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editorial-office@sciformat.ca

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EFFECTS OF ANABOLIC-ANDROGENIC STEROIDS ON MENTAL HEALTH - COMPREHENSIVE REVIEW

Mateusz Kosowski
Independent Public Healthcare Institution in Koło, Koło, Poland
ORCID ID: 0009-0001-0811-5326

Wiktor Śliwiński *(Corresponding Author, Email: wiktorsliwinski2000@gmail.com)*
The Independent Public Healthcare Institution of the Ministry of the Interior and Administration in Łódź, Łódź, Poland
ORCID ID: 0009-0003-3584-9776

Karolina Zarówna
District Hospital Complex in Oleśnica, Oleśnica, Poland
ORCID ID: 0009-0003-6066-7667

Daniel Chołuj
The Independent Public Healthcare Institution of the Ministry of the Interior and Administration in Łódź, Łódź, Poland
ORCID ID: 0009-0009-1445-6336

Jakub Marciniak
University Clinical Hospital No. 2 of the Medical University of Łódź, Łódź, Poland
ORCID ID: 0009-0004-5675-6963

Jakub Mazur
Voivodeship Combined Hospital in Kielce, Kielce, Poland
ORCID ID: 0009-0005-1089-9210

Weronika Pura
The Independent Public Healthcare Institution of the Ministry of the Interior and Administration in Łódź, Łódź, Poland
ORCID ID: 0009-0007-2889-667X

Dominika Matecka
Józef Struś Municipal Hospital, Multispecialty Medical Centre, Poznań, Poland
ORCID ID: 0009-0000-4644-5943

ABSTRACT

This review summarizes current information on the mechanisms and adverse outcomes of anabolic-androgenic steroid (AAS) usage. Chronic use of AAS induces significant structural and functional changes in the brain, especially in the prefrontal cortex, hippocampus, and amygdala, through monoaminergic and GABAergic dysregulation, alterations in the tryptophan-kynurenine pathway, and neuroinflammation. Moreover, abuse of AAS causes remodeling of fronto-limbic circuits, as well as suppression of the hypothalamic-pituitary-gonadal (HPG) axis. Clinically, excessive AAS consumption is associated with psychotic, depressive, and anxiety-related symptoms, cognitive decline, and increased aggressive behaviors.

KEYWORDS

Mental Health, Anabolic-Androgenic Steroids, Psychiatry, Neuroinflammation

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Introduction

Anabolic–androgenic steroids (AAS) are increasingly acknowledged as substances that can trigger severe psychiatric complications, with anxiety and psychotic symptoms being among the most prominent neuropsychiatric effects linked to their use. The rising incidence of anabolic steroid misuse among athletes, bodybuilders, and recreational fitness users underscores the need for heightened clinical awareness of these associated psychiatric outcomes.

Structural brain changes during extensive AAS administration

Long-term anabolic-androgenic steroid (AAS) drug use is linked to large changes in brain structure and accelerated brain aging. Studies using machine learning models trained on brain MRI data show that prolonged AAS users experience increased "brain age gap" (BAG)—the difference between chronological age and predicted brain age estimated based on structural parameters. This accelerated aging is particularly pronounced in frontal and insular regions which are the most crucial for executive function, decision making and emotional control (Bjrnebekk et al., 2020). Subsequent structural alterations induced by AAS seem to be a result of several interacting pathways. Long-term AAS exposure damages key neurotransmitter systems, including serotonin and dopamine pathways, which regulate emotions, motivation and behavior. Such neurochemical changes are associated with the established mood impairment, severe aggression and cognitive impairment for AAS users (Chisari et al., 2025). AAS-induced variants in GABAergic (inhibitory) neurotransmission were observed with changes in GABA receptor subunit expression and synaptic transmission characteristics (Penatti et al., 2009).

