MARCINKOWSKA, Julia, MURAS, Mateusz Szymon, LORENC, Tomasz, MICHALIK, Maciej, MARCINKOWSKI, Krzysztof, MIKSZTA, Jakub and MIKSZTA, Natalia. The Impact of Anabolic Androgenic Steroids on Organ Failure in Bodybuilders - a Review of Actual Literature. Quality in Sport. 2025;41:60236. eISSN 2450-3118.
https://doi.org/10.12775/QS.2025.41.60236
https://apcz.umk.pl/QS/article/view/60236

The journal has been 20 points in the Ministry of Higher Education and Science of Poland parametric evaluation. Annex to the announcement of the Minister of Higher Education and Science of 05.01.2024. No. 32553.
Has a Journal's Unique Identifier: 201398. Scientific disciplines assigned: Economics and finance (Field of social sciences); Management and Quality Sciences (Field of social sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 r. Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398.
Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 11.04.2025. Revised: 25.04.2025. Accepted: 06.05.2025. Published: 11.05.2025.

The Impact of Anabolic Androgenic Steroids on Organ Failure in Bodybuilders – a review of actual literature

Julia Marcinkowska
Śląski Uniwersytet Medyczny
https://orcid.org/0009-0005-7006-4303
Mateusz Szymon Muras
Śląski Uniwersytet Medyczny
https://orcid.org/0009-0005-1392-3773
Tomasz Lorenc
Śląski Uniwersytet Medyczny
https://orcid.org/0009-0008-9902-469X
Maciej Michalik
Śląski Uniwersytet Medyczny
https://orcid.org/0009-0009-7799-7252
Krzysztof Marcinkowski
Śląski Uniwersytet Medyczny
https://orcid.org/0009-0002-5759-7285
Jakub Mikszta
Uniwersytet Opolski
https://orcid.org/0009-0000-4194-9915
Natalia Mikszta
Śląski Uniwersytet Medyczny
https://orcid.org/0009-0003-8444-7650

Abstract

Anabolic-Androgenic Steroids (AAS), despite their therapeutic applications in the management of selected medical conditions, are increasingly being misused for non-medical purposes—particularly among bodybuilders and individuals seeking to enhance their physique. This review article examines the impact of chronic AAS administration on the functionality of selected human organs and physiological systems.

Based on current clinical and preclinical evidence, the biological mechanisms of AAS action are discussed, with particular emphasis on their affinity for androgen receptors and modulation of intracellular signaling pathways. The paper presents robust evidence of the adverse effects associated with AAS use, including cardiomyopathies, renal dysfunction, neuropsychiatric disorders (notably increased aggression and depressive symptoms), hepatotoxicity, and metabolic disturbances.

Special attention is given to the potential mechanisms underlying AAS-induced toxicity, as well as the reversibility of the observed pathological alterations. The findings underscore the necessity for intensified health education and systematic monitoring of individuals using AAS, due to the elevated risk of irreversible, multisystem complications.

Keywords: anabolic androgenic steroids; depression; left ventricular dysfunction; acute kidney injury

Introduction

The use of anabolic-androgenic steroids (AAS) has become a widespread phenomenon not only among professional athletes but also among amateur bodybuilders and individuals seeking to enhance their physical appearance. Although AAS are approved for medical use in the treatment of specific conditions such as hypogonadism and cachexia, their non-therapeutic application is associated with numerous adverse effects, which may result in irreversible organ damage and systemic dysfunction [1].

The pharmacodynamic mechanism of AAS primarily involves binding to androgen receptors, leading to enhanced protein synthesis and skeletal muscle hypertrophy. However, excessive stimulation of the hypothalamic-pituitary-gonadal axis, as well as direct cytotoxic effects on various tissues, can induce pathological alterations in the liver, heart, kidneys, and nervous system [2]. Accumulated epidemiological and clinical data indicate a significant correlation between long-term AAS use and the occurrence of conditions such as hypertrophic

cardiomyopathy, hepatocellular necrosis, renal failure, and psychiatric disturbances, including depression and aggression [3][4].

