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ANABOLIC STEROIDS IN SARCOPENIC OBESITY: A THERAPY WORTH REVISITING

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ABSTRACT

Aims: This narrative review critically examines the literature on sarcopenic obesity (SO), emphasizing its definition, pathophysiology, limitations of conventional therapies, and the therapeutic potential of anabolic androgenic steroids (AAS), particularly oxandrolone and nandrolone. Ethical considerations and cardiovascular risks associated with AAS use are also discussed. **Study Design:** Narrative literature review. **Methods:** Studies were identified through PubMed using the following primary search terms: "obesity", "sarcopenia", "sarcopenic obesity", "anabolic androgenic steroids", and "therapeutics". Additional searches were conducted using the "Find Topics" and "Literature Review" tools within the AI-powered Scispace platform. Relevant citations from key authors were also manually screened for inclusion. **Results:** Sarcopenic obesity is a multifactorial condition that significantly compromises functional and metabolic health, particularly in older adults. Conventional therapies—namely diet and exercise—often yield limited efficacy, especially in individuals with hormonal impairments or chronic inflammation. AAS such as oxandrolone and nandrolone have demonstrated beneficial effects on muscle mass preservation and recovery in select clinical settings. Nevertheless, concerns regarding cardiovascular safety and adverse effects persist, especially in cases of non-medical use. When ethically prescribed and carefully monitored, AAS may represent a viable adjunct in the management of refractory SO. **Conclusion:** While further large-scale, controlled studies are warranted, current evidence suggests that AAS may serve a legitimate therapeutic role in select cases of sarcopenic obesity unresponsive to conventional interventions. Their inclusion in multimodal rehabilitation strategies—when clinically justified and closely supervised—should be considered. Clear distinctions between medical use and abuse must guide both clinical decision-making and scientific discourse to ensure rational, patient-centered care.

KEYWORDS: Obesity, Sarcopenia, Anabolic Androgenic Steroids, Nandrolone, Oxandrolone, Therapeutics, Medical Ethics.

1. INTRODUCTION

The global health landscape is currently witnessing the simultaneous rise in the prevalence of obesity and sarcopenia, two interrelated syndromes often addressed independently and, as a result, ineffectively. Although each condition independently contributes to increased morbidity and mortality, their coexistence, referred to as sarcopenic obesity (SO), defines a distinct and particularly deleterious clinical phenotype (Prado et al., 2012). SO is characterized by the accumulation of visceral adiposity in conjunction with a progressive decline in skeletal muscle mass, strength, and function. This dual burden is associated with increased frailty, loss of independence, cardiometabolic complications, cancer progression, and premature mortality (Cruz-Jentoft et al., 2019) (Figure 1).

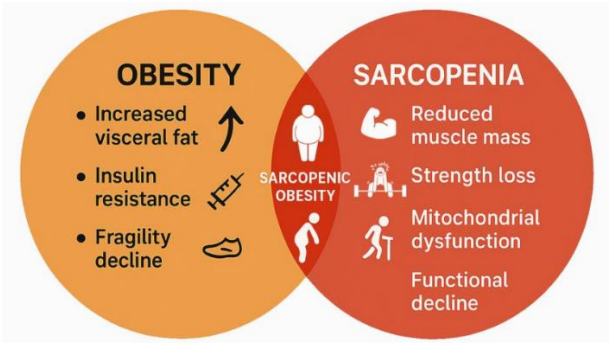

Fig. 1: Double burden of sarcopenic obesity.

Despite its growing prevalence, particularly among older adults, postmenopausal women, and individuals with metabolic syndrome, SO remains underdiagnosed, poorly characterized, and largely undertreated (Donini et al., 2022). The absence of a unified diagnostic framework, coupled with the underutilization of body composition assessment tools in clinical practice, contributes to its clinical invisibility. Conventional weight loss interventions, such as caloric restriction and aerobic exercise, may unintentionally worsen the sarcopenic component by inducing catabolism of lean mass, especially when implemented without concurrent anabolic support (Zamboni et al., 2008; Weinheimer et al., 2010). Conversely, strategies targeting sarcopenia through increased protein intake and resistance training may hinder or delay fat loss, thereby complicating obesity management.

In this context, the limitations of conventional therapeutic strategies may call for a paradigm shift toward anabolic interventions. There is growing consensus that treatment should aim to counteract muscle degradation through clinical approaches capable of stimulating protein synthesis, enhancing neuromuscular performance, and preserving metabolic health. Among the available options, anabolic androgenic steroids (AAS), despite strong evidence supporting their therapeutic efficacy in various catabolic states such as sarcopenia, frailty, and age-related muscle loss, remain stigmatized due to their association with misuse and performance enhancement in non-medical contexts (Woerdeman & De Ronde, 2011).

