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Sarcopenic Obesity and Hormones: A Narrative Review

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Abstract

Sarcopenic Obesity (SO) is an increasingly prominent public health issue associated with the aging process, characterized by a reduction in muscle mass and an elevated body fat percentage. As the condition progresses, it often leads to an increased risk of cardiovascular events, cognitive dysfunction, frailty, fractures, prolonged hospitalization, and even mortality. However, the mechanisms underlying the development and progression of SO remain incompletely elucidated, and there are currently no systematic and standardized therapeutic approaches specifically targeting this condition. Hormonal imbalances play a significant role in the pathogenesis of SO. We conducted a systematic search of the published literature available on PubMed and summarized the pertinent articles. This review summarizes the current literature on hormones implicated in SO and discusses the potential role of various hormones in its treatment. Multiple hormones play pivotal roles in their pathophysiology: deficiencies in vitamin D, testosterone, IGF-1, and GH are implicated in the loss of muscle mass and concomitant accumulation of adipose tissue. Conversely, elevated levels of catabolic hormones such as cortisol may exacerbate this process. Furthermore, dysregulation of estrogen and thyroid hormones is also linked to alterations in body composition.

Keywords

Sarcopenic Obesity, Hormones, Endocrine Dysregulation, Body Composition

1. Introduction

Aging and lifestyle modifications lead to significant alterations in human body composition. Sarcopenic Obesity (SO) has emerged as an increasingly prominent health concern among the elderly population. The concept built upon the foun-

dation of sarcopenia research, was first introduced in 1996, initially described as "reduced lean mass with excess fat as a percentage of body weight" [1]. Growing recognition of its adverse health outcomes and associated healthcare burden has accelerated research into its development and progression. Since 2010, multiple international working groups have been established, leading to progressive refinement of diagnostic criteria for SO. A significant milestone was reached in 2022 when the European Society for Clinical Nutrition and Metabolism (ESPEN) and the European Association for the Study of Obesity (EASO) jointly published the first international diagnostic consensus, defining SO as "the co-existence of adiposity-based obesity and sarcopenia" [2]. A notable limitation of this consensus, however, is the lack of universally established diagnostic cut-off values, particularly reference standards tailored for Chinese populations. In May 2025, the Asia-Oceania Association for the Study of Obesity released "The Asia-Oceania Consensus: Definitions and Diagnostic Criteria for Sarcopenic Obesity" [3], representing the first diagnostic consensus specifically developed for Asian and Oceanian populations. Furthermore, research into SO has identified several related subtypes, such as osteopenic-sarcopenic obesity, dynapenic obesity (the coexistence of decreased muscle strength and obesity), and low muscle mass obesity.

SO is recognized as a clinical and functional condition strongly associated with frailty, fractures, cognitive dysfunction, cardiovascular disease, and increased all-cause mortality [4]-[7]. Against the backdrop of a rapidly aging global population, its prevalence continues to rise, imposing a substantial burden on individuals and society. Despite its clinical significance, the precise pathophysiological mechanisms underlying SO remain incompletely understood. They are believed to involve a complex interplay of multiple factors, including lifestyle changes, aging, oxidative stress, chronic low-grade inflammation, insulin resistance, and hormonal dysregulation [5] [8]-[10].

Hormonal alterations are not merely independent risk factors for sarcopenia or obesity alone. Age-related endocrine system decline synergistically interacts with these conditions, disrupting the dynamic balance of muscle protein and fat metabolism, thereby promoting the onset and progression of SO. Although existing research has explored the relationships between hormones and sarcopenia, obesity, and aging separately, the complex crosstalk between muscle and adipose tissue suggests that the hormonal profile in SO may be distinct, rather than a simple combination of features seen in the isolated conditions. Therefore, this review aims to systematically elucidate the characteristic hormonal changes associated with SO, providing a theoretical basis for mechanistic research and clinical intervention strategies.

