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eISSN 2450-3118 · Open Access · Peer-reviewed

apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

Cite as: KARDASZEWSKI, Piotr, KAPTURSKA, Natalia, CYMERYS, Kinga, MĄKA, Magdalena, KADŁUBEK, Sabina, KALINOWSKI, Szymon, WOŹNIAK, Julia, KAMIŃSKI, Jakub, SIKORA, Dominik and GÓRALCZYK, Ewa. Impact of Anabolic Androgenic Steroids on Cardiovascular and Mental Health: Current Literature Review. Quality in Sport. 2026;58:72653. https://doi.org/10.12775/QS.2026.58.72653

ARTICLE TIMELINE

Received: 26.05.2026. Revised: 30.05.2026. Accepted: 31.05.2026. Published: 20.06.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines: Medical Sciences; Health Sciences.

Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Nauki medyczne; Nauki o zdrowiu.

Impact of Anabolic Androgenic Steroids on Cardiovascular and Mental Health: Current Literature Review

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ABSTRACT:

Introduction and purpose: Anabolic Androgenic Steroids were introduced in bodybuilding and treatment of medical conditions due to anabolic effect. Nowadays their popularity is rising, so is accessibility and range of substances present. Evidence shows severe impact AAS abuse has on overall health, especially on cardiovascular and mental domains. The aim of this review is to consolidate knowledge about AAS adverse effects.

Materials and methods: Medical databases such as PubMed, Google Scholar and PMC were searched, data analyzed and put into the single article.

State of knowledge: AAS use becomes a rising issue these days with millions engaged in various sports, taking supraphysiological doses. AAS abuse is taking its toll having a negative impact on cardiovascular health increasing probability of diseases or death. Moreover AAS-induced psychiatric morbidity is also observable causing serious impairment of everyday functioning. Although evidence is solid and well documented there are still many understudied areas that require further research.

Conclusions: AAS use is an independent, major and historically underrecognized risk factor for both cardiovascular disease and psychiatric morbidity. Due to persisting underrepresentation of a matter future research priorities should include large prospective inception cohorts with co-primary cardiac and psychiatric endpoints, compound-specific risk profiling adolescent exposure studies and adequately powered treatment trials.

Key Words: Anabolic-androgenic steroids, Cardiovascular toxicity, Psychiatric morbidity, Athletes, Bodybuilders

1. INTRODUCTION AND EPIDEMIOLOGY

Anabolic–androgenic steroids (AAS) are synthetic derivatives of testosterone that started to appear in the global markets in the 70s and the 80s. They were engineered to amplify anabolic effects such as muscle protein synthesis, nitrogen retention, increased rate of muscle mass, strength and endurance gain and red blood cells production boost, simultaneously attenuating their androgenic effect such as virilization, although this separation wasn't fully completed to this day. The positive effects of AAS are undisputed i.e. treating medical conditions such as accelerated recovery from injuries, burns, surgeries, therapy of hypogonadism, osteoporosis, bone marrow related diseases or even cancers **[1]**, however what should always be kept in mind is that even the best remedy can become poison when used incorrectly. Commonly known and used compounds in AAS group include testosterone esters (enanthate, cypionate, propionate), nandrolone decanoate, stanozolol, trenbolone, oxandrolone and methandrostenolone, often used in polypharmacy "stacks" at doses 5–40× physiological levels with addition of insulin, growth hormone, and Selective Androgen Receptor Modulators **[2]**. At first their use wasn't

regulated due to novelty and insufficient knowledge about them. Nowadays they are Schedule III controlled substances in the United States, prohibited by WADA and increasingly misused beyond competitive sport. Estimated global prevalence stands at 6,4 % in males and 1,6 % in females with 3-4 million lifetime users in the US alone and tens of millions worldwide **[3, 4]**. The community of users consists of elite athletes, recreational bodybuilders and fitness enthusiasts around the world using supraphysiological doses, in many cases from unregulated sources and without proper medical supervision. This review synthesizes evidence published between 2017 and 2026 across two areas of AAS-induced systemic harm: cardiovascular toxicity and psychiatric morbidity. Together, these domains represent the leading causes of premature death and severe disability in AAS-using populations. The key findings about the adverse effect of AAS are concerning. AAS users face an 8.9× higher risk of cardiomyopathy, 3.6× higher heart failure risk and 3.0× higher myocardial infarction risk compared to matched controls over 11 years of follow-up **[5]**. Moreover, they demonstrate a 2.81× higher all-cause mortality and 3.64× higher unnatural death hazard driven by accidents, violence and suicide compared to matched controls **[6]**. The Danish anti-doping registry underpins both landmark findings, with converging evidence from prospective cohorts (HAARLEM), experimental meta-analyses, neuroimaging and clinical case series. Both harm domains share mechanistic roots such as Hypothalamic-Pituitary-Gonadal axis suppression, androgen receptor overactivation, neurobiological and vascular dysregulation and a prothrombotic, pro-inflammatory milieu.