This review proposes a critical reassessment of AAS as legitimate therapeutic agents in the management of sarcopenic obesity, highlighting their physiological rationale, clinical applicability, and the ethical imperative of distinguishing medically supervised use from recreational abuse. Drawing from current evidence in pathophysiology, epidemiology, and therapeutic interventions, we advocate for the reconsideration of anabolic strategies as part of a comprehensive response to this increasingly prevalent and debilitating syndrome.

2. METHODS

This narrative literature review was conducted following a comprehensive search strategy to identify relevant studies on sarcopenic obesity and the therapeutic use of anabolic androgenic steroids (AAS). Two independent PubMed searches were performed on February 12, 2025, using combinations of controlled vocabulary (MeSH terms) and free-text keywords, as described in **Table 1**.

Databases	Search Strategy	Nº
PUBMED	((((Obesity[majr] OR "adipose tissue hyperplasia"[tiab] OR adiposit*[tiab] OR corpulency[tiab] OR "fat overload syndrome"[tiab] OR obesit*[tiab] OR overweight[tiab]) AND (Sarcopenia[majr] OR Sarcopenia*[tiab] OR "age-related muscle atrophy"[tiab])) OR (sarcopenic obesit*[tiab])) AND ("Anabolic Androgenic Steroids"[majr] OR Anabolic Androgenic Steroid[tiab] OR Anabolic Steroid*[tiab] OR anabolic agent*[tiab] OR anabolic drug*[tiab] OR anabolic hormone*[tiab] OR anabolic steroid*[tiab] OR anabolizing agent*[tiab] OR anabolizing cream[tiab] OR anabolizing drug[tiab] OR anabolizing treatment[tiab] OR oxandrolone[majr] OR oxandrolone[tiab] OR Nandrolone[majr] OR Nandrolone[tiab])) AND (Therapeutics[mh] OR Therapeutic*[tiab] OR Therapies[tiab] OR Therapy[tiab] OR Treatment*[tiab] OR "therapeutic use"[tiab])	319
PUBMED	(sarcopenic obesity) AND ("physiopathology"[MeSH Subheading] OR "pathophysiology"[tiab]) AND (("2015"[Date - Publication] : "2025"[Date - Publication]))	285
Total		**604**

In addition to database queries, complementary searches were conducted using the **Scispace** artificial intelligence platform (https://scispace.com/), employing its "Find Topics" and "Literature Review" tools to identify additional peer-reviewed literature. Manual citation tracking was also performed, targeting references cited in key articles identified during the initial screening.

Articles were selected based on their relevance to the following domains:

- Definitions and diagnostic criteria of sarcopenic obesity;
- Pathophysiological mechanisms linking adiposity and muscle loss;
- Clinical trials evaluating AAS (testosterone, nandrolone, oxandrolone) in muscle-wasting conditions;
- Guidelines and expert consensus statements on pharmacological and non-pharmacological interventions in sarcopenia and SO.

Studies in English, Portuguese and Spanish were used as language restrictions, and both randomized controlled trials and observational studies were considered. Review articles and meta-analyses were included to provide contextual depth where appropriate.

3. RESULTS AND DISCUSSION

3.1 Sarcopenic Obesity: Definition, Diagnosis, and Clinical Impact

Sarcopenic obesity (SO) is defined by the coexistence of excessive adiposity, particularly central or visceral fat, with reduced skeletal muscle mass, strength, and physical performance. Rather than a simple quantitative alteration in body composition, SO represents a profound impairment in functional capacity and metabolic health (Zamboni et al., 2008; Donini et al., 2022). Its dual phenotype confers greater morbidity and mortality compared to obesity or sarcopenia alone (Prado et al., 2012; Cruz-Jentoft et al., 2019). Diagnosis of SO involves a two-tiered approach: an initial screening to identify individuals at risk, followed by diagnostic confirmation based on body composition analysis and muscle function assessment. These steps allow clinicians to classify patients across multiple axes of disease severity (Donini et al., 2022).

Ahead, Figure 1 illustrates the diagnostic algorithm currently proposed in expert consensus documents.

