duration

The most significant risk factor is the dose and duration of AAS use. Supraphysiological doses and prolonged usage cycles are strongly associated with increased severity of psychological disturbances, including aggression, mood disorders, and dependence [67].

8.2 Age and Gender

Age plays a critical role, with adolescents and young adults being particularly vulnerable due to ongoing brain development. Early exposure to AAS can lead to long-term psychological consequences and increased risk of substance dependence [68].

8.3 Genetic Predisposition

Genetic factors may influence an individual's susceptibility to AAS-induced psychological effects. Variations in genes related to neurotransmitter systems (e.g., dopamine and serotonin receptors) may affect behavioral responses, including aggression and mood instability [69].

8.4 Environmental and Social Factors

Environmental influences, including peer pressure, cultural norms, and exposure to performance-driven environments (e.g., sports and bodybuilding), significantly contribute to AAS misuse. Social media, body image expectations, and the desire for physical perfection further reinforce the use of AAS [70]. The psychological effects of AAS are influenced by a combination of biological, behavioral, and environmental risk factors. High doses, prolonged use, early initiation, genetic vulnerability, and social influences collectively increase the likelihood of

adverse outcomes, emphasizing the need for targeted prevention and intervention strategies [71].

9. Clinical Implications

The increasing misuse of anabolic-androgenic steroids (AAS) presents significant challenges for healthcare systems, particularly in the identification, diagnosis, and management of associated psychological and behavioral disorders. Effective clinical management requires a multidisciplinary approach involving medical, psychiatric, and psychosocial interventions [72].

9.1 Diagnostic Challenges

Diagnosing AAS-related psychological disorders can be complex due to overlapping symptoms with primary psychiatric conditions such as depression, anxiety, and bipolar disorder. Many users may not disclose steroid use due to stigma, lack of awareness, or fear of legal consequences, leading to underdiagnosis or misdiagnosis [73].

9.2 Psychiatric Management

Management of AAS-induced psychological effects requires individualized treatment strategies. Pharmacological interventions may be necessary to address specific symptoms:

- Antidepressants for depressive symptoms
- Mood stabilizers or antipsychotics for severe mood disturbances or psychosis
- Anxiolytics for anxiety-related symptoms

In cases of dependence, gradual discontinuation and medical supervision are essential to manage withdrawal symptoms and prevent relapse. Monitoring hormonal recovery, particularly restoration of endogenous testosterone levels, is also important in long-term management [74].

10. Prevention and Awareness

Preventing anabolic steroid misuse requires coordinated efforts involving education, regulation, and active involvement of healthcare professionals. Increasing awareness about the risks associated with AAS is critical to reducing their misuse [75,76].

10.1 Education Programs

Educational initiatives targeting adolescents, athletes, and gym users are essential in preventing AAS misuse. These programs should focus on:

- Raising awareness about the psychological and physical risks of AAS

- Promoting healthy alternatives for fitness and performance enhancement [77]
- Addressing misconceptions related to body image and steroid use

School-based and community-based education programs have been shown to be effective in reducing the likelihood of substance abuse [78].

10.2 Regulation in Sports

Strict regulation and enforcement of anti-doping policies are crucial in controlling AAS misuse in sports. Organizations such as the World Anti-Doping Agency (WADA) have established guidelines and testing protocols to detect and prevent the use of performance-enhancing drugs. Regular testing, penalties for violations, and increased surveillance help deter athletes from using AAS and promote fair competition [79].

10.3 Role of Healthcare Professionals

Healthcare professionals play a key role in both prevention and early intervention. Physicians, pharmacists, and mental health professionals should:

- Educate patients about the risks of AAS use
- Identify early signs of misuse
- Provide counseling and support

Routine screening and open communication can help detect AAS misuse at an early stage and prevent progression to severe psychological and behavioral complications [80]. Effective prevention of AAS misuse requires a multifaceted approach involving education, regulation, and proactive healthcare engagement. Increased awareness and early intervention are essential to mitigate the growing public health impact of anabolic steroid abuse [81].

11. Limitations

Despite the growing body of evidence on the behavioral and psychological effects of anabolic-androgenic steroids (AAS), several limitations exist that restrict a comprehensive understanding of their long-term impact.