The objective of this review article is to provide a comprehensive analysis of the available scientific data regarding the effects of anabolic steroids on selected organs and physiological systems in the human body. The review incorporates clinical observations, preclinical studies, and toxicological literature in order to elucidate the potential mechanisms of tissue injury, the extent of reversibility of these effects, and the broader health consequences for individuals engaging in chronic and unsupervised AAS use.

Biological Mechanism of Action of Anabolic-Androgenic Steroids (AAS)

Anabolic-androgenic steroids (AAS) are synthetic derivatives of testosterone that exhibit high affinity for androgen receptors (AR), thereby exerting complex effects on various tissues in the human body. Their activity encompasses both anabolic effects (e.g., stimulation of protein synthesis and skeletal muscle hypertrophy) and androgenic effects (e.g., development of secondary sexual characteristics and modulation of reproductive function) [5].

Binding to Androgen Receptors (AR):
AAS diffuse across the cell membrane and bind to cytoplasmic androgen receptors. The resulting ligand–receptor complex undergoes conformational activation and translocates into the cell nucleus, where it binds to specific DNA sequences known as androgen response elements (AREs), thereby regulating the transcription of genes involved in cellular growth and differentiation [6].

Conversion to Dihydrotestosterone (DHT):
In certain tissues, such as the prostate gland and the skin, testosterone is enzymatically converted to dihydrotestosterone (DHT) by 5α-reductase. DHT possesses a higher binding affinity for ARs than testosterone, which amplifies its androgenic potency [7].

Aromatization to Estradiol:
A fraction of testosterone molecules undergo aromatization, resulting in the formation of estradiol. Estradiol activates estrogen receptors and plays a critical role in the regulation of bone mass; however, it may also contribute to adverse effects such as gynecomastia [8].

Nongenomic Effects:
AAS are also capable of eliciting rapid cellular responses independent of gene transcription, mediated by the activation of membrane-bound receptors such as GPRC6A. These nongenomic effects include intracellular calcium fluxes and activation of kinase signaling pathways [9].

Influence on Satellite Cells and the Wnt/β-Catenin Pathway:
Anabolic steroids stimulate the activity of satellite cells in skeletal muscle, promoting their proliferation and differentiation into myotubes. Concurrently, AAS favor the myogenic over

the adipogenic lineage commitment of mesenchymal stem cells by activating the Wnt/β-catenin signaling pathway [10].

Cardiovascular System Disorders

Studies conducted since 2020, including meta-analyses, provide growing evidence for the detrimental impact of anabolic-androgenic steroid (AAS) use on the cardiovascular system.

According to a 2024 meta-analysis by Guida et al., which included 17 studies involving 1,023 athletes, long-term AAS administration is associated with interventricular septal thickening, reduced left ventricular ejection fraction (LVEF), and impaired global longitudinal strain. Diastolic dysfunction was also observed, as indicated by a decreased E/A ratio and elevated E/e' index [11]. These findings have been corroborated by additional studies. For example, a 2023 study by Abdullah et al. demonstrated that both current and former long-term AAS users exhibit significant biventricular cardiomyopathy, characterized by reduced LVEF and right ventricular dysfunction [12]. Furthermore, a 2023 review confirmed the association between AAS use and the development of cardiomyopathy, emphasizing the need for further investigation in this domain [13].

In a 2024 study by Frisenberg et al., conducted as part of the Fitness Doping in Denmark (FIDO-DK) project and involving 164 participants—including active and former AAS users as well as non-users—cumulative AAS exposure was identified as an independent predictor of coronary artery calcification and the presence of non-calcified atherosclerotic plaques in males [14]. Similarly, research by Tungesvik et al. demonstrated that chronic AAS use is significantly associated with reduced carotid artery reactivity and diminished flow-mediated dilation (FMD), factors contributing to increased cardiovascular event risk [15].

