



WRJBHS-24-011

Effects of Anabolic Steroids on Brain Waves and Neurofeedback-Based Treatment Perspectives

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Citation: Lombardo C (2024) Effects of Anabolic Steroids on Brain Waves and Neurofeedback- Based Treatment Perspectives. J Biol & Heal Sci 1: 11.

Abstract

This article explores the neurological and psychological effects of Anabolic-Androgenic Steroids (AAS) and evaluates the potential of neurofeedback as a therapeutic intervention. AAS, widely used for performance and aesthetic enhancement, impact brain mechanisms such as the dopaminergic system, Hypothalamic-Pituitary-Adrenal (HPA) axis, hippocampus and amygdala. These effects result in reduced neuroplasticity, emotional dysregulation and cognitive impairments. Specific steroids like testosterone propionate, Decadurabolin and Stanozolol show distinct effects on brain wave activity, exacerbating symptoms like aggression, anxiety and insomnia. Neurofeedback emerges as a promising treatment for restoring brain wave balance in users discontinuing AAS and for mitigating damage in those who continue use. The article highlights the need for integrated approaches, combining neurofeedback with pharmacological and psychological support and calls for further research to address gaps in understanding long-term AAS effects and optimizing interventions.

Keywords: Anabolic steroids; Neurofeedback; Brain waves; Drugs; Hippocampus; Neural plasticity

Received date: December 15, 2024; Accepted date: December 24, 2024; Published date: December 31, 2024

Introduction

Anabolic-Androgenic Steroids (AAS) are a class of drugs widely used not only for medical purposes but also to enhance athletic performance and aesthetics. While their physiological effects are well- documented, the long-term neurological and psychological consequences remain an expanding area of research. Recent studies have highlighted that the use of anabolic steroids can negatively impact brain waves, with significant implications for individuals' mental and cognitive health. This article aims to explore the brain mechanisms affected by anabolic steroids, link them to specific drugs and propose neurofeedback as a therapeutic strategy in two distinct scenarios: Recovery from dependency and mitigation of negative effects for those who continue usage.

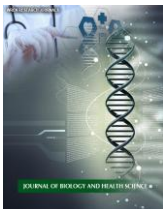
Brain mechanisms affected by anabolic steroids

Anabolic steroids directly modulate the dopaminergic

system, which is crucial for the brain's reward circuitry. AAS intake temporarily increases dopamine availability in the nucleus accumbens, generating feelings of pleasure and satisfaction that can reinforce repetitive behaviors and dependency [1].

However, prolonged use induces desensitization of dopamine receptors, causing reduced dopaminergic activity and contributing to symptoms such as anhedonia, apathy and depression. These effects are also reflected in brain waves, with a marked decline in alpha and beta waves, which regulate wakefulness and concentration [2].

Additionally, the reward system becomes progressively hyperactive, fueling impulsive behaviors and compulsive AAS-seeking akin to the mechanisms of substance addiction. Studies suggest that dysregulation of the dopaminergic system leads to reduced behavioral inhibition, increasing the risk of aggressive conduct [3].



Alterations of the Hypothalamic-Pituitary-Adrenal Axis (HPA)

AAS profoundly affect the HPA axis, a central component in stress regulation and hormonal homeostasis. Chronic AAS use reduces endogenous cortisol production, disrupting the normal negative feedback of the axis [4]. This translates into a decrease in delta waves during deep sleep, which are critical for brain recovery and stress regulation.

These effects manifest as symptoms such as irritability, circadian rhythm disruptions and increased vulnerability to stress. Furthermore, low cortisol compromises the brain's ability to respond adequately to stress stimuli, rendering individuals more susceptible to anxiety and depression disorders [4]. HPA axis dysfunction is also linked to reduced neuroplasticity, further aggravating cognitive impairments.

Effects on the hippocampus and neural plasticity

The hippocampus, a critical structure for memory and learning, is particularly vulnerable to the effects of anabolic steroids. Preclinical studies in animal models demonstrate that AAS use significantly reduces hippocampal neurogenesis, impairing the formation of new neural connections [2]. While theta wave alterations critical for memory processing and consolidation have been observed in individuals taking testosterone propionate, human-specific data remain sparse, limiting the generalizability of these findings. Additionally, AAS impair Long-Term Potentiation (LTP) a key mechanism for synaptic strengthening essential for learning [3]. Although these changes are associated with cognitive deficits, including reduced learning capacity, direct comparisons with other neurotoxic substances or conditions, such as chronic stress, are lacking.

Prolonged AAS use may also increase oxidative stress in the hippocampus due to high concentrations of steroid metabolites, a mechanism linked to neurodegenerative processes. However, the specific pathways driving these changes in human subjects remain underexplored. Targeted interventions, such as antioxidant therapies or neurofeedback techniques, could hold potential for mitigating these effects, but require further investigation. Incorporating longitudinal studies on AAS users would provide essential insight into the progression of hippocampal damage and its reversibility.

Effects on the amygdala and emotional regulation

The amygdala, a region involved in emotion regulation, is also affected by AAS. The overload of androgens disrupts the excitatory inhibitory balance, leading to hyperactivation of the amygdala [4]. This phenomenon has

been associated with increased aggression, emotional reactivity and difficulty controlling impulses [5].