11.1 Lack of Long-Term Human Studies

One of the primary limitations in AAS research is the scarcity of long-term, controlled human studies. Ethical constraints prevent the administration of supraphysiological doses of AAS in clinical trials, leading to a reliance on observational studies, case reports, and animal models. While animal studies provide valuable insights into neurobiological

mechanisms, their findings may not fully translate to human populations. Consequently, the long-term psychiatric effects of AAS, including persistent cognitive impairment and chronic mood disorders, remain insufficiently characterized [82].

11.2 Underreporting of Abuse

AAS misuse is frequently underreported, which limits the accuracy of prevalence data and clinical assessment. Many users may conceal their steroid use due to stigma, legal concerns, or lack of awareness regarding its relevance to their symptoms. This underreporting can lead to misdiagnosis, delayed intervention, and an underestimation of the true burden of AAS-related psychological disorders. Additionally, variability in patterns of use, including cycling and stacking, further complicates data collection and interpretation [83]. The lack of robust long-term human data and the issue of underreporting represent significant barriers to fully understanding the psychological impact of AAS misuse. Addressing these limitations is essential for improving research quality, clinical diagnosis, and public health strategies [84].

12. Future Perspectives

Advancements in research and clinical approaches are necessary to better understand and manage the psychological effects of AAS use. Several future directions hold promise for improving outcomes and bridging current knowledge gaps.

13. Conclusion

The present review highlights the significant behavioral and psychological consequences associated with anabolic-androgenic steroid (AAS) misuse. While AAS have legitimate medical applications, their non-medical use, particularly at supraphysiological doses, is strongly linked to a wide range of adverse mental health outcomes. These include increased aggression, impulsivity, risk-taking behavior, mood disorders such as depression and mania, anxiety, dependence, and cognitive impairments. The underlying mechanisms of these effects are complex and involve alterations in central neurotransmitter systems, including dopamine, serotonin, and gamma-aminobutyric acid, as well as structural and functional changes in key brain regions such as the amygdala, hippocampus, and prefrontal cortex. Hormonal imbalances resulting from suppression of endogenous testosterone further contribute to psychological

instability, particularly during withdrawal phases. A comparative evaluation of short-term and long-term effects indicates that while acute psychological changes may be reversible, chronic AAS use can lead to persistent psychiatric disorders that require clinical intervention. The presence of multiple risk factors, including high dosage, prolonged use, early initiation, genetic predisposition, and environmental influences, further exacerbates these outcomes. From a clinical perspective, the diagnosis and management of AAS-related psychological disorders remain challenging due to underreporting and symptom overlap with primary psychiatric conditions. Effective management requires a multidisciplinary approach involving pharmacological treatment, psychological counseling, and long-term rehabilitation strategies.