Moreover, AAS use has been linked to a heightened risk of sudden cardiac death (SCD). A study by Christou et al. highlighted the synergistic effect of AAS use and high-intensity physical exertion in amplifying hemodynamic stress. Findings included diffuse myocardial fibrosis, left ventricular dilatation, arrhythmogenic changes, and coronary artery thrombi—pathological features strongly associated with SCD [16].

Nervous System and Mental Disorders

Neuroimaging studies have demonstrated that anabolic-androgenic steroid (AAS) use is associated with a reduction in gray matter volume and cortical thinning in the frontal and parietal lobes [17], as well as an increase in amygdala volume accompanied by decreased functional connectivity between the amygdala and the prefrontal cortex—alterations that may underlie impulsivity and impaired emotional regulation [18]. In vitro experimental research by Zelleroth et al. confirmed that structurally distinct AAS (e.g., testosterone, nandrolone, stanozolol) inhibit neurite outgrowth and reduce neuronal viability, indicating potential

neurotoxic effects [19]. Moreover, increased neuronal apoptosis has been observed, particularly within the hippocampus, which may contribute to mood disturbances and cognitive impairments [20].

AAS also modulate the functioning of key neurotransmitter systems, including the serotonergic, dopaminergic, and glutamatergic pathways. These neurochemical alterations are implicated in behavioral changes such as heightened aggression, emotional lability, and depressive symptoms [21]. Such effects may be mediated by the interaction of AAS with androgen receptors located in limbic structures and the prefrontal cortex.

A 2021 meta-analysis by Chegeni et al. found that administration of AAS to healthy males led to a small but statistically significant increase in self-reported aggression levels [22], suggesting a potential impact of AAS on behavioral regulation mechanisms. Likewise, a 2022 meta-analysis by Nelson et al. indicated that bodybuilders using AAS exhibit a higher likelihood of psychopathic traits and engagement in risky behaviors, including those related to sexual activity and substance use [23], highlighting a possible role of AAS in impulsivity and risk-taking behavior.

In a 2023 study by Karagun et al., involving 25 male AAS users and a control group of 25 non-users, significantly higher scores were observed among AAS users on both the Beck Depression Inventory (BDI) and the Beck Anxiety Inventory (BAI) ($p < 0.0001$) [24]. Within the AAS group, seven individuals exhibited symptoms of depression—four with mild and three with moderate severity—whereas no such cases were reported in the control group. Additionally, the study revealed correlations between lactate dehydrogenase (LDH) levels and BAI scores, creatinine levels and both BAI and BDI scores, as well as estradiol levels and BDI scores [24].

Kidneys Disorders

A review of the literature identifies several mechanisms by which anabolic-androgenic steroids (AAS) may induce kidney injury, including stimulation of the renin–angiotensin–aldosterone system, increased endothelin production, generation of reactive oxygen species (ROS), and overexpression of proinflammatory and profibrotic factors [25][26].

In a 2023 study by Ozkurt et al., renal function was assessed in bodybuilders using both AAS and dietary supplements (AAS+DS) and those using only dietary supplements (DS). The AAS+DS group exhibited significantly higher urinary albumin/creatinine and protein/creatinine ratios compared to the DS group ($p<0.001$ and $p=0.006$, respectively). Although estimated glomerular filtration rate based on creatinine (eGFRcr) was similar between groups, cystatin C-based eGFR (eGFRcys) and the eGFRcys/eGFRcr ratio were significantly lower in the AAS+DS group ($p=0.039$ and $p=0.036$, respectively), suggesting a potential link between early kidney injury and direct AAS use [27].

Furthermore, a 2020 study by Passaro et al. described three cases of long-term AAS use in bodybuilders who developed impaired renal function. Kidney biopsies revealed collapsing focal segmental glomerulosclerosis (FSGS). In two of the cases, rapid deterioration of kidney function necessitated renal replacement therapy [28].