Neuroinflammation is another path leading to AAS-induced brain damage. Experimental work showed that constant exposure to AAS raises neuroinflammatory markers and results in changes in microglial activation patterns over time (e.g., Namjoshi et al., 2016). The mechanisms underlying this neurotoxicity are oxidative stress and programmed cell death (apoptosis), which cause loss of neurons and abnormal function at the neuronal level (Pomara et al., 2014). Histological studies, on the cellular level, show abnormal pyramidal neurons displaying a shrunken phenotype, deep stained nuclei, and flame-like projections, predominantly in the prefrontal cortex and hippocampus. Immunohistochemical study of AAS treated animals shows widespread astrocytic activation (glial scar formation) as well as neuroinflammatory mechanisms (Mohamed et al., 2024). Molecular features include the upregulation of overexpressed brain-derived microRNAs post chronic AAS use, reminiscent of the pattern observed in aging and cocaine abuse (Sessa et al., 2020).

In addition, chronic exposure to AAS amplifies neuropathological reactions to traumatic brain injury. In animal models of repetitive mild traumatic brain injury, the effects of AAS treatment had large magnitude and greater degree of axonal injury and microgliosis (microglial activation) than that of injury only although behavioral changes were similar (Namjoshi et al., 2016).

Table 1.

Brain Structure	Primary Change	Severity	Functional Impact	Revesibility
Prefrontal Cortex	Atrophy & cortical thinning	Severe	Impaired executive function, Impulse control ↓	Partial
Hippocampus	Pyramidal neuron degeneration	Severe	Memory impairment, Learning deficits ↓	Partial
Amygdala	Structural enlargement	Moderate	Emotional dysregulation, Hyperreactivity ↑	Minimal
Corpus Callosum	White matter damage	Moderate	Impaired interhemispheric communication ↓	Unknown
Anterior Cingulate	Gray matter alterations	Moderate	Altered emotional/cognitive processing	Unknown
Cortical Regions (frontal, parietal, temporal)	Widespread cortical thinning	Severe	Cognitive decline ↓, Behavioral changes ↑	Partial
Frontoparietal Cortex	Morphometric changes	Moderate	Memory & working memory impairment ↓	Unknown
Putamen	Volume reduction	Moderate	Motor & behavioral changes	Partial
Dentate Gyrus	Neuronal degeneration	Moderate	Learning & memory impairment ↓	Unknown
Entire Brain	Accelerated brain aging	Severe	Premature cognitive decline, Neurological aging ↑	No recovery

Table 1 presents the primary changes of anatomical brain structures during long term AAS usage. It also includes severity, functional impact and reversibility. ↑- increase, ↓ - decrease (Bjrnebekk et al., 2020), (Hauger et al, 2019), (Mohamed et al, 2024).

Anxiety Symptoms Associated with Anabolic Steroid Use

Anxiety is among the most commonly described psychiatric symptoms seen in athletes that use anabolic-androgenic steroids (AAS). Studies concerning the role of AAS on psychiatric well-being have uncovered a clear link between steroid abuse and increased levels of anxiety. In a cross-sectional study of male bodybuilders using AAS, anxiety scores were significantly higher than those of matched controls, (with Beck Anxiety Inventory (BAI) coefficient significantly below 0.0001 among AAS users) (Karagun & Altug, 2024). The absence of anxiety symptoms among patients in the control group and mild anxiety in 7 patients in the AAS group indicates important distinctions in the prevalence of anxiety in the two groups. Previous studies on psychiatric complications among bodybuilding populations have reported that an estimated one-third of anabolic steroid users scored on the Hamilton Anxiety Rating Scale (HAMA) between the moderate and severe ranges for anxiety (J. Amaral et al., 2022). Significantly, the current study also observed no association between psychiatric symptoms and estimated weekly AAS dosage, proving that anxiety might be driven mainly by individual susceptibility rather than a simple dose-response relationship. Furthermore, there was a relationship between anxiety scores and the length of AAS use, implying that long-time users might show different anxiety profiles from shorter time-term use of AAS (J. Amaral et al., 2022).

Mood disorders associated with anabolic steroids

Depression emerges as one of the most prevalent psychiatric complications associated with AAS use, with research documenting both acute mood alterations during active use and depressive episodes during withdrawal phases. (Karagun & Altug, 2024).