2. 25-Hydroxyvitamin D(25(OH)D)

Vitamin D plays a critical role in maintaining calcium homeostasis and regulating bone metabolism. Recent evidence indicates that vitamin D is also implicated in skeletal muscle metabolism [11], influencing it through mechanisms such as mod-

ulating mitochondrial biogenesis and oxidative activity in muscle tissue [12]. On the other hand, vitamin D deficiency may compromise immune function, reduce antioxidant and anti-inflammatory capacity, and adversely affect adipogenesis, lipolysis, and adipose tissue inflammation [13]. A large-scale Mendelian randomization study utilizing the UK Biobank data confirmed a causal relationship between 25(OH)D levels and skeletal muscle health [14]. Cross-sectional studies have reported a significant correlation between low 25(OH)D levels and the presence of SO in the general population [15]-[17]. However, some studies suggest this relationship may be sex-specific, being significant only in men [18]. Associations between low 25(OH)D concentrations and SO have also been reported in populations with comorbidities such as chronic kidney disease [19], alcohol use disorder [20], and rheumatoid arthritis [21]. Furthermore, a longitudinal observational study with a median follow-up of 8.74 years indicated that higher 25(OH)D levels were associated with a lower incidence of SO [22]. Interestingly, conflicting results exist. Several Korean cross-sectional studies found that obese individuals had significantly lower 25(OH)D levels compared to non-obese individuals, irrespective of sarcopenia status [23] [24], although this difference was not observed in women in one study [23]. Most, but not all, interventional studies suggest that vitamin D supplementation can beneficially modify body composition. In a randomized, double-blind, multicenter trial involving 248 participants (baseline 25(OH)D between 10 - 30 ng/mL), daily supplementation with 3750 IU of vitamin D3 for 12 months did not improve sarcopenia or obesity parameters compared to a lower dose of 600 IU [25]. Another 6-month randomized controlled trial in 128 participants (mean baseline 25(OH)D = 12.92 ± 4.3 ng/ml) showed that supplementation with 10,000 IU of vitamin D3 three times per week significantly increased muscle mass and reduced fat mass compared to controls, although handgrip strength remained unchanged [26]. An Italian nutritional intervention study demonstrated that supplementation with whey protein, leucine, and vitamin D in postmenopausal women with SO significantly reduced fat mass while improving muscle strength and preserving muscle mass [27]. Additionally, research on SO-related subtypes, such as dynapenic obesity [28] and osteosarcopenic obesity [29] [30], has also reported relationships between 25(OH)D levels, fat mass, and muscle function.

3. Gonad-Axis

3.1. Testosterone (T)

As a metabolic hormone acting on multiple tissues and organs, T exerts significant regulatory effects on skeletal muscle, bone, and adipose tissue. Studies indicate a progressive age-related decline in T levels [31], which is associated with an increased risk of fractures, sarcopenia, and SO in older adults [32]-[34]. A study on aging in Korean men demonstrated that low free T levels were not only associated with a higher risk of sarcopenia and obesity individually but also significantly increased the risk of SO [34]. Furthermore, androgen deprivation therapy in patients with prostate cancer frequently leads to SO as an adverse outcome [35] [36].