Recent reports, including case studies and systematic reviews, have outlined several pathomechanisms associated with AAS-induced acute kidney injury (AKI). Almukhtar et al. reported four cases involving bodybuilders who consumed AAS in combination with high-protein diets and creatine supplements. All presented with elevated serum creatinine levels (229.84–335.92 μmol/L), and renal biopsies revealed acute tubular necrosis. Although renal function improved after discontinuation of AAS and supplements, two patients showed >30% interstitial fibrosis and tubular atrophy, suggesting potential irreversible damage [29].

Lemiński et al. described a 34-year-old bodybuilder with a history of long-term testosterone and stanozolol abuse who presented with severe lumbar pain suggestive of renal colic and symptoms of AKI. Diagnostic imaging revealed bilateral renal artery thrombosis [30]. Another case, presented by Tarashande and Elyasi, involved a 33-year-old male who experienced oliguria, dark-colored urine, and lower abdominal pain after using oxymetholone. He was diagnosed with rhabdomyolysis-induced acute renal failure, which improved upon cessation of AAS and appropriate medical intervention [31].

The use of AAS in bodybuilding is associated with a risk of rhabdomyolysis—acute skeletal muscle damage that results in the release of muscle contents into the bloodstream. Benjamin et al. described a case of a patient who developed acute rhabdomyolysis with elevated creatine kinase (CK) levels and dysphagia following a 60-day cycle of oxandrolone (Anavar) [32]. Farkash et al. presented the case of a 29-year-old bodybuilder who experienced localized rhabdomyolysis in the deltoid muscle following intensive training and intramuscular AAS injection. MRI revealed edema and increased signal intensity, consistent with muscle injury [33].

Liver Disorders

The use of anabolic-androgenic steroids (AAS), particularly in non-medical and long-term contexts, is associated with the risk of serious hepatological complications. Studies show that AAS can cause liver injury through several mechanisms, including direct hepatocyte toxicity—especially from orally administered 17α-alkylated steroids [34], cholestatic dysfunction, known as anabolic cholestasis, which has been reported in AAS users [35], and peliosis hepatis—a pathological formation of blood-filled cystic spaces within the liver, a rare but documented consequence of steroid use [36]. Additionally, liver tumors, including hepatocellular carcinoma and hepatic adenomas, may develop with long-term AAS use [34].

In a study by Arazi et al., involving active bodybuilders, individuals using AAS had significantly elevated liver enzymes (ALT, AST), as well as abnormal lipid profiles compared

to the control group [35]. A systematic review by Petrovic et al. reported numerous cases of both acute and chronic liver injury among AAS users. Common findings included jaundice, hepatomegaly, and increased total and fractionated bilirubin levels [34]. The literature includes multiple reports of young males developing severe hepatic complications, such as peliosis hepatis and cholestatic hepatitis, following the use of substances like stanozolol or methandrostenolone [36].

It is recommended that AAS users undergo regular medical monitoring, including liver function tests (ALT, AST, ALP, GGT, bilirubin), lipid profile assessments, and abdominal ultrasound imaging. Furthermore, education regarding the risks of using unregulated or contaminated steroid products is essential [34][36].

Metabolic Disorders

The use of anabolic-androgenic steroids (AAS) is associated with an increased risk of metabolic disturbances, such as dyslipidemia, insulin resistance, and abnormalities in glucose metabolism. A meta-analysis by Tenório et al. evaluated the effects of low and moderate doses of AAS on lipid profiles in individuals engaged in resistance training. The analysis of six clinical trials involving 170 participants showed no statistically significant changes in HDL cholesterol levels (-5.62 mg/dL, 95% CI: -12.10 to 0.86, $p = 0.09$) or LDL cholesterol levels (7.76 mg/dL, 95% CI: -9.70 to 25.23, $p = 0.57$). However, these studies demonstrated substantial heterogeneity ($I^2 = 95–97\%$), which may influence the interpretation of results [37].

