Review

At the Other End of the Spectrum: Bigorexia as an Emerging Cardiovascular Phenotype in Adolescents

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At the Other End of the Spectrum: Bigorexia as an Emerging Cardiovascular Phenotype in Adolescents

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Abstract

Bigorexia, or muscle dysmorphia, is an increasingly prevalent body image disorder among adolescents characterized by an excessive drive for muscularity. This behavioral phenotype is associated with a cluster of lifestyle exposures—including anabolic androgenic steroid use, high energy drink consumption, and excessive high-protein diets—that may collectively promote early cardiovascular injury. Emerging evidence suggests that these factors contribute to a distinct cardiovascular phenotype marked by premature atherosclerosis, pathological left ventricular hypertrophy, and increased arrhythmogenic risk. Despite growing recognition of bigorexia as a psychological condition, its cardiovascular implications remain underexplored. This review synthesizes current evidence linking bigorexia-related behaviors to cardiovascular dysfunction and proposes a conceptual framework for this emerging phenotype, highlighting the need for early recognition, risk stratification, and targeted preventive strategies.

Keywords: bigorexia; energy drinks; anabolic proteins; high protein diet; cardiovascular complications

Background

In recent years, a growing body of evidence has highlighted the emergence of a novel behavioral and body image phenotype among adolescents characterized by an excessive preoccupation with muscularity, commonly referred to as muscle dysmorphia or "bigorexia." Unlike traditional eating disorders that emphasize thinness, bigorexia is driven by the pursuit of increased muscle mass and a distorted perception of insufficient muscularity despite normal or even above-average physique [1].

Epidemiological data suggest that features of muscle dysmorphia may affect up to 5–10% of adolescent males, with subclinical traits being even more prevalent, particularly among those engaged in competitive sports, weight training, or fitness-oriented social environments. The rise of this condition has been closely linked to social media exposure, fitness influencers, and algorithm-driven content, which reinforce unrealistic body ideals and normalize extreme behavioral patterns. Adolescents, due to their developmental vulnerability, are particularly susceptible to these influences, leading to early adoption of rigid training regimens, restrictive or excessive dietary practices, and performance-oriented supplementation [2–4].

Importantly, this behavioral phenotype is not isolated but rather embedded within a broader cluster of lifestyle exposures that may carry significant health implications. Adolescents exhibiting features of bigorexia are more likely to engage in high-intensity resistance training, consumption of high-protein or unbalanced diets, frequent intake of energy drinks and stimulants, and, in some cases, early initiation of anabolic androgenic steroid (AAS) use or other performance-enhancing substances. Surveys indicate that energy drink consumption among adolescents exceeds 30–50% in many regions, often in conjunction with exercise, while the use of dietary supplements—including protein

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powders and pre-workout formulations—has become increasingly normalized at younger ages. More concerningly, emerging reports suggest a non-negligible prevalence of AAS experimentation in late adolescence, particularly in gym-based subcultures, where accessibility and peer influence lower the threshold for initiation. These exposures, often perceived as benign or even beneficial within fitness culture, may collectively create a unique and under-recognized cardiometabolic risk profile, distinct from traditional lifestyle-related risk factors observed in pediatric populations [1,5,6]

Despite the increasing recognition of bigorexia as a psychological and behavioral entity, its systemic and cardiovascular implications remain insufficiently characterized, particularly in adolescents who are otherwise considered at low baseline cardiovascular risk. The convergence of performance-enhancing substances, stimulant overuse, and extreme dietary patterns raises concerns regarding the early cardiovascular remodeling and vascular injury, potentially giving rise to a previously underappreciated phenotype [2,7].

In this review, we aim to explore the emerging link between bigorexia-related behaviors and early cardiovascular alterations in adolescents, with a focus on the development of premature atherosclerosis, left ventricular hypertrophy, and arrhythmogenic risk. We further seek to provide a conceptual framework for this phenotype, highlight potential risk modifiers, and identify key gaps that warrant future investigation [6].