Among men living with HIV, those with SO exhibited significantly lower levels of estradiol, total T, and free T compared to those without SO [37]. Although animal studies have confirmed the beneficial effects of T on increasing muscle mass and reducing fat [38], the efficacy of exogenous testosterone supplementation for improving body composition in individuals with SO remains debatable. A small study in hypogonadal men, who underwent dietary and exercise control along with a pre-treatment androgen washout period of at least 12 weeks, found that 10 weeks of weekly 100 mg T supplementation significantly improved fat-free mass, muscle size, and strength, but did not significantly alter fat mass [39]. This aligns with findings from a US study employing 6 months of sublingual T replacement in hypogonadal men [40]. In contrast, another study using the same weekly 100 mg T regimen over 18 months reported increases in lean body mass accompanied by significant reductions in fat mass [41]. Another 3-year intervention using a scrotal patch for T delivery resulted in significant increases in fat-free mass, a non-significant trend towards fat mass reduction, and no change in grip strength [42]. Long-term T gel replacement therapy has been shown to improve lean body mass and reduce fat mass, though the effect on fat mass was observed only in younger patients, and grip strength remained unaffected [43]. A randomized double-blind trial in older men found that a group with low T levels had higher fat mass at baseline. After one year of T intervention compared to a placebo control, the treatment group exhibited significantly increased fat-free mass and reduced fat mass, yet no significant differences were observed in grip strength [44]. Collectively, these findings suggest that exogenous T supplementation can improve muscle mass. Although studies have elucidated the mechanism by which low T levels affect body composition—promoting fat accumulation through reduced β-oxidation and lipolysis, along with increased adipogenesis—increased adipose tissue mass, in turn, suppresses the hypothalamic-pituitary-testicular axis by elevating pro-inflammatory cytokines and insulin resistance, creating a bidirectional interplay [45]. On the other hand, low free T levels also lead to muscle loss by impairing myogenesis, satellite cell differentiation, and protein synthesis, while upregulating the expression of proteins associated with muscle breakdown [46]. Furthermore, inflammatory cytokines elevated by fat accumulation can promote catabolism in skeletal muscle [47]. The interaction of these multiple factors collectively contributes to the development of SO. However, a significant point of contention is that most interventional studies have been conducted in hypogonadal men. It remains unclear whether individuals with SO who do not meet the criteria for hypogonadism would similarly benefit from T therapy in terms of body composition. Furthermore, variations in administration routes and dosages, which have demonstrated distinct pharmacokinetics and clinical outcomes—as seen with T gels versus patches [48] [49]—must be considered when interpreting these results.

3.2. Estrogen

Estrogen exerts both direct and indirect effects on bone to influence bone formation and is also a significant regulator of other body composition components

[50]. Mice with aromatase deficiency or inactivated Estrogen Receptor-alpha (Erα) genes demonstrate a marked increase in fat mass [51] [52]. Murine studies indicate that signaling through both the androgen receptor and ERα is crucial for maintaining muscle mass, and that activation of ERα alone is sufficient to reduce adiposity [53]. A study published in the New England Journal of Medicine, which administered varying doses of T to healthy men with or without concomitant inhibition of testosterone-to-estrogen conversion, not only underscored the relationship between T and muscle mass, size, and strength but also highlighted the correlation between estrogen deficiency and increased fat accumulation [54]. Given the close metabolic relationship between T and estrogen—where over half of the estrogen in men is derived from T conversion—a decline in androgen levels directly leads to reduced estrogen synthesis. The synergistic action of both hormones contributes to the pathogenesis of SO. This provides a plausible explanation for the observed improvements in body composition following T intervention mentioned previously: the benefits may stem not only from the direct physiological actions of T itself but also from the combined effects of increased estrogen levels resulting from its aromatization. A meta-analysis of 23 studies indicated that estrogen-based therapy can indeed improve muscle strength [55]. However, studies have also reported a range of adverse outcomes associated with estrogen supplementation in postmenopausal women, including increased risks of breast cancer, cardiovascular disease, and pulmonary embolism [56]. Consequently, estrogen replacement is not currently recommended for the prevention or treatment of declining muscle strength in postmenopausal women.

4. Growth Hormone-Insulin-like Growth Factor-1 Axis

4.1. Growth Hormone (GH)

Growth Hormone (GH) is an anabolic hormone whose secretion declines with age. It plays a role in protein metabolism within skeletal muscle [57] and significantly influences lipid metabolism. GH exerts a lipolytic effect, primarily mobilizing Free Fatty Acids (FFAs) from visceral adipose tissue over subcutaneous fat by increasing the activity of Hormone-Sensitive Lipase (HSL) [58]. Research indicates that GH secretion peaks during adolescence and begins to decline in adulthood, with daily secretion in elderly men being 5 to 20 times lower than in young adults [59]-[61]. Beyond aging, increased abdominal visceral fat is strongly associated with reduced pituitary GH secretion [62]. Although obesity itself suppresses GH secretion, it has been reported that individuals with SO exhibit a more profound suppression of GH secretion compared to those with obesity alone [63]. Early studies in patients with congenital GH deficiency, characterized by markedly low levels of both GH and IGF-1, revealed that this population typically presents with increased fat mass and decreased muscle mass [64]. Several intervention studies have found that GH supplementation in individuals with obesity effectively reduces fat mass [65], although some evidence suggests this effect may be sex-dependent [66]. In one study designed to test the hypothesis that GH re-