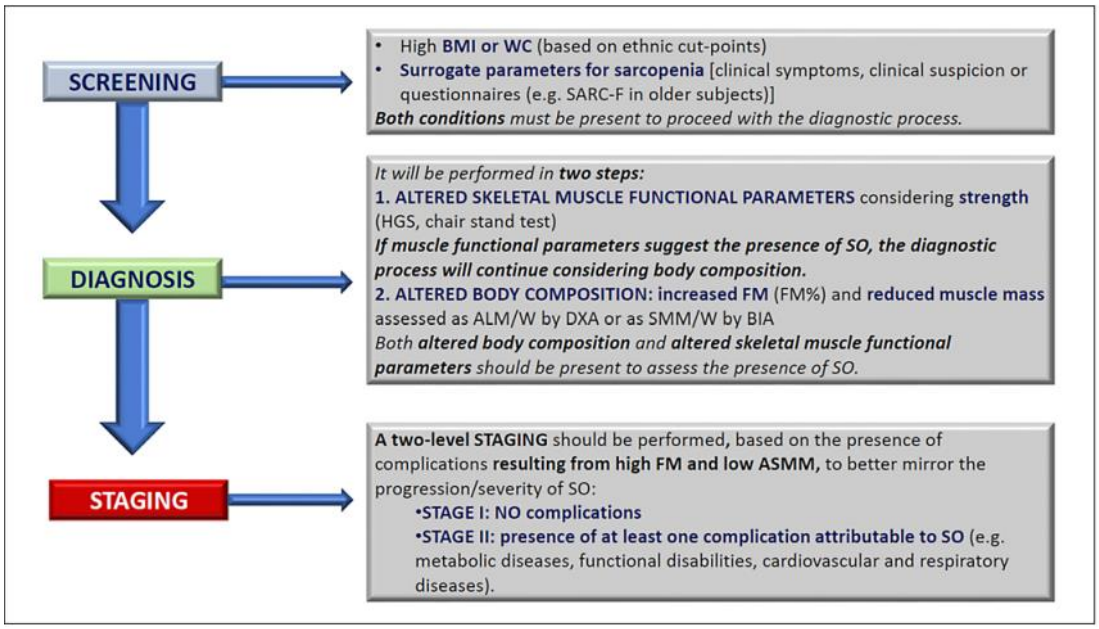

Fig. 1: Diagnostic procedure for the assessment of sarcopenic obesity. Abbreviations: ALM/W, appendicular lean mass adjusted for weight; ASMM, absolute skeletal muscle mass; BIA, bioelectrical impedance analysis; BMI, body mass index; DXA, dual X-ray absorptiometry; FM, fat mass; HGS, handgrip strength; SMM/W, skeletal muscle mass adjusted by weight; WC, waist circumference; SARC-F, Strength, Assistance with walking, Rising from a chair, Climbing stairs and Falls.] (Donini et al., 2022).

Globally, over 1.9 billion adults are overweight or obese, according to the World Health Organization (WHO). Simultaneously, sarcopenia affects between 10% and 30% of individuals aged 60 years and older, reaching rates above 50% in high-risk groups such as those with type 2 diabetes, institutionalized older adults, sedentary individuals, and patients with chronic inflammatory diseases (Batsis & Villareal, 2018). The convergence of aging, food insecurity, sedentary behavior, and obesity has rendered SO a highly prevalent and underrecognized clinical entity (Fonseca-Pérez et al., 2022; Axelrod, Dantas & Kirwan, 2023).

The growing incidence of SO is also linked to current obesity management paradigms, which often prioritize weight loss as the primary endpoint. This reductionist

approach overlooks the nuances of body composition and fails to address functional capacity. Emerging classifications of obesity now emphasize metabolic health and anthropometric indicators beyond body mass index (BMI), advocating for evaluation of waist circumference, fat distribution, and related complications to better guide therapeutic decisions (Rubino et al., 2025).

Pharmacotherapies that induce weight loss, particularly glucagon-like peptide-1 receptor agonists (GLP-1 RAs), have been shown to reduce lean mass as well as fat mass. Although the proportion of lean mass lost is generally smaller, concerns remain about their impact on muscle function, especially in older adults or those with pre-existing sarcopenia (Anyiam et al., 2025; Karakasis et al., 2025). Blaming the medication alone, however, is reductive. The primary limitation lies in inadequate clinical supervision: insufficient follow-up, lack of personalized interventions, and failure to incorporate anabolic support compromise outcomes. Pharmacological therapy should be seen as an adjunct; the principal risk arises from its unsupervised use in the absence of a structured clinical framework.

Clinical suspicion for SO should be heightened in individuals with chronic inflammatory diseases, organ failure (cardiac, renal, hepatic), neurodegenerative disorders, cancer, or endocrine conditions such as diabetes mellitus. Additional red flags include recent acute stressors, such as hospitalization, surgery, or involuntary weight loss, as well as signs of dynapenia, including recurrent falls, fatigue, muscle weakness, or progressive functional limitation (Donini et al., 2022).

Definitive diagnosis requires a two-step process: (1) evaluation of muscle strength and function (e.g., handgrip strength, chair rise test); and (2) analysis of body composition to detect increased fat mass percentage (FM%) and reduced skeletal muscle mass, using tools such as DXA or BIA. A diagnosis of sarcopenic obesity is established when both muscle dysfunction and adverse body composition are concurrently identified (Donini et al., 2022).

Failure to recognize and intervene early in SO results in a significant therapeutic bottleneck. As the condition progresses, patients exhibit accelerated frailty, functional dependency, and diminished response to conventional lifestyle interventions. Effective management requires integration of nutritional, physical, and pharmacological strategies tailored to the disease's pathophysiology. An anabolic-oriented therapeutic model is essential to interrupt the cycle of decline and restore functional independence and quality of life.