Bigorexia and Energy Drinks

The global rise in energy drink (ED) consumption among both the general population and athletic communities has been driven largely by highly targeted and effective marketing strategies, often emphasizing enhanced performance, alertness, and endurance. Adolescents represent a particularly vulnerable and rapidly expanding consumer group, with multiple epidemiological surveys indicating that 52–68% of adolescents report regular or occasional ED intake, compared with an estimated prevalence of approximately 30–35% in adults. This disproportionate uptake in younger populations is further amplified by peer influence, sports culture, and digital media exposure, where EDs are frequently positioned as integral components of an active and performance-oriented lifestyle [8].

The composition of EDs is highly variable and often lacks full transparency. Caffeine content typically ranges from 75 to 240 mg per serving, although some formulations may exceed these values depending on portion size and concentration. Taurine, another key ingredient, is commonly present at an average concentration of approximately 300–400 mg per 100 mL, resulting in total doses that can reach several grams with typical consumption patterns. In contrast, the precise quantities of other constituents—such as guarana, ginseng, B vitamins, and various proprietary blends—are frequently incompletely disclosed, raising concerns regarding cumulative stimulant exposure and potential interactions. From a functional standpoint, caffeine (at doses of approximately 3–6 mg/kg) and taurine (approximately 1–6 g) are considered the principal ergogenic components. However, current evidence suggests that additive or synergistic performance-enhancing effects between these compounds remain unproven and biologically uncertain [9–11].

Importantly, despite widespread perception of benefit, the relationship between ED consumption and improved physical performance remains inconsistent across studies, largely due to heterogeneity in study design, dosing protocols, participant characteristics, and outcome measures. This variability, coupled with increasing patterns of frequent, high-volume, or combined use with other stimulants, raises important concerns regarding the safety profile of EDs—particularly in adolescents, where physiological responses to stimulants may differ and long-term effects remain insufficiently characterized [12,13].

The cardiovascular effects of excessive energy drink (ED) consumption are largely mediated by caffeine, which acts as a competitive antagonist of adenosine receptors. This antagonism leads to enhanced sympathetic nervous system activation, characterized by increased endogenous catecholamine release, resulting in tachycardia, elevated blood pressure, and augmented myocardial contractility [6]. Collectively, these effects promote a mismatch between myocardial oxygen supply

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and demand, thereby predisposing susceptible individuals to ischemia and increasing the risk of major adverse cardiovascular events (MACEs)[14].

Beyond these acute hemodynamic effects, chronic ED consumption has been increasingly associated with endothelial dysfunction and platelet hyperreactivity. Reduced nitric oxide bioavailability, coupled with increased oxidative stress, represents a central mechanism underlying microvascular injury, which in turn contributes to vascular stiffness and accelerates atherogenesis. Additionally, the prothrombotic milieu induced by platelet activation may further exacerbate plaque instability [15].

Evidence in a systematic review by Gualberto and colleagues consistently shows that energy drink consumption can produce measurable acute changes in cardiovascular parameters. In a systematic review including 37 studies and 1,597 participants, increases in heart rate were reported in approximately 60.9% of studies. Significant increases in systolic and diastolic blood pressure were also observed in 53.8% and 61.5% of studies, respectively. A meta-analysis of randomized clinical trials further demonstrated that acute energy drink consumption significantly increases both systolic and diastolic blood pressure compared with placebo beverages. These findings suggest that stimulant ingredients in energy drinks can acutely alter cardiovascular hemodynamics even in healthy individuals [8].

Although the adverse health effects of energy drinks (EDs) are increasingly recognized, the precise mechanisms underlying their cardiovascular complications remain incompletely defined. Acute hemodynamic responses to ED consumption in healthy individuals have been relatively well characterized, typically demonstrating modest increases in systolic and diastolic blood pressure (approximately 3–5 mmHg) with minimal or inconsistent effects on heart rate. While such transient changes are unlikely to pose significant risk in most young consumers, rapid ingestion of large volumes or use in susceptible individuals may precipitate more clinically relevant hemodynamic disturbances [9].