As mentioned above the population of AAS users has evolved from elite athletes to recreational trainees. The contemporary user is most commonly a male between 20-40 years old engaged in strength or aesthetic sports sourcing compounds often from on-line markets. However, female use which is estimated at 10-20% of all users is also increasing in fitness, aesthetic and transgender context but sadly remains dramatically understudied **[7]**. The mentioned trend is worrisome and clearly shows how easily accessible AAS have become in recent years without the need for proper cause and medical supervision. Moreover, FIDO-DK study, an ongoing nationwide cross-sectional cohort captures preclinical coronary disease, vascular plaque, myocardial structure, depression severity, aggression, body image concerns and cognitive function. The aim of this article is to consolidate knowledge about AAS, simultaneously raising social awareness about the adverse impact AAS can have on the human body, emphasizing the strain on cardiovascular and mental health **[8]**.

2. STATE OF KNOWLEDGE

2.1 Structural Cardiac Remodelling

The consequences of AAS abuse considering the cardiovascular system are dire and became notably better documented over recent years. The first aspect which should be looked at is structural cardiac remodelling caused by AAS. The Left Ventricular Hypertrophy (LVH) is the most consistently documented structural consequence of AAS use. Unlike the physiological "athlete's heart" which produces eccentric hypertrophy with preserved diastolic function, the AAS-induced LVH follows a concentric pattern driven by androgen receptor overactivation on cardiomyocytes, Renin-Angiotensin-Aldosterone System dysregulation and ERK1/2/mTOR pathway signalling **[2, 9]**.

The HAARLEM Study demonstrated casualty directly in 31 male amateur athletes followed through a complete AAS cycle (median of duration 16 weeks) with a median 8 month recovery period. The results showed an increase in LV mass by 28.3 g during the cycle in a dose dependent fashion also representing the positive correlation with average weekly AAS dose. Three dimensional Left Ventricular Ejection Fraction declined by 4,9 %, E/A ratio declined by 0.45 and LA volume increased by 9.2 mL all reversing fully over the course of 1 year. Although the results indicate that harm induced by one cycle can be reversed, the repeated cycling may cause cumulative, irreversible damage **[10]**.

Reversibility is not guaranteed in all patients. A case series reports a 53 year old bodybuilder presenting with LVEF 15% and testosterone levels beyond normal range (30 160 ng/dl). Additionally an Olympic athlete misusing AAS for 20 years developed ventricular tachycardia and severe combined systolic/diastolic heart failure at age of 73 in the absence of coronary heart disease. In such cases the patients with cumulative exposure lasting years or decades often require Left Ventricular Assisting Device (LVAD) bridge or even cardiac transplantation and reversibility correlates inversely with cumulative dose and duration of use **[11, 12]**.

Lingirredy et al. and Saimeh et al. confirmed biventricular dysfunction as a dominant cardiomyopathy phenotype related to AAS abuse presenting following results: LVEF 51% among users versus 55-57% in non-users in cardiac MRI, moreover Right Ventricular dysfunction was also present however it was detectable only by 2D speckle tracking echocardiography. The additional risk factor the authors discovered using late gadolinium enhancement CMR (Cardiovascular Magnetic Resonance) was myocardial fibrosis that creates arrhythmogenic substrate predisposing to sudden cardiac death **[2, 13]**.

2.2 Coronary artery disease and arteriosclerosis

Baggish et al. demonstrated in a cross-sectional study of 140 male weightlifters (ages 34–54) that each additional 10 years of cumulative AAS exposure was associated with a 0.60 SD increase in coronary plaque volume rank. Through that evidence establishing a dose-response relationship between AAS duration and subclinical atherosclerosis. AAS users also had significantly reduced LVEF and diastolic function compared to drug-free controls represented in early peak tissue velocity 7.4 vs. 9.9 cm/s **[14]**.