3.2 Pathophysiology of Sarcopenic Obesity

Sarcopenic obesity (SO) is a multifactorial and progressive syndrome marked by a pathological imbalance between adipose tissue expansion and skeletal muscle degradation. Unlike isolated sarcopenia or obesity, SO constitutes a unique clinical entity in which excess visceral fat coexists with qualitative and functional deterioration of muscle tissue. This dual dysfunction is sustained by complex, interrelated mechanisms involving chronic low-grade inflammation, insulin resistance, intramuscular lipid accumulation (myosteatosis), mitochondrial dysfunction, and reduced anabolic hormone signaling. The resulting decline in strength, mobility, metabolic adaptability, and regenerative capacity contributes to a vicious cycle of progressive disability (Prado et al., 2024; Axelrod, Dantas & Kirwan, 2023).

Aging is accompanied by a redistribution of fat, characterized by a reduction in subcutaneous adipose tissue (SAT) and an increase in visceral adipose tissue (VAT), along with a shift in adipokine secretion. Pro-inflammatory mediators (e.g., TNF-α, IL-6) become dominant, while protective adipokines such as adiponectin decline. This promotes a state of "inflammaging" exacerbated by tissue hypoxia, macrophage infiltration, and activation of pro-inflammatory pathways such as NF-κB and JNK. These processes directly disrupt muscle protein homeostasis, impair mitochondrial function, and suppress satellite cell activity (Li et al., 2022).

The bidirectional crosstalk between adipocytes and myocytes—mediated by adipokines, myokines, and adipomyokines—plays a central role in metabolic regulation, inflammation, thermogenesis, and tissue repair. In SO, this signaling network becomes dysregulated: excessive leptin (in the context of central resistance), elevated myostatin, and reduced expression of thermogenic genes such as *UCP1* and *PGC-1α* inhibit muscle regeneration and promote white adipose tissue expansion (Zamboni et al., 2022).

Myosteatosis, a hallmark of SO, refers to the infiltration of lipids both within and between muscle fibers—namely intramyocellular lipids (IMCL) and intermuscular adipose tissue (IMAT). This lipid accumulation impairs mitochondrial oxidative capacity, increases reactive oxygen species (ROS) production, and disrupts insulin signaling. The resulting anabolic resistance limits muscle hypertrophy and regeneration, even in the presence of adequate nutrient intake or preserved lean mass. Notably, muscle strength often declines earlier and more significantly than muscle mass, underscoring the importance of evaluating muscle quality rather than quantity alone (Kalinkovich & Livshits, 2017; Kim & Kim, 2021).

Insulin resistance is a central driver of sarcopenic obesity and is exacerbated by the accumulation of bioactive lipids—such as ceramides and diacylglycerols (DAGs)—within myocytes. These lipotoxic species activate cellular stress pathways and inhibit the PI3K/Akt/mTOR axis, which is essential for muscle protein synthesis and

anabolic maintenance (Axelrod, Dantas & Kirwan, 2023). In parallel, impaired mitochondrial flexibility reduces ATP production and limits substrate switching, favoring ectopic lipid storage and further metabolic dysfunction. Additionally, the secretion of beneficial myokines such as IL-15, irisin, and FGF-21 declines, reducing the capacity for white fat browning and lipolysis. This creates a self-perpetuating cycle of adiposity, muscle loss, and systemic dysfunction (Li et al., 2022).

Hormonal dysregulation also contributes significantly to SO. Age-related declines in testosterone, growth hormone (GH), insulin-like growth factor-1 (IGF-1), and dehydroepiandrosterone (DHEA) impair satellite cell activation and reduce the anabolic response to exercise and nutrition (Axelrod, Dantas & Kirwan, 2023). This hormonal environment promotes a chronic catabolic state and limits the effectiveness of standard rehabilitation efforts.

From a regenerative perspective, the inflammatory and lipotoxic milieu disrupts the muscle stem cell niche. In murine models of SO, mitochondrial dysfunction in muscle progenitor cells impairs regenerative capacity and blunts the response to anabolic stimuli. As a result, even with nutritional and physical interventions, muscle recovery remains incomplete (Axelrod, Dantas & Kirwan, 2023).

A pivotal concept in understanding SO is the **"metabaging cycle"**, a bidirectional pathological loop between adipose and muscle tissues (Ma & Shyh-Chang, 2022). In this model, chronic inflammation, insulin resistance, and lipid overload reinforce each other, leading to a downward spiral of muscle loss and metabolic decline that is difficult to reverse clinically.