More recently, attention has shifted toward the electrophysiological effects of EDs, particularly their potential to induce QT interval prolongation—a known substrate for malignant ventricular arrhythmias. In a well-designed double-blind, placebo-controlled crossover study, Shah et al. demonstrated that consumption of two commercially available EDs resulted in a mean increase in corrected QT interval (QTc) of 18–20 ms, with effects persisting for up to four hours. Notably, while no participant exhibited QTc values exceeding 500 ms, QTc prolongation greater than 50 ms—an established threshold associated with increased arrhythmic risk—was observed in a subset of subjects. These findings contrast with placebo exposure, which produced only minimal and transient QTc changes, and are consistent with prior reports of modest blood pressure elevations without significant chronotropic effects [16].

From a regulatory perspective, these observations raise important safety concerns. In the context of pharmaceutical development, agents producing this degree of QT prolongation would mandate extensive evaluation through dedicated “thorough QT/QTc” studies prior to approval. In contrast, EDs—marketed as beverages or dietary supplements—are not subject to equivalent safety requirements. Instead, regulatory frameworks rely on the classification of individual components as “generally recognized as safe” (GRAS), without adequately addressing the potential for synergistic or cumulative effects arising from complex multi-ingredient formulations [9,10,13].

Notably, existing evidence does not implicate caffeine alone as a consistent cause of QT prolongation, and other commonly included ingredients—such as taurine, guarana, L-carnitine, sugars, and B vitamins—have not been independently associated with significant QT interval changes at typical concentrations. Although certain compounds, such as ginseng, have been suggested to influence cardiac repolarization, controlled comparisons indicate no significant isolated effect on QT duration. The observation that different ED formulations may produce similar electrophysiological effects raises the possibility of a class effect, potentially mediated by unidentified interactions between ingredients or dose-dependent cumulative exposure. However, given the

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marked heterogeneity across more than 100 commercially available products, each with proprietary and often undisclosed compositions, definitive conclusions remain limited [9,11,16].

Bigorexia and Anabolic Steroids

Anabolic-androgenic steroids (AAS), which include testosterone and its synthetic analogues, are commonly utilized within resistance-training and bodybuilding settings to accelerate muscle hypertrophy and improve physical performance. Epidemiological data indicate that the lifetime prevalence of AAS use is approximately 6–7% in men and around 1–2% in women, with markedly higher rates observed among competitive athletes, recreational bodybuilders, and individuals with coexisting substance use behaviors. Of particular concern is the increasing uptake among adolescents and young adults, reflecting the growing influence of body image ideals and performance-driven environments [17].

The use of AAS has been linked to a wide array of neuropsychiatric and systemic complications, encompassing mood disturbances, impaired cognition, behavioral changes such as irritability and aggression, endocrine disruption, and cardiovascular pathology. A substantial proportion of users—estimated at up to one-third—develop features of dependence, characterized by compulsive use, difficulty in cessation, and persistence despite adverse consequences [18]. Discontinuation is often associated with a withdrawal syndrome driven by hypogonadism, manifesting as fatigue, depressive symptoms, anxiety, reduced libido, and sexual dysfunction, which may further reinforce ongoing use. The pathogenesis of AAS dependence is likely multidimensional, involving both physiological mechanisms—such as suppression of endogenous hormonal pathways—and psychological reinforcement linked to perceived gains in strength, physique, and self-esteem. Individuals with established dependence are particularly susceptible to harm due to longer durations of exposure and higher cumulative dosing [19].

A central factor contributing to the initiation and maintenance of AAS use is bigorexia, also referred to as muscle dysmorphia, a subtype of body image disorder characterized by a persistent conviction of being inadequately muscular despite objective evidence to the contrary. While engagement in structured exercise and nutritional optimization may be considered normative, in the context of bigorexia these behaviors become excessive, rigid, and disproportionately influential on daily functioning and self-worth. This condition has been strongly associated with the use of appearance- and performance-enhancing substances, including AAS.[5]

Individuals involved in strength-based sports—particularly bodybuilding—exhibit a higher burden of bigorexia-related symptoms compared with the general population, often accompanied by additional maladaptive patterns such as compulsive training, disordered eating behaviors, and unsupervised supplement use. Moreover, bigorexia frequently coexists with broader psychological vulnerabilities, including anxiety, depressive symptoms, perfectionistic traits, and heightened neuroticism, suggesting that it may reflect a more complex psychiatric profile [3].