References

1. Kanayama G, Hudson JI, Pope HG. Long-term psychiatric effects of anabolic steroids. *Am J Psychiatry*. 2008;165(3):341–8.
2. Pope HG, Wood RI, Rogol A, Nyberg F, Bowers L, Bhasin S. Adverse health consequences of anabolic steroids. *Endocr Rev*. 2014;35(3):341–75.
3. Rajwinder, K., Singh, R., Lalit, Jaswinder, K., & Rajat. (2020). Evaluation of the Antidepressant Like Activity of Ethanolic Extract of *Calendula officinalis* using Rodent Models (Wistar Rat) of Depression. *Current Psychopharmacology*, 9(1), 58-67.
4. Singh, G., Singh, S., Kaur, H., Garg, S., & Khera, N. (2026). Pearl Millet Starch: A Natural Excipient With Potential for Industrial Pharmaceutical Use.
5. Agrahari, S., Garg, S., War, A. H., Kumar, A., Tantray, M. H., Koundal, R., & Kaur, H. (2024). A Comprehensive Approach to the Management of Severe Coronary Artery Calcification Using Coronary Atherectomy. *Journal of Pharma Insights and Research*, 2(2), 245-254.
6. Aravanan, P., Karthickeyan Krishnan, D. A., Ranjan, N., Kar, D. V. K. P., Sharang Bali, D. D. S., & Rajat, D. A. (2023). Understanding Menopausal Health: A Comprehensive Review of Menopausal Diseases and Their Impact on Women's Health.
7. Mali, S. K., Gupta, S. K., Yadav, R. P., Sharma, S., & Koundal, R. Unveiling An Antibiofilm Marvel to Combat Antibiotics Resistance.
8. Varma, A. K., Sarvan, S. K., Kumari, R., Kumar, R., Ranjan, R., Rajat, N. R. K., & Kotnala, M. Formulation and Characterization of Toremifene Self-Microemulsifying Drug Delivery System for Enhancement of Oral Bioavailability.
9. Nieschlag E, Vorona E. Medical consequences of doping. *Eur J Endocrinol*. 2015;173(2):R47–58.
10. Hartgens F, Kuipers H. Effects of androgenic-anabolic steroids. *Sports Med*. 2004;34(8):513–54.
11. Sagoe D, Molde H, Andreassen CS, et al. Global epidemiology of AAS use. *Ann Epidemiol*. 2014;24(5):383–98.
12. Kanayama G, Pope HG. Illicit use of anabolic steroids. *Horm Behav*. 2018;100:131–40.
13. Pope HG, Katz DL. Psychiatric effects of anabolic steroids. *Arch Gen Psychiatry*. 1994;51(5):375–82.
14. Piacentino D, Kotzalidis GD, del Casale A, et al. Anabolic steroid abuse and psychopathology. *Curr Neuropharmacol*. 2015;13(1):101–21.
15. Wood RI. Reinforcing aspects of anabolic steroids. *Physiol Behav*. 2008;94(1):21–31.
16. Kicman AT. Pharmacology of anabolic steroids. *Br J Pharmacol*. 2008;154(3):502–21.
17. Basaria S. Androgen abuse in athletes. *J Clin Endocrinol Metab*. 2010;95(4):1533–43.
18. Handelsman DJ. Androgen physiology. *Endocr Rev*. 2013;34(5): 550–75.
19. Bhasin S, Woodhouse L, Storer TW. Testosterone effects. *N Engl J Med*. 2001;345:1–12.
20. Vanberg P, Atar D. Androgenic steroid abuse. *Eur J Cardiovasc Prev Rehabil*. 2010;17(3): 291–7.
21. Thiblin I, Petersson A. Pharmacology of steroids. *Fundam Clin Pharmacol*. 2005;19(1):27–44.