Therapeutic implications arise directly from this integrated pathophysiology. Isolated interventions such as caloric restriction may exacerbate muscle loss, whereas multimodal strategies that combine resistance training, adequate protein intake, hormonal support, and, in selected cases, pharmacological agents (e.g., SARMs, GH secretagogues, PPAR agonists, or AAS) offer more promising outcomes. Monitoring of functional parameters and muscle quality indices, such as the LAMA/NAMA ratio on imaging, may guide individualized treatment and track progression of myosteatosis (Vieira et al., 2025).

3.3 Limitations of Conventional Therapies

The conventional management of sarcopenic obesity (SO) is primarily based on two pillars: (1) obesity pharmacotherapy, usually focused on inducing adherence to hypocaloric diets, and (2) exercise-based interventions combining aerobic and resistance training. While these strategies may yield satisfactory outcomes in cases of isolated obesity, they are frequently inadequate in addressing the complex pathophysiology of SO.

Caloric restriction without concurrent anabolic support, particularly when implemented without sufficient protein intake, leads to significant losses in fat-free mass (FFM). This is especially problematic in older adults and postmenopausal women, who already exhibit blunted anabolic responsiveness and diminished muscle regenerative capacity (Villareal et al., 2011; Donini et al., 2020). The resulting negative nitrogen balance has been linked to reduced physical performance, delayed recovery, and increased vulnerability to infections, falls, and hospitalizations (Gong et al., 2019).

Beyond physiological limitations, behavioral adherence represents another significant barrier. Many individuals with SO experience musculoskeletal pain, joint stiffness, chronic fatigue, or depressive symptoms, all of which impair adherence to structured exercise regimens (Ghiotto et al., 2022). In addition, aerobic training alone, when not integrated with resistance components has limited effects on muscle mass and may even exacerbate sarcopenia through increased muscle catabolism in an already compromised musculature (Chen et al., 2017).

Progressive resistance training (PRT) has consistently demonstrated efficacy in improving muscle strength, lean mass, and physical function (Peterson et al., 2011; Trouwborst et al., 2018, Câmara et al. 2012). However, its success depends on several factors, including proper intensity, load progression, nutritional adequacy (especially protein intake), supervision, and a permissive hormonal milieu. In individuals with advanced anabolic resistance, such as elderly adults or those with endocrine deficiencies, PRT alone may be insufficient to induce meaningful functional recovery in the absence of pharmacological support.

Furthermore, hormone replacement therapy (HRT), particularly estrogen-based regimens, remains underutilized due to longstanding misconceptions stemming from early interpretations of the WHI trial. Contemporary analyses suggest that when properly indicated, the risks of HRT are relatively low, and its benefits in preserving muscle mass and bone health may outweigh potential harms in specific populations (Greising et al., 2009). Nonetheless, the anabolic effect of HRT, although relevant, is typically modest and unlikely to reverse severe functional decline in advanced sarcopenia (Javed et al., 2019).

As SO progresses, patients may become refractory to conventional interventions. The interplay between chronic inflammation, mitochondrial dysfunction, hormonal deficits, and insulin resistance creates a biological environment resistant to lifestyle modification alone (Axelrod, Dantas & Kirwan, 2023). Even in cases of excellent adherence to diet and exercise, improvements in muscle strength, quality, or metabolic flexibility may remain minimal (Bhasin et al., 2005; Donini et al., 2020). These limitations reinforce the position of recent ESPEN and EASO consensus

statements, which acknowledge that nutritional and exercise-based strategies alone are insufficient to reverse moderate to severe SO.

Taken together, these insights support the need for a more integrated therapeutic approach, one that combines physical training, nutritional optimization, and, when appropriate, anabolic pharmacologic interventions, to overcome resistance mechanisms and improve functional outcomes in patients with refractory sarcopenic obesity.

3.4 Therapeutic Use of Anabolic Steroids

Anabolic androgenic steroids (AAS) represent one of the most extensively studied pharmacological strategies for the treatment of muscle wasting syndromes, particularly in populations affected by anabolic resistance, chronic catabolism, and functional decline (Basaria, Wahlstrom & Dobs, 2001; Bhasin et al., 2005). As illustrated in **Figure 3**, the therapeutic use of AAS may simultaneously address multiple components of sarcopenic obesity by promoting muscle hypertrophy, improving strength, reducing visceral adiposity, enhancing physical performance, and correcting endocrine deficiencies.