The mechanisms underlying AAS-induced depression involve the modulation of tryptophan metabolism and serotonin and dopamine pathways. Aggression, anxiety, depression, personality disorders, and psychosis observed on withdrawal of AAS or with large doses can be caused by decreased serotonin synthesis due to

tryptophan-2,3-dioxygenase (TDO) induction on withdrawal, excess tryptophan inhibiting the two enzymes of serotonin synthesis, and increased cerebral levels of neuroactive kynurenines (Badawy, 2018). There may be a link between sex steroid hormones and serotonergic neurotransmission, which provides crucial mechanistic insight into AAS-induced depression. Testosterone's effects on serotonergic regulation depend on its conversion to estradiol via aromatase, which influences the expression of serotonin transporter (SERT) and 5-HT2A receptors in critical brain regions (Wei & Chiu, 2025). When AAS use produces supraphysiological hormone concentrations with dysregulated aromatization, this fundamentally disrupts the normal balance of serotonergic signaling that underlies mood homeostasis.

One study compared three groups: competitive athletes with doping, competitive athletes without doping and persons with no sports activities. The doping cohort showed higher rates of depression and emotional and physical neglect during childhood among males. They were also less optimistic, perceived less social support, and displayed personality traits marked by lower extraversion and higher neuroticism (Berger et al., 2024). People who use AAS and have personality disorders are much more likely to report aggression and suicidal thoughts than those without these disorders. (Brjesson et al., 2020). This raises key questions about whether AAS worsens existing vulnerabilities, attracts certain personality types, or both. One of the most serious effects of AAS-related depression is the increased risk of suicidal thoughts and self-harm. Large health record studies show that testosterone is linked to both major depression and suicide attempts, while post-cycle therapy may reduce suicidal thoughts by about 50%, suggesting that treating hormonal imbalance can lower this risk. (Nackeeran et al., 2022).

Clinical Presentation and Magnitude of Aggression and Irritability in AAS Users

Quantitative meta-analytic evidence provides empirical documentation of AAS-induced aggression effects in controlled settings, though effect sizes remain modest when examining diverse measurement approaches. A comprehensive meta-analysis of 12 randomized controlled trials encompassing 562 healthy males found that AAS administration produced a statistically significant increase in self-reported aggression, with a small effect magnitude (Hedges $g = 0.171$, 95% CI: 0.029-0.312) (Chegeni et al., 2021). When restricting analysis to acute AAS administration, the effect remained significant under fixed-effects modeling ($g = 0.291$, 95% CI: 0.014-0.524, $p = .014$), suggesting that short-term exposure produces observable increases in subjective aggressive cognitions and behavioral inclinations (Chegeni et al., 2021). However, critical limitations emerged when analyzing observer-rated aggression or examining higher doses (exceeding 500 mg) and longer-term administration (3 days to 14 weeks), where effects were not replicated, indicating substantial heterogeneity in how aggression manifests across measurement modalities and dosing paradigms. Descriptive accounts from users indicate cognitive and emotional perturbations accompanying aggression, with subjective experiences of mental clarity reduction and emotional lability that may partially explain the disconnect between observer-reported and self-reported aggression in controlled trials. Polysubstance use patterns further complicate aggression manifestation in AAS users, as some individuals combine AAS with other substances producing independent behavioral dyscontrol, and chronic AAS use generates psychiatric complications including labile mood, impaired impulse control, and heightened violence (Mhillaj et al., 2015).

Female weightlifters with AAS use history demonstrated significantly higher levels of aggressive traits and antisocial problems as well as anxiety and depression (Scarth et al., 2025). Notably, AAS dependence symptoms correlated strongly with aggressive phenotypes: tolerance to AAS effects showed robust correlation with aggressive behavior ($r = 0.79$, $p < 0.001$), while broader dependence on AAS was associated with marked increases in both verbal and physical aggression (Esteves et al., 2024). These associations persisted even after accounting for baseline behavioral differences, suggesting direct pharmacological mechanisms underlying aggression amplification rather than pre-existing personality traits driving both substance use and behavioral dyscontrol.