The mechanisms of coronary injury are multifactorial. AAS dramatically reduces HDL-C by ≥50% from baseline while elevating LDL-C and lipoprotein(a), creating a severely atherogenic lipid profile. Inflammatory cytokine upregulation (TNF-α, IL-6, hs-CRP), platelet activation and a prothrombotic state further accelerate plaque formation and destabilisation **[15]**. Importantly, coronary events in young AAS users may occur through thrombotic and erosive mechanisms entirely distinct from classical atherosclerotic rupture. Optical coherence tomography of a 30-year-old AAS user with NSTEMI revealed intimal hyperplasia, plaque erosion and thrombus formation in the left anterior descending artery without underlying plaque **[16]**. A separate case report documented "Tombstone" STEMI after only 2 months of AAS use in a 34-year-old which is attributable to AAS-induced hypercoagulability and platelet aggregation rather than atherosclerotic plaque **[17]**. These cases underscore that young AAS users presenting with acute coronary syndromes may have normal-appearing coronary arteries on angiography.

2.3 Arrhythmias and Sudden Cardiac Death

Sudden cardiac death (SCD) is the most feared complication of AAS misuse. Torrisi et al. systematically reviewed 33 autopsy-confirmed SCD cases linked to AAS, published between 1993 and 2020. The mean victim age was 29.8 years, 94% were male from whom 39% were bodybuilders. Myocardial fibrosis was present in 79% of cases and myocardial necrosis in 52%, followed by cardiomegaly (33%) and LVH (30%). Remarkably 72% had no prior cardiac history. The most frequently detected compounds were nandrolone scoring 30%, testosterone at 27% and stanozolol at 21% **[18]**.

The arrhythmogenic substrate is multifactorial: myocardial fibrosis, concentric LVH, gap junction remodelling and prolonged cardiac action potential duration, all consequences of chronic androgen receptor overactivation. Gołdyn et al. describe impaired global longitudinal strain persisting years after AAS cessation and PET/CT evidence of persistent coronary microvascular dysfunction even after stopping which represents durable arrhythmogenic vulnerability independent of active use **[19]**.

2.4 Pathophysiological mechanisms

The cardiovascular toxicity of AAS is diverse in case of mechanisms leading to it. There are four different pathways that converge on myocardial injury **[2, 15]**. The atherogenic pathway is driven by AAS-induced dyslipidemia: HDL-C reductions of 50% or more, combined with pro-inflammatory cytokine release, accelerate foam cell formation, plaque progression and endothelial dysfunction. The thrombotic pathway operates through altered prostaglandin metabolism, increased platelet aggregation, stimulation of pro-coagulant factor synthesis and polycythemia explaining acute Myocardial Infarction in young users with minimal plaque burden. The vasospastic pathway involves upregulation of voltage-gated calcium channels, increased angiotensin II and thromboxane, and inhibition of nitric oxide synthase causing enhanced vascular reactivity and measurably reduced Fibromuscular dysplasia (FMD) and Carotid artery reactivity (CAR) **[20]**. The direct myocardial injury pathway results from androgen receptor overactivation on cardiomyocytes, triggering ERK1/2 and mTOR pathways, promoting pathological hypertrophy, collagen deposition and myocardial fibrosis, while oxidative stress and RAAS dysregulation amplify fibrosis and arrhythmogenesis **[15]**.

2.5 Vascular dysfunction

Tungesvik et al. in Scientific Reports 2024 directly quantified vascular dysfunction in 56 young AAS users versus 67 matched weightlifting controls using validated non-invasive measurements. The results showed FMD % on brachial arteries was lower in the AAS users group (3,99%) than in the not enhanced control group (6,72%). The Carotid Artery Reactivity followed the same trend being 3,58 % in the AAS group and 6,33% in the control group. The baseline brachial diameter and diastolic BP (MAP) were higher in the enhanced group than in the control group. Reduced FMD reflects endothelial dysfunction - an established prognostic marker for future atherosclerosis and cardiovascular events. Reduced CAR indicates heightened susceptibility to turbulent blood flow and clot formation. These findings, observed in young men with a median age of 39, explain sudden cardiac events even in the absence of morphological plaque changes and were independent of blood pressure or training load **[20]**.