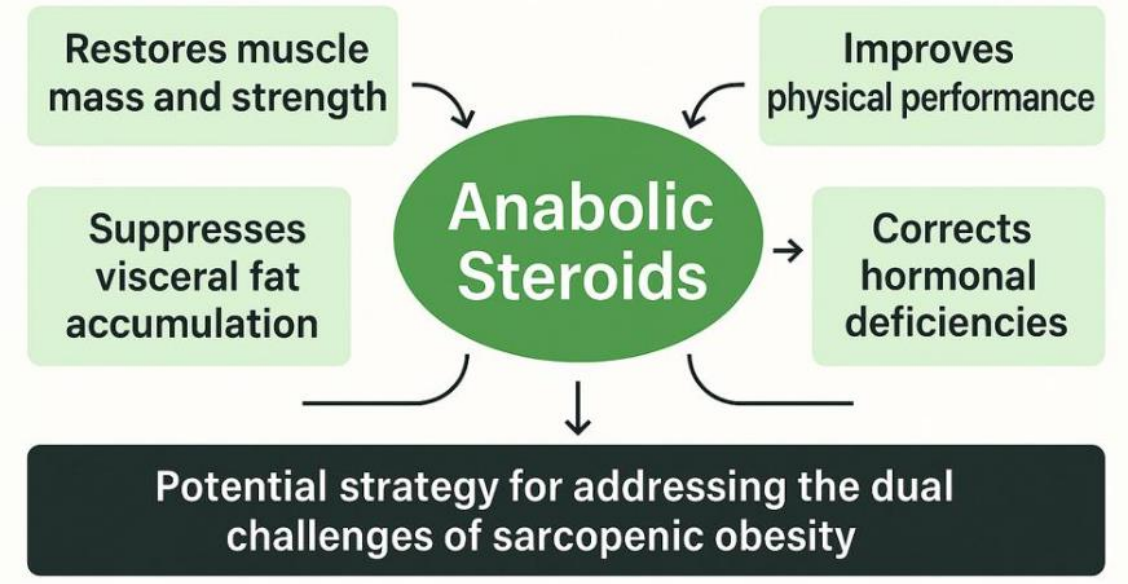

Fig. 3: Therapeutic use of anabolic steroids in sarcopenic obesity.

Despite the stigma associated with recreational and non-medical use of AAS, there is strong evidence that, when used under clinical supervision, these agents have clear therapeutic indications and demonstrate safety when administered for a limited duration. The clinical use of testosterone, oxandrolone, and nandrolone has been well documented in contexts such as sarcopenia, HIV-associated cachexia, liver disease, osteoporosis, and other chronic catabolic conditions (Grinspoon et al., 1998; Shahidi, 2001; Kong & Edmonds, 2002; Frisoli, Chaves, Pinheiro & Szejnfeld, 2005; Chan, Wong & Lee, 2006; Rambaldi & Gluud, 2006; Taylor, Laor & Warner, 2008; Sardar et al., 2010; Li et al., 2016; Sinclair et al., 2016; Power et al., 2022; Tapper, Chen & Parikh, 2025).

As proposed by (Câmara, 2024) there are four distinct scenarios for AAS use: replacement in hypogonadal men, therapeutic use in cases of dynapenia and catabolism, aesthetic or performance-driven use at moderately supraphysiological doses for defined periods, and underground abuse at highly supraphysiological doses. It is critical to separate these scenarios for an unbiased and medically appropriate analysis of therapeutic use versus abuse. And for clarity and ethical appropriateness, only the first two scenarios fall within legitimate medical practice.

3.4.1 Testosterone

Testosterone replacement therapy (TRT) is well established for male hypogonadism and is supported by extensive literature. In elderly hypogonadal men, TRT has been shown to increase lean body mass, reduce fat mass, enhance muscle strength, and improve quality of life (Bhasin et al., 2003; Snyder et al., 1999; Brochu et al., 2001). Testosterone also restores exercise responsiveness and has been associated with lower rates of hospitalization and improved functional outcomes in frail older men (Baillargeon et al., 2016).

In specific contexts, supraphysiological doses may also provide therapeutic benefit. The landmark study by Bhasin et al. (1996) demonstrated that 600 mg/week of testosterone enanthate significantly increased muscle size, strength, and cross-sectional area in eugonadal men, particularly when combined with resistance training. Storer et al. (2003) further confirmed the dose-response effect of testosterone on strength and power, without compromising muscle endurance.

In HIV-positive men with wasting syndrome, a supraphysiological testosterone regimen combined with oxandrolone produced greater gains in lean mass than either agent alone or resistance training alone, even in eugonadal individuals (Strawford et al., 1999). In eugonadal men with osteoporosis, TRT has been shown to improve bone mineral density and physical function (Anderson et al., 1997). Similarly, in patients with cirrhosis, testosterone administration has led to improved muscle strength and metabolic status without significant adverse effects (Sinclair et al., 2016; Tapper et al., 2025).

Taken together, these findings suggest that when used judiciously, under clinical supervision, and for a defined therapeutic purpose, supraphysiological testosterone may serve as a safe and effective intervention in selected cases of sarcopenic obesity.

3.4.2 Oxandrolone

Oxandrolone is a non-aromatizable synthetic derivative of dihydrotestosterone (DHT), characterized by high anabolic potency and low androgenic activity. It is particularly suitable for female patients due to its low virilization profile and favorable hepatic safety at therapeutic doses (Orr R, et al, 2004).