The relationship between bigorexia and AAS use appears to be complex and potentially reciprocal. On one hand, bigorexia may act as a precipitating factor, driving individuals toward AAS use in pursuit of rapid and sustained increases in muscle mass. On the other, exposure to AAS may further entrench maladaptive body image perceptions and reinforce compulsive behaviors, thereby contributing to the persistence of the disorder. However, existing findings remain heterogeneous, and the extent to which prolonged AAS exposure exacerbates or alters the natural course of bigorexia has not been conclusively established. This bidirectional interaction highlights the importance of understanding AAS use within a broader framework that integrates behavioral, endocrine, and cardiovascular dimensions, particularly in younger populations [17,20].

The pharmacological effects of AAS are mediated through androgen receptor activation, leading to enhanced protein synthesis, accelerated skeletal muscle hypertrophy, and reduced recovery time following exertion. In addition, AAS may improve exercise capacity through stimulation of erythropoiesis, thereby increasing oxygen-carrying capacity and delaying fatigue. However, these perceived benefits are accompanied by a broad spectrum of adverse effects involving multiple organ

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systems, including hepatic toxicity, renal dysfunction, endocrine disruption, and reproductive impairment. Among these, the cardiovascular system represents the most clinically significant and potentially life-threatening target of AAS toxicity, particularly in the context of prolonged use and supraphysiological dosing [19,21].

Chronic exposure to AAS has been consistently associated with a range of structural, functional, and electrical cardiac abnormalities, collectively contributing to an elevated risk of adverse cardiovascular events. At the myocardial level, AAS exert direct effects through androgen receptor signaling within cardiomyocytes, promoting maladaptive remodeling characterized by left ventricular hypertrophy (LVH), myocardial fibrosis, and progressive impairment of both systolic and diastolic function. Unlike physiological adaptations observed in athletic training, AAS-induced hypertrophy is often pathological, asymmetric, and associated with increased myocardial stiffness, resulting in reduced ventricular compliance and impaired filling dynamics [22,23].

The development of LVH in this context appears to be mediated not only by direct anabolic signaling but also through neurohormonal dysregulation, particularly activation of the renin–angiotensin–aldosterone system (RAAS). Elevated levels of angiotensin II and aldosterone contribute to increased afterload, myocardial hypertrophy, and interstitial fibrosis, thereby amplifying structural remodeling. Over time, these changes compromise diastolic relaxation and may ultimately impair systolic performance, predisposing individuals to heart failure phenotypes [20,23,24].

A key pathological hallmark of AAS cardiotoxicity is the development of myocardial fibrosis, driven by the activation of cardiac fibroblasts and increased collagen deposition. This process is thought to be mediated through a combination of androgen receptor stimulation and angiotensin II–dependent pathways, leading to progressive stiffening of the myocardium. The coexistence of fibrosis and hypertrophy creates a substrate for both mechanical dysfunction and electrical instability, significantly increasing the risk of arrhythmogenesis [25].

In parallel, AAS use has been linked to hemodynamic alterations, including the development of systemic hypertension. Data from the HAARLEM study and other observational cohorts indicate that AAS users may experience mean increases of approximately 5–10 mmHg in systolic and 3–5 mmHg in diastolic blood pressure. Although modest in isolation, sustained elevations in blood pressure contribute to increased myocardial workload, vascular injury, and long-term cardiovascular risk, including the development of coronary artery disease and heart failure [26].

AAS misuse is also associated with profound disturbances in lipid metabolism, representing a critical mechanism underlying accelerated atherosclerosis. Specifically, AAS use has been shown to increase low-density lipoprotein (LDL) levels while significantly reducing high-density lipoprotein (HDL) concentrations, thereby promoting an atherogenic lipid profile. These alterations, particularly when sustained over time, contribute to endothelial dysfunction, plaque formation, and increased risk of coronary events [27].