duces fat mass and increases lean body mass, healthy older men received exogenous GH three times per week for six months. The intervention resulted in an 8.8% increase in lean body mass, a 14.4% reduction in adipose tissue mass, and a 7.1% increase in skin thickness, whereas no significant changes were observed in the control group. This led to the conclusion that age-related GH deficiency contributes to the decline in lean mass, expansion of adipose tissue, and thinning of the skin in the elderly [67]. Another study found that while exogenous testosterone supplementation in community-dwelling older men increased lean body mass, muscle strength, and reduced fat mass, co-administration of GH augmented these beneficial effects on body composition [68]. Current evidence suggests that GH has a significant impact on body composition. However, there is insufficient evidence to support the routine use of exogenous GH supplementation in specific populations for this purpose. Furthermore, future research must carefully consider the potential for adverse effects associated with GH therapy.

4.2. Insulin-Like Growth Factor-1 (IGF-1)

IGF-1 is an anabolic hormone whose levels decline with age. It promotes muscle repair and maintenance by enhancing cell cycle progression, proliferation, and differentiation [69], and by activating satellite cells, stimulating their proliferation and fusion [70]. Ultimately, IGF-1 exerts a beneficial effect on skeletal muscle by reducing proteolysis [71]. Animal studies have demonstrated that overexpression of IGF-1 in skeletal muscle induces hypertrophy and regeneration, while preserving muscle strength and function in both aged mouse models and those with neuromuscular disease [72] [73]. Conversely, reduced IGF-1 expression in obese mice exacerbates muscle loss [74]. Epidemiological and clinical studies consistently report a strong association between circulating IGF-1 levels, muscle function, and fat mass. A cross-sectional study suggested that impairment of the Growth Hormone (GH)/IGF-1 axis is associated with an increased risk of SO and ectopic fat deposition in the liver [75]. An intervention study involving 24 healthy volunteers found that leucine supplementation revealed age-related declines in muscle strength and endurance, which were correlated with lower circulating IGF-1 levels. This was attributed to reduced myosin heavy chain synthesis and impaired mitochondrial protein synthesis [76]. Patients with heart failure exhibit significantly lower IGF-1 levels compared to matched healthy controls [77]. However, aerobic exercise in these patients can increase IGF-1 expression, downregulate catabolic factors like myostatin, and improve skeletal muscle function [78]. Similar benefits are observed in individuals with type 2 diabetes, where combined aerobic and resistance training improves muscle function, reduces fat mass, and elevates serum IGF-1 levels [79]. In breast cancer patients, 16 weeks of combined aerobic and resistance training led to increased IGF-1 levels, significant gains in lean body mass, and reduced BMI compared to baseline and control groups [80]. A randomized controlled trial in older adults with SO compared 8 weeks of resistance training, aerobic exercise, and combined training against a non-exercising control. All ex-

ercise groups showed significant improvements in muscle mass, fat mass, and serum IGF-1 levels compared to the control, with resistance training yielding particularly notable effects on IGF-1 [81]. Several meta-analyses have further substantiated that exercise training effectively improves body composition and elevates IGF-1 levels in individuals with SO [82] [83]. Another exercise intervention in women with obesity and sarcopenia demonstrated that 12 weeks of circuit training improved fat-free mass, fat mass, and serum IGF-1 levels, while also reducing cardiovascular risk factors and inflammatory markers [84]. Similarly, 36 weeks of personalized resistance training improved body composition and IGF-1 levels in women with probable SO [85]. In conclusion, the influence of IGF-1 on body composition is evident. Utilizing exercise as a modality to elevate endogenous IGF-1 levels appears to be a viable strategy for improving muscle function and reducing adiposity.