Oxandrolone has been extensively studied in catabolic conditions such as sarcopenia, HIV-associated wasting, severe burns, Turner syndrome, and chronic illness (Orr R, et al, 2004).

In a randomized trial involving elderly women, 10 mg/day of oxandrolone for 12 weeks significantly increased lean body mass in the limbs and trunk, while reducing fat mass, particularly in the lower extremities, without increasing adverse events (Mavros et al., 2015).

In HIV-positive men, oxandrolone administration for 12 weeks in doses ranging from 20 to 80 mg/day resulted in dose-dependent increases in lean body mass and overall weight, with favorable tolerability profiles at 40 and 80 mg (Grunfeld et al., 2006). In pediatric and adult burn patients, oxandrolone improved lean mass retention, bone health, and muscle strength, with persistent benefits even after treatment discontinuation (Porro et al., 2012; Real et al., 2014). In girls with Turner syndrome, early initiation of oxandrolone significantly increased final height without serious adverse effects (Zeger et al., 2011).

Typical therapeutic dosing ranges from 0.1 to 0.2 mg/kg/day, as seen in previously (Orr R, et al, 2004; Câmara LC, et al, 2023), oral route, low hepatotoxicity at physiological doses, and proven efficacy in increasing lean mass, make it a valuable option in female patients with sarcopenic obesity.

3.4.3 Nandrolone

Nandrolone decanoate is a long-acting injectable AAS with low aromatization, favorable safety profile, and proven efficacy in multiple clinical contexts, including osteoporosis, sarcopenia, HIV-associated wasting, and chronic kidney disease (Kochakian C, 1976; Taylor W, 2002; Geusens P, 1995).

In postmenopausal women with osteoporosis, nandrolone improves bone mineral density, increases lean mass, and enhances physical performance, with benefits sustained over long-term follow-up (Hassager et al., 1989; Geusens, 1995; Dave et al., 2023; Câmara et al., 2023). In HIV-infected men and women, nandrolone therapy increased lean body mass and was associated with superior perceived functional improvements when compared to testosterone in some domains (Mulligan et al., 2005; Sardar et al., 2010).

Compared to recombinant human growth hormone (rhGH), nandrolone has been shown to produce equivalent gains in fat-free mass at a lower cost and with fewer adverse effects (Storer et al., 2005). In dialysis patients, monthly doses of 50–100 mg significantly improved muscle mass, hemoglobin levels, and body composition, with additional anti-anemic effects (Johansen, Mulligan & Schambelan, 1999).

In clinical practice, nandrolone's monthly injectable administration, minimal virilizing effects, and metabolic safety profile make it especially suitable for elderly or chronically ill individuals. When combined with resistance training, the anabolic synergy exceeds the effects of either intervention alone (Falqueto, Santos & Manfredi, 2022).

Importantly, withholding clinically indicated AAS therapy due to stigma or misconceptions may deprive patients of a viable intervention with the potential to reverse frailty and functional decline. In properly selected cases, when prescribed within therapeutic ranges and with defined functional goals, AAS should be considered not only medically appropriate, but ethically necessary (Kochakian C, 1976; Taylor W, 2002).

3.5 Ethical and Plausible Use of Anabolic Steroids in Sarcopenic Obesity

Given the complex pathophysiology and therapeutic refractoriness of sarcopenic obesity (SO), the use of anabolic androgenic steroids (AAS) emerges as a scientifically grounded and ethically defensible pharmacological strategy. When prescribed appropriately, AAS aim to restore muscle mass and function, mitigate chronic catabolism, and interrupt the trajectory of progressive functional decline (Kochakian C, 1976; Taylor W, 2002; Orr R, et al, 2004).

This therapeutic approach is neither experimental nor anecdotal. It is supported by a robust body of evidence derived from randomized controlled trials, systematic reviews, and expert consensus statements addressing sarcopenia, cachexia, and other clinical states marked by muscle wasting. The rationale for AAS use in SO is

underpinned by strong physiological plausibility: AAS have demonstrated the capacity to reverse core pathogenic mechanisms of the syndrome, including anabolic resistance, mitochondrial dysfunction, systemic inflammation, and impaired muscle regeneration (Falqueto H, et al, 2022; Câmara LC, et al, 2023; Basaria S, et al, 2001; Woerdeman, J., & De Ronde, W. 2011).

Moreover, when used under clinical supervision, AAS have been shown to improve muscle quality by enhancing specific strength, functional performance, and mobility, critical factors in the prevention of falls, hospitalization, and loss of independence in older adults (Falqueto H, et al, 2022; Câmara LC, et al, 2023; Basaria S, et al, 2001; Woerdeman, J., & De Ronde, W. 2011).

In sarcopenic populations, including elderly, HIV-positive, cirrhotic, or nephropathic patients, testosterone, oxandrolone, and nandrolone have produced clinically meaningful improvements with a low incidence of adverse effects when dosed within therapeutic limits (Shahidi, 2001; Mulligan, 2005; Johansen et al., 1999; Kochakian C, 1976; Taylor W, 2002; Orr R, et al, 2004).