Beyond structural and metabolic effects, AAS use is strongly implicated in the development of cardiac arrhythmias, encompassing both atrial and ventricular disturbances. The arrhythmogenic potential of AAS is likely multifactorial, arising from the combined effects of myocardial hypertrophy, fibrosis, autonomic imbalance, and electrolyte disturbances. These changes disrupt normal electrical conduction and increase susceptibility to malignant arrhythmias, including those that may culminate in sudden cardiac death, particularly in otherwise young and ostensibly low-risk individuals [21–24].

Furthermore, AAS use promotes a prothrombotic state, further compounding cardiovascular risk. By stimulating erythropoiesis, AAS can lead to polycythemia and increased blood viscosity, impairing microcirculatory flow and predisposing to thrombosis. In addition, alterations in platelet function and coagulation pathways may enhance hypercoagulability, increasing the likelihood of acute vascular events such as myocardial infarction and ischemic stroke [28].

Taken together, these interrelated mechanisms—spanning myocardial remodeling, vascular dysfunction, dyslipidemia, arrhythmogenesis, and thrombogenicity—define a complex and multifaceted cardiovascular risk profile associated with AAS misuse. In the context of adolescents

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and young adults, particularly those with coexisting behavioral drivers such as bigorexia, these effects may contribute to the early emergence of a distinct cardiovascular phenotype characterized by premature atherosclerosis, pathological hypertrophy, and electrical instability [20,23,24,28–30].

Bigorexia and High Protein Diet

Individuals with bigorexia often internalize and normalize behaviors that would otherwise be considered excessive or potentially harmful, particularly in relation to exercise and dietary practices. Within this population, high-protein intake is not only perceived as acceptable but is frequently regarded as essential and non-negotiable for achieving and maintaining an idealized muscular physique. Protein consumption is often pursued at levels that substantially exceed recommended dietary allowances, driven by the belief that continuous protein surplus is necessary to sustain muscle growth, prevent catabolism, and optimize recovery [31].

This perception is reinforced by fitness culture, peer environments, and online content, where elevated protein intake is consistently promoted as a cornerstone of physical transformation. As a result, dietary patterns characterized by rigid structuring of meals, frequent protein supplementation (e.g., shakes, bars, and powders), and prioritization of macronutrient targets over nutritional balance become deeply ingrained. Importantly, such practices are rarely viewed as excessive by the individuals themselves; rather, they are interpreted as markers of discipline, commitment, and adherence to a performance-oriented lifestyle [32].

Consequently, this normalization of chronic high-protein consumption—often coupled with low-fat or otherwise unbalanced dietary patterns—may obscure potential metabolic and cardiovascular consequences, particularly when integrated with other risk behaviors such as supplement overuse, stimulant intake, and anabolic steroid exposure.[31]

However, although increased protein intake offers clear benefits for athletic performance and muscle development, its long-term cardiovascular effects remain debated. The myocardium is sensitive to metabolic changes influenced by diet, including alterations in lipid metabolism, amino acid signaling, and systemic inflammation. Several studies have examined whether excessive protein consumption may contribute to cardiovascular diseases such as atherosclerosis, myocardial infarction, and heart failure. Findings remain mixed: some research suggests a possible link between very high protein intake and increased cardiovascular risk, while other studies report neutral or beneficial effects depending on factors such as protein source, age, and lifestyle. Therefore, despite its widespread use in sports nutrition, the long-term cardiovascular impact of high-protein diets remains under investigation [33–35].