Negative effects on the amygdala also include reduced functional connectivity with the prefrontal cortex, a region significant for emotional control and planning. This neurofunctional imbalance is reflected in hyperactive gamma waves observed in individuals with psychotic symptoms or impulsive behaviors during chronic AAS use [6].

Association between specific steroids and brain effects

Testosterone propionate

Testosterone propionate is one of the most commonly used AAS. Its prolonged use has been associated with a reduction in alpha waves, crucial for mental calmness and relaxation [2]. Negative effects include anxiety, irritability and cognitive deficits, also evident during withdrawal periods.

Decadurabolin (nandrolone decanoate)

Decadurabolin is known for its powerful effects on muscle strength but presents significant risks to the brain. Recent research has demonstrated an increase in gamma waves, often indicating a state of cerebral hyperactivation related to psychotic symptoms and insomnia [3]. Additionally, chronic use compromises delta waves, essential for brain recovery during sleep.

Stanozolol

Stanozolol, commonly known as Winstrol, is a synthetic anabolic steroid derived from dihydrotestosterone. It is used to enhance muscle definition and athletic performance but presents significant negative effects on the brain. Recent studies have shown that chronic stanozolol use alters beta waves, associated with alertness and focus [5]. These changes can lead to irritability, increased aggression and difficulty concentrating. Additionally, stanozolol interferes with synaptic plasticity in the amygdala, increasing the risk of dysfunctional emotional reactions.

What is neurofeedback?

Neurofeedback is a non-invasive therapeutic technique that enables self-regulation of brain activity through real-time monitoring of brain waves. Using EEG sensors placed on the scalp, patients receive immediate feedback in the form of visual or auditory signals, guiding them to modify specific brain activity patterns. This approach is particularly effective in treating disorders related to brain dysregulation, such as anxiety, insomnia and addictions, promoting greater neuroplasticity and better cognitive function balance.



Neurofeedback as a treatment modality

Case 1: Individuals wishing to discontinue steroid use

For individuals choosing to discontinue steroid use, neurofeedback represents an opportunity to restore brain wave balance. During withdrawal periods, symptoms include depression, apathy and cognitive deficits. Neurofeedback can be tailored to address these specific issues:

- **Rebalancing alpha and theta waves:** Stimulating alpha wave production promotes a state of calm and focus. Simultaneously, enhancing theta waves supports neural plasticity and hippocampal recovery. This approach is supported by Thibault et al., who demonstrated neurofeedback's effectiveness in normalizing brain patterns altered by substances.
- **Personalized monitoring:** Each neurofeedback session is calibrated to monitor and correct imbalances specific to the individual, allowing gradual improvement in emotional and cognitive regulation.
- **Pharmacological support:** During the initial withdrawal phase, drugs such as Clomid (clomiphene citrate) or Tamoxifen may be used to stimulate endogenous testosterone production. This pharmacological treatment synergizes with neurofeedback, reducing withdrawal symptoms and improving emotional stability [7].
- **Long-term effects:** With continuous treatment, patients report significant improvements in sleep quality, reduced anxiety and a return to normal neuroplasticity [8].

Case 2: Individuals continuing steroid use

For individuals choosing to continue AAS intake, neurofeedback can be implemented as a mitigative measure to reduce negative effects on the brain. This approach aims to minimize harm without requiring complete cessation:

- **Reducing gamma waves:** Hyperactive gamma waves, often associated with irritability and insomnia, can be reduced through regular neurofeedback sessions. This intervention promotes better emotional control and reduces acute symptoms [7].
- **Preventing long-term damage:** Continuous monitoring of brain waves allows for early detection of significant imbalances, preventing irreversible damage to the central nervous system.
- **Synergy with drugs for cyclical use:** During cyclical use periods, drugs such as Gonasi (human chorionic gonadotropin) may be employed to maintain balanced HPA axis functioning. This combined approach reduces

the likelihood of permanent cerebral alterations [5].

- **Psychosocial support:** For these individuals, integrating neurofeedback with psychological counseling provides an additional level of support, helping patients maintain a balance between perceived benefits and the risks of AAS use.

Discussion

The use of anabolic steroids presents a multidimensional challenge to global health, with profound impacts on physiology, neurochemistry and behavior. The mechanisms of cerebral alteration involve the dopaminergic system, the HPA axis, the hippocampus and the amygdala, with deleterious effects ranging from memory loss to behavioral and psychiatric disorders.

Evidence demonstrates that targeted interventions like neurofeedback offer significant potential in mitigating these effects. Neurofeedback, in particular, emerges as an innovative and customizable tool that addresses not only cerebral alterations but also the emotional and behavioral aspects related to steroid use.

However, gaps remain in the scientific literature, particularly regarding the long-term effects of AAS on the human brain and the comparative efficacy of neurofeedback vs. other therapeutic approaches. Longitudinal and randomized studies are necessary to consolidate knowledge and optimize treatment protocols.

Conclusion

The use of anabolic steroids presents a multidimensional challenge to global health, with profound impacts on physiology, neurochemistry and behavior. The mechanisms of cerebral alteration involve the dopaminergic system, the HPA axis, the hippocampus and the amygdala, with deleterious effects ranging from memory loss to behavioral and psychiatric disorders.

Evidence demonstrates that targeted interventions like neurofeedback offer significant potential in mitigating these effects. Neurofeedback, in particular, emerges as an innovative and customizable tool that addresses not only cerebral alterations but also the emotional and behavioral aspects related to steroid.

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