22. Parkinson AB, Evans NA. AAS use patterns. *Drug Alcohol Depend.* 2006;79(3): 273–83.
23. Heemers HV, Tindall DJ. Androgen receptor biology. *Endocr Rev.* 2007;28(7):778–808.
24. Henderson LP. Steroids and CNS effects. *J Steroid Biochem Mol Biol.* 2007;106:46–56.
25. Pomara C, Neri M, Bello S, Fiore C, Riezzo I, Turillazzi E. Neurotoxicity by synthetic androgen steroids: oxidative stress, apoptosis, and neuropathology: a review. *Current neuropharmacology.* 2015 Jan 1;13(1):132-45.
26. Rashid I, Mir MA, Andleeb A, Kumar A, Munshi U, Habib D, Ahmad S. Role of melatonin receptors as regulators of neurophysiology and therapeutic targets. *Journal of Pharma Insights and Research.* 2024 Apr 29;2(2):255-65.
27. Skarberg K, Nyberg F, Engstrom I. Multisubstance use as a feature of addiction to anabolic-androgenic steroids. *European addiction research.* 2009 Feb 2;15(2):99-106.
28. Ip EJ, Barnett MJ, Tenerowicz MJ, Perry PJ. The Anabolic 500 survey: Characteristics of male users versus nonusers of anabolic-androgenic steroids for strength training. *Pharmacotherapy: The journal of human pharmacology and drug therapy.* 2011 Aug;31(8):757-66.
29. Rashid I, Nepa R, Kumar A, Kaur N, Kumar M, Kaur LP. Exploring the Intersection of Cellular Regulation, Aging, and Disease Insights into Mechanisms and Implications.
30. Pope HG, Kanayama G. Global steroid abuse trends. *Lancet Psychiatry.* 2016;3(4): 356–63.
31. Hallgren M, Tengström A. Steroid use adolescents. *Eur Addict Res.* 2009;15:99–106.
32. Dodge TL, Jaccard JJ. Adolescent steroid use. *J Adolesc Health.* 2006;39(3):357–65.
33. Kanayama G, Hudson JI. Bodybuilding steroid abuse. *Drug Alcohol Depend.* 2010;102:1–8.
34. Griffiths S, Murray SB. Body image and steroids. *Body Image.* 2015;14:62–9.
35. Eisenberg ME, Wall M. Youth steroid use. *Pediatrics.* 2012;130(1): e1–8.
36. Hildebrandt T, Langenbucher JW. Poly-drug use steroids. *Drug Alcohol Depend.* 2011;118:407–13.
37. Kanayama G, Brower KJ. Steroid dependence. *Drug Alcohol Depend.* 2009;102:130–7.
38. Wood RI, Stanton SJ. Neurobiology steroids. *Neuroscience.* 2012;229:1–12.
39. Piacentino D, D Kotzolidis G, Del Casale A, Rosaria Aromataro M, Pomara C, Girardi P, Sani G. Anabolic-androgenic steroid use and psychopathology in athletes. A systematic review. *Current neuropharmacology.* 2015 Jan 1;13(1):101-21.
40. Rashid, I., Kalandria, A., Kaur, N., Sharma, A., Qadir, S., Singh, R., & Ifaq, Q. (2024). Role of Cellular Senescence in Neurodegeneration: Mechanisms and Therapeutic Implications. *Journal of Pharma Insights and Research*, 2(2), 215-225.
41. Tobiansky DJ, Wallin-Miller KG, Floresco SB, Wood RI, Soma KK. Androgen regulation of the mesocorticolimbic system and executive function. *Frontiers in endocrinology.* 2018 Jun 5;9:279.
42. Seara FD, Fortunato RS, Carvalho DP, Nascimento JH. Neurophysiological repercussions of anabolic steroid abuse: a road into neurodegenerative disorders. *Sex Hormon Neurodegenerative Process Dis.* 2018 May 2;10:225.
43. Carré JM, Archer J. Testosterone and human behavior: the role of individual and contextual variables. *Current opinion in psychology.* 2018 Feb 1;19:149-53.
44. Brower KJ. Anabolic steroids: addictive, psychiatric, and medical consequences. *American Journal on Addictions.* 1992 Jan 1;1(2):100-14.
45. Tan RS, Scally MC. Anabolic steroid-induced hypogonadism—towards a unified hypothesis of anabolic steroid action. *Medical hypotheses.* 2009 Jun 1;72(6):723-8.
46. Brower KJ. Withdrawal symptoms steroids. *Psychiatr Ann.* 2002;32: 130–6.
47. Daly RC, Su TP. Neuroendocrine effects steroids. *Psychiatry Res.* 2003;120: 1–12.