A large cohort study examining the relationship between dietary protein intake and cardiovascular outcomes included 19420 participants, who were categorized into two groups: a high protein intake group (≥1.8 $g \cdot kg^{-1} \cdot day^{-1}$) and a low protein intake group (<1.8 $g \cdot kg^{-1} \cdot day^{-1}$). Outcomes were assessed using three primary data sources: hospital inpatient records, national death registries, and self-reported information obtained during follow-up interviews. The findings indicated that a high-protein diet was associated with an increased incidence of heart failure, cardiovascular mortality, and all-cause mortality, as well as a heightened risk of major adverse cardiovascular events (MACE) in individuals consuming ≥1.8 $g \cdot kg^{-1} \cdot day^{-1}$ of protein. These results suggest that excessive protein intake may contribute to cardiovascular pathology, despite its recognized metabolic benefits.[34]

The same study also analyzed the association between high protein intake and cardiovascular risk in different age groups. In participants aged 55 years or older, a high-protein diet was associated with an increased risk of major adverse cardiovascular events (MACE) as shown by Kaplan–Meier analysis. In contrast, no significant association between high-protein intake and MACE was observed among participants younger than 55 years, who are typically athletes or metabolically healthier individuals, which may reduce their risk. Notably, age demonstrated a significant interaction effect with protein intake for both MACE and stroke, indicating that the relationship between dietary protein and cardiovascular outcomes may be age dependent [36].

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An experimental study investigating the biological mechanisms linking high-protein diets to cardiovascular disease examined cellular pathways involved in atherosclerosis. The researchers found that elevated amino acid levels resulting from high protein intake can activate the mechanistic target of rapamycin (mTOR) signaling pathway in macrophages, key immune cells involved in plaque formation. Activation of mTOR suppresses mitophagy, the cellular process responsible for removing damaged mitochondria, leading to mitochondrial dysfunction and increased cellular damage caused by reactive molecules. This promotes macrophage apoptosis and contributes to the growth and instability of atherosclerotic plaques. Plaque progression in coronary arteries can reduce blood flow to the myocardium, increasing the risk of ischemic heart disease and myocardial infarction. Although these findings were primarily derived from experimental models, they provide important insight into how excessive protein intake could contribute to cardiovascular pathology [36,37].

Beyond biological mechanisms, epidemiological studies suggest that cardiovascular effects may also depend on the source of dietary protein. Cohort evidence indicates that protein derived from animal sources, particularly meat, may be associated with less favorable cardiovascular outcomes compared with plant-derived protein sources. In contrast, plant-based proteins, including legumes, nuts, and seeds, have been linked with improved cardiovascular health indicators and lower cardiovascular mortality in large population studies. These differences may reflect the overall nutritional composition of the foods providing the protein, as plant-based protein sources are typically part of dietary patterns associated with better cardiometabolic profiles. Findings from large cohort studies and recent meta-analyses therefore suggest that the type of protein consumed, rather than total protein intake alone, may influence long-term cardiovascular health and risk of heart disease.[31,32,38]

List of Abbreviations

Abbreviation	Full Term
AAS	Anabolic-Androgenic Steroids
ACS	Acute Coronary Syndrome
APEDs	Appearance- and Performance-Enhancing Drugs
BP	Blood Pressure
CVD	Cardiovascular Disease
ED	Energy Drink
HDL	High-Density Lipoprotein
LDL	Low-Density Lipoprotein
LVH	Left Ventricular Hypertrophy
MACE	Major Adverse Cardiovascular Events
MD	Muscle Dysmorphia (Bigorexia)
mTOR	Mechanistic Target of Rapamycin
QTc	Corrected QT Interval
RAAS	Renin–Angiotensin–Aldosterone System
RBC	Red Blood Cells

Conclusions

Bigorexia represents a growing yet underrecognized driver of cardiovascular risk in adolescents, extending beyond its psychological dimensions into a complex cardiometabolic syndrome. The convergence of anabolic steroid exposure, stimulant overuse, and extreme dietary practices creates a synergistic environment that promotes myocardial remodeling, vascular dysfunction, and electrical instability. This emerging phenotype challenges traditional paradigms of cardiovascular risk, as it affects individuals typically considered low-risk based on age and conventional factors. Early identification of at-risk individuals, particularly within athletic and fitness-oriented populations, is essential. Future research should aim to better characterize this phenotype, establish screening strategies, and evaluate targeted interventions. Addressing bigorexia-related behaviors may

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represent a critical opportunity to prevent early-onset cardiovascular disease in this vulnerable population.

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