Impairement of cognitive functions associated with anabolic-androgenic steroids

Prolonged use of high-dose anabolic-androgenic steroids (AAS) is associated with significant impairments in cognitive function across multiple domains (Bjrnebekk et al., 2019). Research has demonstrated that individuals with long-term AAS exposure perform substantially worse on tasks measuring speed of processing, working memory, and problem-solving abilities compared to non-users, with these deficits persisting even after controlling for age, education, and verbal intelligence (Bjrnebekk et al., 2019). The cognitive decline appears to be dose and duration-dependent, as longer periods of AAS use correlate with particularly pronounced impairments in memory function (Bjrnebekk et al., 2019). It is notable across multiple

domains, including significant effects on speed of processing, working memory, and problem solving. Within AAS users themselves, individuals with better memory and working memory performance demonstrate thicker frontoparietal cortex and larger medial frontal surface area, suggesting a brain-behavior relationship (Bjrnebekk et al., 2019). Of all the AAS, trenbolone was viewed as the greatest risk of agressive behaviors. Among its users there was reported an extreme shift in risk profile for psychosocial harms, particularly increased aggression and violent behaviour, as well as impulsivity regulation issues (Piatkowski et al., 2023).

Supraphysiological AAS doses trigger significant alterations in multiple neurotransmitter systems critical for learning and memory, including reductions in acetylcholine, dopamine, norepinephrine, glutamate, and serotonin levels in brain regions essential for cognitive processing (Dragica et al., 2018). These neurochemical changes are accompanied by decreased production of growth factors such as nerve growth factor (NGF) and brain-derived neurotrophic factor (BDNF), which further compromise cognitive capacities (Dragica et al., 2018). Structural brain imaging reveals that chronic AAS exposure associates with evidence of deviant brain aging, increased brain age gap relative to chronological age, and reduced cortical thickness in frontoparietal regions critical for memory and executive function (Bjrnebekk et al., 2020). Individuals dependent on AAS demonstrate significantly worse performance on measures of emotional and cognitive aspects of social understanding (Vaskinn et al., 2020). Importantly, cognitive deficits in processing speed and working memory appear to develop early during AAS use in young men, though these specific impairments may partially stabilize by middle age, raising questions about whether certain deficits plateau rather than continue to worsen (Kaufman et al., 2024).

Psychotic Symptoms and AAS Abuse

The emergence of psychotic symptoms is among the most serious psychiatric consequences of anabolic steroid misuse. These symptoms—including delusions, hallucinations, and disorganized thinking—may occur during active use or persist after cessation. Psychotic symptoms in AAS users commonly involve persecutory delusions, auditory or visual hallucinations, and disorganized thought. Research on male athletes has shown higher ocurrence of psychotic symptoms and increased suicidal ideation among steroid users compared with non-users (Hussain et al., 2022). These effects are thought to result from supraphysiological androgen levels disrupting dopaminergic and related neurotransmitter systems involved in psychosis. The psychotic effects of anabolic steroids range from mild symptoms to severe, potentially life-threatening conditions.

One case report described a 33-year-old man who developed severe aggression, hostility, and destructive impulses after using high doses of injectable testosterone propionate, testosterone cypionate, and trenbolone acetate (up to 200 mg/day) (Khoodoruth & Khan, 2020). His presentation included labile affect, flight of ideas, and persecutory delusions, consistent with steroid-induced delirium with psychotic features. He improved after treatment with haloperidol and quetiapine. Similar reports indicate that anabolic steroids can cause anxiety, agitation, visual hallucinations, bizarre behavior, delirium, and psychosis (Talabaki et al., 2024). Another report described a patient treated for AAS abuse who developed intense psychomotor agitation with verbal and physical aggression toward family members after high-dose steroid use (Remelhe et al., 2023). In the weeks before psychiatric evaluation, he experienced increasing anxiety, persecutory thoughts, aggression, and repeated household conflicts, eventually requiring emergency psychiatric assessment.