It is imperative that the distinction between therapeutic use and recreational abuse be clearly established in both clinical education and medical guidelines (Câmara LC, 2024). Conflating these two scenarios undermines scientific discourse, perpetuates stigma, and may ultimately compromise patient autonomy and access to appropriate care. **Figure 4** illustrates the ethical divergence between these practices.

Therapeutic Use	Recreational Abuse
Supervised	Unsupervised
Physiological Doses	Supraphysiological Doses
Defined Indications	Aesthetic/Misuse
Low Adverse Events	High Risk

Fig. 4: Ethical use versus abuse of anabolic steroids.

The failure to consider evidence-based anabolic therapies in patients with refractory SO should be recognized as a lapse in care (Falqueto H, et al. 2022). When prescribed with clear functional objectives, such as restoring mobility, preventing falls, improving quality of life, and reducing hospitalizations, AAS use is not controversial, but rather consistent with ethical and evidence-based medical practice (Falqueto H, et al. 2022).

Clinicians must balance therapeutic innovation with rigorous oversight, ensuring that anabolic agents are reserved for appropriate indications and administered with the same degree of responsibility as any other class of medication. Dismissing the therapeutic potential of AAS solely on the basis of non-medical misuse would represent a missed opportunity for functional rehabilitation in a growing and underserved clinical population (Kochakian C, 1976; Taylor W, 2002; Orr R, et al, 2004; Câmara LC, 2024; Morgentaler A, et al. 2024; Hoffman JR, & Ratamess NA 2006).

In this sense, the adverse outcomes documented in observational studies related to AAS abuse do not belong (nor should they ever be considered) as part of the same clinical context as therapeutic use (Câmara LC, 2024; Hoffman JR, & Ratamess NA 2006).

On one side, we observe a completely uncontrolled scenario, characterized by supraphysiological dosing, the simultaneous use of multiple AAS compounds, the addition of various other substances (polypharmacy, including diuretics, thyroid hormones, ephedrine and amphetamines, growth hormone, insulin, and beta-agonists), and the concomitant abuse of legal (e.g., alcohol and tobacco) and illicit drugs (e.g., cocaine, cannabis, and heroin). Furthermore, the use of AAS sourced from the black market(which are adulterated in at least 30–40% of cases) combined with the absence of professional oversight and prolonged, uninterrupted use, further exacerbates the risks.

In contrast, randomized controlled trials evaluate pharmaceutical-grade AAS administered at therapeutic or, at most, moderately supraphysiological doses, typically as monotherapy. These are prescribed under strict clinical supervision, including pre-treatment screening, regular laboratory monitoring, guidance regarding polypharmacy and recreational drug use, and administration for the shortest duration necessary, with precise clinical indications and clearly defined therapeutic goals (Kochakian C, 1976; Taylor W, 2002; Orr R, et al, 2004; Falqueto H, et al. 2022; Câmara LC, 2024; Câmara LC et al, 2025).

4. CONCLUSION

Sarcopenic obesity represents a neglected and insufficiently addressed clinical phenotype whose prevalence is rising silently among aging, sedentary, and metabolically compromised populations. By combining the inflammatory burden of visceral obesity with the functional deterioration of sarcopenia, SO establishes a state of systemic vulnerability that accelerates frailty, disability, and loss of autonomy.

Conventional therapeutic approaches that focus solely on weight loss or physical rehabilitation are frequently inadequate, especially in advanced stages where anabolic resistance and metabolic dysfunction compromise treatment response. In such cases, the incorporation of anabolic pharmacologic interventions may be necessary to achieve clinically meaningful outcomes.

While further large-scale randomized controlled trials are warranted, existing literature provides compelling preliminary evidence supporting the therapeutic potential of anabolic androgenic steroids (AAS) in contexts such as sarcopenia, cachexia, HIV-related wasting, and chronic liver or kidney disease. The same pathophysiological rationale applies to sarcopenic obesity, potentially with even greater urgency due to its compounded mechanisms of decline.

As novel agents such as bimagrumab (Kanbay M, et al, 2024) and selective androgen receptor modulators (SARMs) (Wen J, et al, 2025) continue to be investigated for broad clinical use, currently available AAS may serve as pragmatic and effective tools in carefully selected patients. AAS use, when clinically indicated, properly dosed, and rigorously monitored, should not be dismissed based on stigma or misuse narratives, but rather evaluated through the lens of evidence-based, patient-centered care.

Scientific discourse must clearly distinguish between therapeutic use and abuse. AAS, when administered ethically and judiciously, may represent a valuable component of multimodal rehabilitation strategies for sarcopenic obesity. Their role in this setting, as we think, merits open discussion, critical reassessment, and thoughtful clinical application, not categorical exclusion.

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