2.6 Depression and Anxiety

The most consistent psychiatric finding across study designs is an elevated burden of depressive symptoms and antidepressant use among AAS users. The Danish registry study by Horwitz et al. which is the largest controlled study in this area, identified 545 laboratory-confirmed AAS users and 5,450 matched controls followed over a mean of 17 years. Adjusted hazard ratios for antidepressant prescription were 1.44 (95% CI: 1.11–1.87), with cumulative antidepressant prevalence of 26.2% in AAS users versus 16.3% in controls. Antipsychotic prescriptions and anxiolytics were similarly elevated, rising further in the 2 years following the sanction in self-controlled analysis. Notably, elevated antidepressant use was detectable even before the index sanction, suggesting pre-existing vulnerability or chronic sub-threshold depression preceding formal AAS use **[21].**

At the individual clinical level, Karagun & Altug demonstrated significantly elevated Beck Depression Inventory and Beck Anxiety Inventory scores in AAS-using bodybuilders compared to matched non-using controls, with 28% meeting criteria for mild-to-moderate depression. Biochemical correlates creatinine and estradiol as independent BDI predictors, with

creatinine BDI area under curve 0.853 pointing to potential biomarker applications **[22]**. Börjesson et al. found a 41% prevalence of depressive disorder by SCID-I in a help-seeking sample with 25% reporting suicidal thoughts **[23]**.

A critical mechanistic pathway is AAS-withdrawal hypogonadism which is a suppression of the HPG axis. During chronic use it produces profound testosterone deficiency upon cessation, with hypogonadal depression persisting months to years. Remelhe et al. describe a biphasic model - an initial hyperadrenergic/opioid-withdrawal-like phase followed by a prolonged depressive phase driven by hypogonadism. Moreover this withdrawal depression coexists with the partial reversal of cardiac remodelling seen in the HAARLEM study - the same months during which HDL- C and LV function are recovering are often the months of greatest psychiatric vulnerability. **[10, 24]**

Evidence for anxiety as a direct AAS effect is more heterogeneous. While the Danish registry found a 2.3-fold adjusted HR for anxiolytic prescriptions, Deslandes et al. found no dose-response between AAS dose and HAM-A anxiety scores and Nelson et al. found no significant adjusted odds ratio for anxiety symptoms (OR 1.14, CI: 0.71–1.80) after controlling for polysubstance use. That suggests that anxiety may be primarily mediated by comorbid conditions or withdrawal states **[21, 25]**.

2.7 Aggression and Violence

The aggression–AAS relationship is characterised by statistical significance but modest effect magnitude in experimental work compared with more striking findings in observational and forensic data. The 2021 meta-analysis by Sagoe et al. of 11 Randomized Clinical Trials reported an overall small but significant increase in self-reported aggression with acute AAS administration producing a larger effect. The modest experimental effect is likely an underestimate due to RCT doses rarely exceeding 600 mg/week, while real-world polypharmacy regimens commonly exceed 1,000 mg/week **[26]**.

Observational data demonstrate far stronger effects. Ceto et al. found markedly elevated Buss–Perry Aggression Scale scores in AAS users versus non-users (99.75 ± 17.77 vs. 73.86 ± 18.36, t = 10.81), with a dose-frequency response. They found that more cycles per year predicted higher physical aggression and hostility **[27]**. Börjesson et al. found that AAS users with comorbid personality disorders had dramatically elevated odds of aggressive feelings, suicidality and criminality **[23]**.

The population-level correlation is the unnatural death HR of 3.64 in the Danish mortality cohort , driven primarily by accidents and violence **[6]**. Kanayama et al. argue that AAS-induced violence is biologically plausible, caused by amygdala hyperreactivity, impaired frontal-limbic connectivity, idiosyncratic and affects a minority of users **[4]**. The neuroimaging substrate is provided by Bjørnebekk et al. who demonstrated accelerated brain aging correlated with AAS dependence duration on structural MRI, with direct implications for cognitive and impulse-control deficits **[28]**.

2.8 Psychosis and Mania

Psychotic and manic presentations are documented but comparatively rare. Pooling four high-dose placebo-controlled trials, Kanayama et al. found that 4.6% of men developed hypomanic or manic syndromes on supraphysiological doses versus 0% on placebo **[4]**. Case reports document frank delirium, acute psychomotor agitation and persecutory delusions **[30]**. The Danish registry found no significantly elevated prevalence of schizophrenia diagnoses (1.7% vs. 1.9%), but antipsychotic prescription rates were more than doubled (HR 2.22), suggesting sub-syndromal or active psychotic presentations . The mechanisms proposed include dopaminergic dysregulation and HPG axis disruption leading to testosterone-mediated increases in amygdala reactivity — the same limbic overdrive that underlies the aggression data **[21]**.