Withdrawal Syndrome and Dependence

Anabolic-androgenic steroid dependence is formally recognized and classified in the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5), representing one of the more recent formal acknowledgments of AAS as a substance of abuse with established diagnostic criteria (Piacentino et al., 2015). Dependence on AAS manifests as a constellation of behavioral, cognitive, and physiological symptoms. The core features of AAS dependence include tolerance, withdrawal symptoms upon cessation, using larger amounts or for longer periods than intended, persistent desire or unsuccessful attempts to cut down use, substantial time spent obtaining or using the substance or recovering from its effects, continued use despite knowledge of adverse consequences, and giving up important social, occupational, or recreational activities in favor of AAS use (Carter & Boardley, 2024).

The mechanisms underlying AAS dependence operate through multiple interconnected neurobiological pathways. Recent evidence demonstrates that AAS do not produce their addictive effects directly through androgen receptor stimulation, but rather through an indirect mechanism mediated by endogenous opioid signaling within dopaminergic neurons (Bontempi & Bonci, 2020). Beyond the dependence, chronic AAS use produces profound neuroendocrine changes affecting the hypothalamic-pituitary-gonadal (HPG) axis through

negative feedback suppression. During AAS use, supraphysiological androgen levels suppress luteinizing hormone (LH) and follicle-stimulating hormone (FSH) through classical hypothalamic-pituitary feedback inhibition, leading to suppression of endogenous testosterone production and cessation of spermatogenesis (Rasmussen et al., 2016).

This endocrine dysregulation establishes the biological foundation for withdrawal symptomatology, which is characterized by a biphasic temporal pattern. It comprises an initial hyperadrenergic phase characterized by opioid-like withdrawal symptoms, followed by a prolonged depressive phase characterized by mood disturbance and intense craving. The acute withdrawal phase occurs within days to weeks following AAS cessation and is characterized by symptoms resembling opioid withdrawal, likely mediated through the endogenous opioid system disruption, such as anxiety, restlessness, irritability, tachycardia, tremors, and insomnia (Remelhe et al., 2023). This acute phase is typically relatively brief, lasting from several days to a few weeks, after which the clinical picture shifts dramatically to the prolonged depressive phase. During this phase, symptoms such as fatigue, muscle and joint pain, insomnia, anxiety, and depression may persist for weeks to months, with depression being the predominant manifestation (Remelhe et al., 2023). The depressive manifestations during withdrawal can be severe enough to pose suicide risk (Grant et al., 2023).