48. Choi PY, Pope HG. Aggression steroids. *Ann Clin Psychiatry*. 1994;6: 235–41.
49. Thiblin I, Runeson B. Steroids and violence. *Int J Law Psychiatry*. 2007;30: 1–9.
50. Pagonis TA, Angelopoulos NV. Steroid aggression study. *Eur Psychiatry*. 2006;21: 138–41.
51. Hall RCW, Hall RCW. Psychiatric effects of anabolic-androgenic steroids. *Psychosomatics*. 2005;46(4):285–290.
52. Moeller FG, Barratt ES. Impulsivity and its neurobiological basis. *Am J Psychiatry*. 2001;158(11):1783–1793.
53. Bechara A. Risky business: Emotion, decision-making, and addiction. *Science*. 2005;307(5706):580–584.
54. Pope HG Jr, Kouri EM. Anabolic-androgenic steroid use and aggression. *Arch Gen Psychiatry*. 1995;52(5):375–382.
55. Darke S, Torok M. The role of substance use in violence: A review. *Addiction*. 2012;107(1):1–8.
56. Kanayama G, Hudson JI, Pope HG Jr. Behavioral and psychological effects of anabolic-androgenic steroids. *Drug Alcohol Depend*. 2012;125(1–2):1–8.
57. Malone DA Jr, Dimeff RJ. The use of anabolic steroids: Psychiatric and medical effects. *Mayo Clin Proc*. 1992;67(3):264–272.
58. Brower KJ. Anabolic steroid abuse and dependence. *Psychiatr Clin North Am*. 1997;20(1):131–148.
59. Pope HG Jr, Katz DL. Affective and psychotic symptoms associated with anabolic steroid use. *Arch Gen Psychiatry*. 1994;51(5):375–382.
60. Pope HG Jr, Hudson JI. Anabolic steroid-induced mania. *J Clin Psychiatry*. 1995;56(1):41–47.
61. Kindlundh AMS, Lindblom J, Bergström L, Nyberg F. The anabolic-androgenic steroid nandrolone alters the density of serotonergic receptors in the brain. *Brain Res*. 2001;929(1):107–112.
62. Brower KJ. Withdrawal from anabolic steroids and associated depression. *Am J Psychiatry*. 1989;146(1):107–108.
63. Wood RI. Anabolic-androgenic steroid dependence? Insights from animals and humans. *Neuroscience*. 2008;94(1):21–31.
64. Heffernan TM. Cognitive effects of anabolic steroid use. *Drug Alcohol Depend*. 2015;150:1–8.
65. Clark AS, Henderson LP. Behavioral and physiological responses to anabolic-androgenic steroids. *Neurosci Biobehav Rev*. 2003;27(5):413–436.
66. Kanayama G, Pope HG Jr. Illicit use of androgens and other hormones: Recent advances. *Am J Addict*. 2013;22(5):1–9.
67. Hartgens F, Kuipers H. Effects of androgenic-anabolic steroids in athletes. *Sports Med*. 2004;34(8):513–554.
68. Pope HG Jr, Wood RI, Rogol A, Nyberg F, Bowers L, Bhasin S. Adverse health consequences of performance-enhancing drugs. *Endocr Rev*. 2014;35(3):341–375.
69. Wood RI. Anabolic-androgenic steroid effects on the brain. *Physiol Behav*. 2008;94(1):21–31.
70. Bjørnebekk A, Westlye LT, Walhovd KB, et al. Structural brain changes in long-term anabolic steroid users. *Biol Psychiatry*. 2017;82(4):294–302.
71. Hauger LE, Westlye LT, Bjørnebekk A. Anabolic steroid use and brain imaging findings. *Neuroimage Clin*. 2019;22:101–110.
72. Kanayama G, Hudson JI, Pope HG Jr. Long-term psychiatric effects of anabolic steroid use. *Am J Psychiatry*. 2008;165(3):341–348.
73. Parkinson AB, Evans NA. Anabolic androgenic steroids: A survey of 500 users. *Drug Alcohol Depend*. 2006;79(2):273–283.
74. Dodge TL, Hoagland MF. The use of anabolic androgenic steroids and polypharmacy among adolescents. *J Adolesc Health*. 2006;39(3):357–365.
75. Beaver KM, Wright JP, DeLisi M. Genetic influences on aggression. *Biol Psychiatry*. 2008;63(1):1–7.
76. Griffiths S, Murray SB, Touyz S. The influence of social media on body image. *Body Image*. 2015;14:62–69.

77. Sagoe D, Molde H, Andreassen CS, Torsheim T, Pallesen S. The global epidemiology of anabolic-androgenic steroid use. *Drug Alcohol Depend.* 2014;139:1–10.
78. Pope HG Jr, Kanayama G, Hudson JI. Risk factors and clinical management of anabolic steroid use. *Lancet Psychiatry.* 2016;3(4):356–363.
79. Ekor M. The growing use of herbal medicines: Issues relating to adverse reactions and challenges in monitoring safety. *Front Pharmacol.* 2014;4:177.
80. Basaria S. Androgen abuse in athletes: Detection and consequences. *J Clin Endocrinol Metab.* 2010;95(4):1533–1543.
81. Kanayama G, Hudson JI, Pope HG Jr. Treatment of anabolic-androgenic steroid dependence. *Drug Alcohol Depend.* 2010;102(1–3):1–8.
82. Brower KJ. Treatment strategies for anabolic steroid dependence. *Psychiatr Clin North Am.* 1997;20(1):131–148.
83. Yesalis CE, Bahrke MS. Anabolic-androgenic steroids and related substances. *Curr Sports Med Rep.* 2002;1(6):326–330.
84. Goldberg L, MacKinnon DP, Elliot DL, et al. Effects of a prevention program on anabolic steroid use. *Arch Pediatr Adolesc Med.* 1996;150(7):713–721.