A literature review by Perry et al. investigated the association between AAS use and cardiovascular disease risk. The findings indicated that AAS use is linked to lipid metabolism disorders, hypertension, and cardiomyopathy, which collectively increase the risk of myocardial infarction and arrhythmias [38].

Tavares et al. conducted a study focusing on the effects of supraphysiological doses of AAS on glucose and insulin levels in male bodybuilders. The results suggest that high doses of AAS may impair carbohydrate metabolism [39]. Similarly, a study by Di Girolamo et al. analyzed the impact of the abuse of insulin, growth hormone, and AAS on selected metabolic parameters in bodybuilders. The findings revealed that misuse of these substances is associated with multiple changes in metabolic markers, including a reduction in HDL cholesterol levels, which may elevate the risk of developing metabolic diseases [40].

Endocrine Disorders

The use of anabolic-androgenic steroids (AAS) by bodybuilders is associated with significant endocrine disturbances, including hypogonadism, infertility, and thyroid dysfunction. A systematic review by Vilar Neto et al. analyzed cases of AAS-induced hypogonadism in men. The results indicate that AAS use leads to suppression of the hypothalamic-pituitary-gonadal

axis, resulting in a decrease in endogenous testosterone levels and symptoms of hypogonadism, such as reduced libido, erectile dysfunction, and muscle mass loss [41].

A 2023 meta-analysis by Mulawkar et al. evaluated the impact of AAS on semen parameters and male fertility. It was found that AAS use leads to a significant reduction in sperm count and motility, which may result in infertility [42]. The study by Deyssig et al. demonstrated that bodybuilders using AAS experience thyroid dysfunction, including reduced levels of thyroxine (T4) and triiodothyronine (T3), suggesting a negative effect of AAS on thyroid hormone metabolism [43]. Solanki et al. analyzed the process of recovering endocrine function after cessation of AAS use. The results suggest that some men experience persistent hormonal disturbances, such as sustained hypogonadism, indicating that the negative effects of AAS may be long-lasting or even irreversible [44].

Conclusion

The use of anabolic-androgenic steroids (AAS)—though medically registered—is becoming increasingly common among athletes and bodybuilding enthusiasts. Unfortunately, their chronic and non-medical use carries serious health consequences. AAS primarily act through androgen receptors, stimulating muscle growth, but they also negatively affect multiple systems of the body.

- **Cardiovascular System**: Long-term AAS use leads to cardiomyopathy, impaired heart function, atherosclerotic changes, and an increased risk of sudden cardiac death (SCD).
- **Nervous System and Mental Health**: Neuroimaging and clinical studies indicate changes in brain structure, increased aggression, emotional instability, depression, and even psychopathic traits among AAS users.
- **Kidneys**: AAS use is associated with kidney damage, including oxidative stress, activation of the RAAS system, rhabdomyolysis, and the development of conditions like FSGS and tubular necrosis.
- **Liver**: Oral 17α-alkylated steroids are particularly toxic, potentially causing cholestasis, peliosis, liver cancer, and elevated liver enzymes.
- **Metabolic Disorders**: AAS can lead to dyslipidemia, insulin resistance, and disturbances in glucose-lipid metabolism, thereby increasing the risk of cardiovascular diseases.

Conceptualization, Julia Marcinkowska; methodology, Julia Marcinkowska; software, Mateusz Muras; check, Maciej Michalik; formal analysis, Krzysztof Marcinkowski; investigation, Tomasz Lorenc; resources, Julia Marcinkowska; data curation, Jakub Mikszta; writing - rough preparation, Julia Marcinkowska; writing - review and editing, Maciej Michalik; visualization, Natalia Mikszta; supervision, Natalia Mikszta; project administration,

Tomasz Lorenc; receiving funding, Krzysztof Marcinkowski, Natalia Mikszta. All authors have read and agreed with the published version of the manuscript.

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