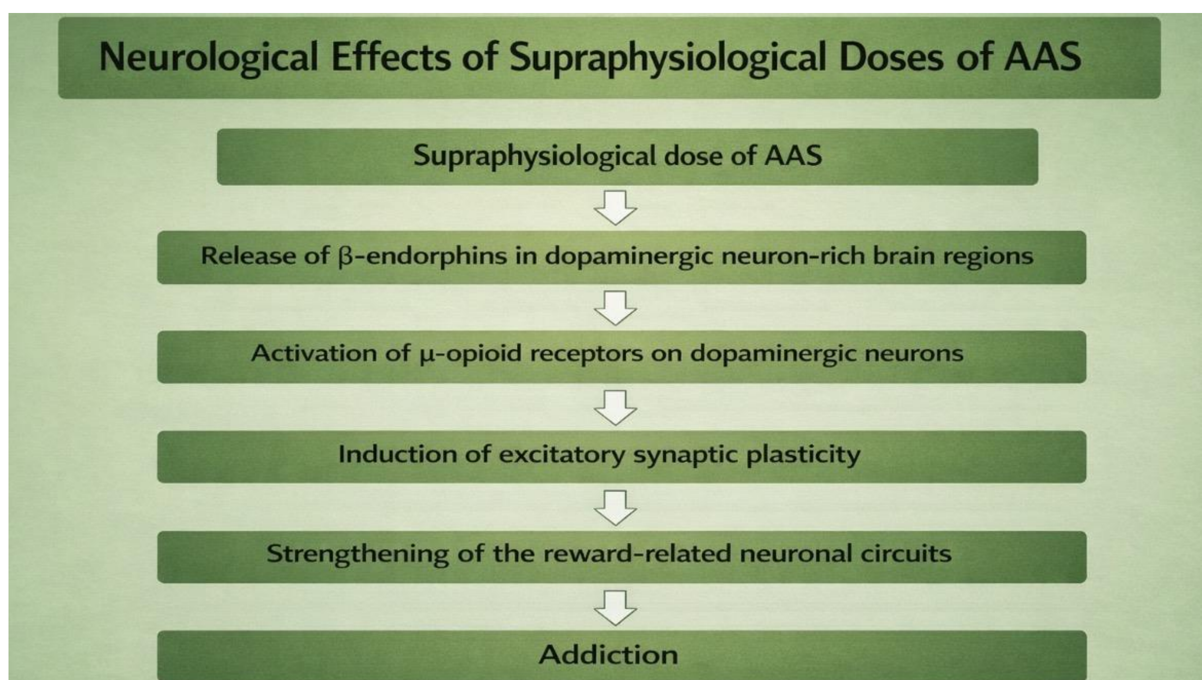

Fig. 1.

Figure 1 presents the mechanism by which anabolic steroids cause addiction (Bontempi & Bonci, 2020).

Treatment of AAS induced psychiatric symptoms and withdrawal syndrome

Management of anxiety and psychotic symptoms due to anabolic steroids typically includes immediate intervention followed by sustained long-term treatment. Haloperidol has also been referred to as effective in treating acute agitation, but the literature of this type has been limited (Remelhe et al., 2023). This, as may quetiapine, may have an effect to reduce psychotic symptoms (Talabaki et al., 2024). Although benzodiazepines can be used as well, they have limited use for AAS-associated disorders. One clinical report suggested successful treatment of anxiety and suicidal thoughts using fluoxetine despite ongoing androgenic-anabolic steroid use, suggesting that serotonergic medications may be effective for mood and anxiety disturbances attributable to steroid exposure (J. M. X. Amaral et al., 2020). The degree and continuation of withdrawal-induced depression can compete with or surpass depressive episodes in other substance use disorders. Post-cycle therapy (PCT)—medicine that attempts to restore normal testicular function and limit hypogonadal symptoms—is at least partially beneficial in reducing withdrawal symptoms. Among 470 men using AAS, PCT use correlated significantly with less withdrawal symptomatology (according to a report), including a 50% reduction in suicidal thoughts in those employing post-cycle therapy compared with no

intervention (Grant et al., 2023).). However, this incomplete symptom resolution highlights that depression during AAS withdrawal often requires comprehensive management beyond simple hormone replacement. For long-term management, discontinuation of AAS remains essential, combined with appropriate treatment of withdrawal symptoms and any coexisting psychiatric disorders.

Summary

Long-term use of anabolic-androgenic steroids (AAS) induces severe neurobiological and psychiatric complications, involving accelerated brain aging and structural degradation in areas responsible for executive function and memory, e.g., prefrontal cortex and hippocampus. These neurobiological alterations are mediated by neuroinflammation, oxidative stress and the "glial scarring" process with the consequent dysregulation of the serotonin, dopamine, as well as GABAergic neurotransmitter systems which are significant for emotional stability. Clinically, AAS abuse presents as a wide variety of psychiatric phenotypes such as severe anxiety (which one-third of AAS users experience) and major depression which is more likely to lead to suicide, especially during withdrawal phases. Although controlled studies indicate that aggression increases slightly, high-dose use and dependence are linked to aggressive tendencies such as violence, antisocial behavior, and cognitive impairment of processing speed and working memory. The severity of cases may also reach steroid-induced psychosis or delirium, with hallucinations and delusions leading to an acute psychiatric diagnosis. In addition, AAS dependency is also characterized by a biphasic withdrawal syndrome secondary to hypothalamic-pituitary-gonadal (HPG) axis suppression; an initial hyperadrenergic phase resembling opioid withdrawal is followed by a protracted depressive state. Successful clinical treatment requires a two-pronged approach, employing 2nd generation antipsychotics and SSRIs to alleviate symptoms, but also post-cycle therapy (PCT) to support hormonal homeostasis, associated with a 50% reduction in suicidal ideation. New studies are required to evaluate the adverse effects caused by misusing AAS as it is reported to alter brain structures and their function.