2.9 Muscle Dysmorphia

Muscle dysmorphia (MD) which is a form of body dysmorphic disorder characterised by pathological preoccupation with muscularity functions as both a precursor and consequence of

AAS use. The PROSPERO-registered meta-analysis by Çınaroğlu & Yılmazer found a positive correlation between MD symptom severity and obsessive-compulsive traits, moreover MD symptoms in AAS users were significantly higher in comparison to non-users **[30]**. Luty et al. report MD prevalence as high as 58% in some AAS-using bodybuilder samples in contrast to 2–6% in the general population **[31]**. Sexual minority populations are at further elevated risk, likely mediated by stigma-related body image pressures **[7]**.

The network analysis by Scarth et al. found no significant symptom bridges between AAS dependence and MD clusters suggesting they are not merely variants of the same disorder and require separate treatment targets **[32]**. This was confirmed by the first RCT of cognitive behavioural therapy for MD, which demonstrated large effects on MD symptoms and moderate-large effects on depression and distress without directly addressing AAS use, establishing the first evidence-based intervention in this field **[33]**.

2.10 Psychopathology Clustering and Personality Disorders

A critical recent advance is the recognition of distinct psychopathological subtypes among AAS users. Bjørnebekk et al. identified four clusters in 214 participants: "no psychopathology," "mild externalising," "severe multipathology," and "mild multipathology." Approximately 50% of AAS users fell into multipathology clusters, with 17.9% in the severe cluster versus 2.1% of non-users. Antisocial personality disorder patterns were most discriminating. AAS users with severe multipathology had earlier onset of use (mean 18.2 years), confirming early initiation as a marker of a high-risk subpopulation requiring intensive clinical observation. **[34]**

3. CONCLUSIONS

The evidence from 2017–2026 establishes with high consistency that Androgenic Anabolic Steroids use is an independent, major and historically underrecognised risk factor for both cardiovascular disease and psychiatric morbidity. At the population level, the Danish registry being the most methodologically rigorous data source in this literature, demonstrates 8.9-fold elevated cardiomyopathy risk, 2.81-fold elevated all-cause mortality and 3.64-fold elevated

unnatural death risk over approximately a decade of follow-up **[5, 6]**. Cardiovascular evidence regarding negative effects of AAS is the strongest for structural cardiac remodeling mainly considering LVH and biventricular cardiomyopathies, coronary atherosclerosis, arrhythmias and SCD. Data gathered in this literature review clearly shows that AAS misuse puts a serious strain on cardiovascular health and especially without proper medical supervision can lead to severe disability or death **[10, 14, 18, 19]**. Four interdependent pathophysiological mechanisms responsible for AAS adverse effect were found: atherogenic, thrombotic, vasospastic and direct myocardial injury and they converge to explain the full phenotypic spectrum **[15]**. Psychiatric evidence is strongest for elevated depression and psychopharmacological treatment burden, small-moderate causal contribution to aggression and muscle dysmorphia **[21, 26, 30, 33]**. Psychopathological heterogeneity among AAS users is increasingly recognised, with an estimated 18% displaying severe multipathology requiring intensive intervention and treatment **[34]**. However both domains are limited by the near-total absence of prospective studies with primary endpoints in both areas, systematic under-representation of women, heavy reliance on selected samples, insufficient dose-response data and heterogeneous compound exposure. Despite wide-spread popularity and ever increasing use case of AAS-related adverse effects is still understudied and underrepresented in scientific literature. Future research priorities should include large prospective inception cohorts with co-primary cardiac and psychiatric endpoints, compound-specific risk profiling adolescent exposure studies and adequately powered treatment trials. Moreover, the focus should gravitate towards presenting the treatment values of AAS simultaneously raising awareness of the risks related to unsupervised use, these substances could have on the general health **[8]**.

DISCLOSURE

Funding Statement: The study was conducted without special funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Not applicable.

Acknowledgments: Not applicable.

Conflict of Interest Statement: The authors report no conflicts of interest.

All authors have read and approved the published version of the manuscript

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