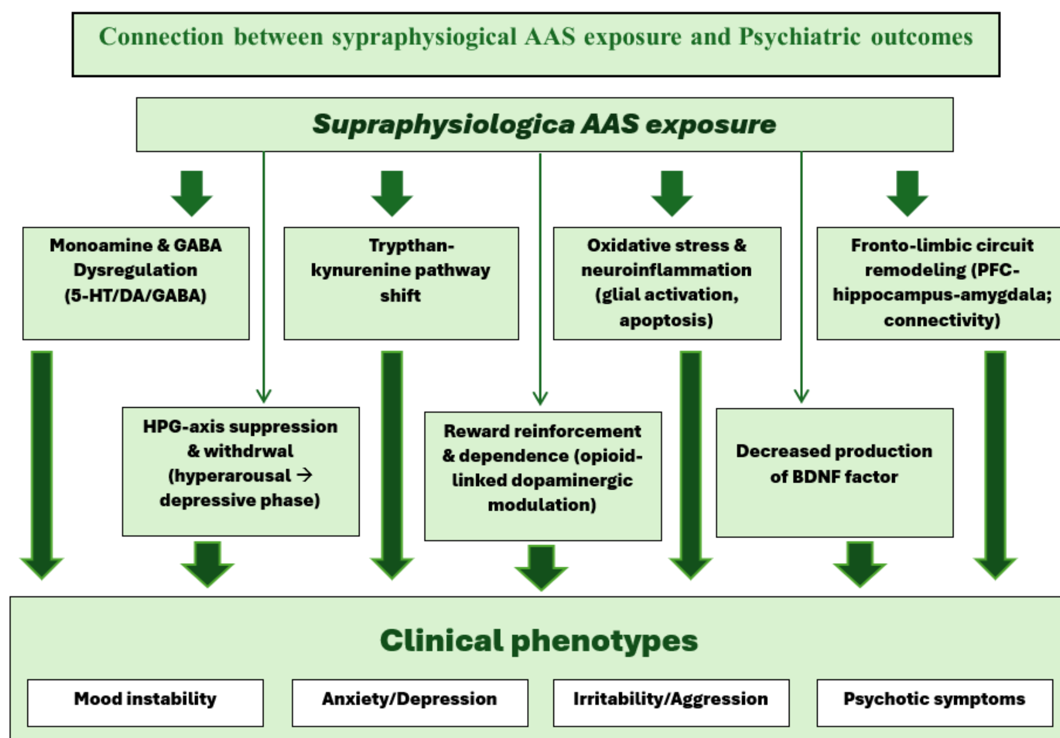

Fig. 2.

Figure 2. proposed model linking supraphysiological AAS exposure to core neurobiological mechanisms and downstream clinical phenotypes. Abbreviations: AAS, anabolic–androgenic steroids; BDNF, brain-derived neurotrophic factor; HPG axis, hypothalamic–pituitary–gonadal axis. Adapted from Chisari et al. (2025), Namjoshi et al. (2016), Dragica et al. (2018), Pomara et al. (2014), Badawy (2018), Bjørnebekk et al. (2020), Hauger et al. (2019), Mohamed et al. (2024), Penatti et al., 2009 and Wei & Chiu, 2025.

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AI: AI was utilized for assistance in refining the academic English language of the manuscript, ensuring clarity, consistency, and adherence to scientific writing standards. 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. In preparing this work, the author(s) used Grammarly for the purpose of checking and improving grammatical correctness. After using this tool, the author(s) have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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