5. Hypothalamic-Pituitary-Adrenal Axis

5.1. Cortisol

Cortisol, a well-known catabolic hormone, promotes protein degradation by activating the ubiquitin-proteasome and lysosomal systems, while simultaneously inhibiting protein synthesis [86]. Furthermore, it contributes to muscle loss, dysfunction, and fat deposition by antagonizing the actions of anabolic hormones such as testosterone and IGF-1 [87] [88]. An elevated cortisol-to-dehydroepiandrosterone sulfate (DHEA-S) ratio has been identified as an independent risk factor for sarcopenia, likely reflecting increased catabolic activity from cortisol coupled with diminished protective effects from DHEA-S [89]. Consistently, elevated levels of both serum and salivary cortisol have been associated with lower skeletal muscle mass and impaired muscle function [90] [91]. Individuals with obesity often present with lower muscle mass, lower bone mass, higher concentrations of nocturnal salivary cortisol, and elevated circulating high-sensitivity C-reactive protein compared to lean controls [92]. Previous research has confirmed that sleep deprivation elevates cortisol levels, which are strongly associated with increased fat mass and sarcopenia. Additionally, elevated cortisol drives increased levels of multiple inflammatory factors and is additionally closely associated with insulin resistance [93]. The activation of inflammatory cytokines, reduced insulin sensitivity, and increased food intake linked to enhanced dopamine receptor activity are recognized as significant contributors to the development of both obesity and sarcopenia [94]. Although a minority of studies have reported no significant difference in salivary cortisol levels between individuals with SO and control groups [95], such discrepancies in findings may be attributed to variations in study populations and methodological differences in assessment techniques.

5.2. Dehydroepiandrosterone (DHEA)

DHEA serves as a precursor to sex steroid hormones. It is converted to testosterone via enzymes including 17β-hydroxysteroid dehydrogenase (17β-HSD) and

3β-HSD [96]. Testosterone can subsequently be metabolized into 5α-Dihydrotestosterone (DHT) by 5α-reductase, or into estrogens via the action of aromatase cytochrome P-450 (P450arom). The presence of these multiple catalytic enzymes in skeletal muscle suggests that muscle tissue can locally synthesize and metabolize various sex steroids from DHEA [97]. As no specific receptor for DHEA has been identified to date, its effects on muscle and adipose tissue are believed to be mediated primarily through its conversion to active metabolites like testosterone and estrogen. Additionally, DHEA may indirectly influence body composition by modulating the levels of other hormones, for instance, by increasing bioavailable IGF-1 through the reduction of IGF-binding protein levels [70]. Limited interventional studies indicate that DHEA supplementation can elevate testosterone levels in women and increase IGF-1 levels in men. However, these hormonal changes have not consistently translated into significant improvements in muscle mass or function [98]. A systematic review summarizing the effects of DHEA supplementation on body composition and physical function in older adults concluded that there is currently no definitive evidence supporting its efficacy for enhancing muscle mass or function [99]. Given the limited number of studies, coupled with substantial heterogeneity in intervention duration, DHEA dosage, and participant characteristics, larger and more rigorous clinical trials are warranted to substantiate these findings.

6. Thyroid Hormones

Thyroid hormones are established as key regulators of skeletal muscle regeneration, development, and metabolism, exerting their effects through mechanisms such as influencing myogenesis, the proportion of type II fibers, and mitochondrial function [100]. Several cross-sectional studies conducted in euthyroid populations have reported positive correlations between Free Triiodothyronine (FT3) levels and skeletal muscle mass, handgrip strength, and physical performance. These studies found no significant associations with Free Thyroxine (FT4) or thyrotropin (TSH) [101] [102]. The skeletal muscle mass index has been found to correlate negatively with FT4 and positively with FT3, an observation noted exclusively in men [103]. A large-scale Korean longitudinal study similarly identified an inverse relationship between low muscle mass and FT4 levels in men, but not in women [104]. Another extensive longitudinal study, after adjusting for multiple confounders, corroborated that higher FT3 levels and a higher FT3/FT4 ratio were positively associated with handgrip strength [105]. Numerous studies have also explored the relationship between thyroid hormones and obesity. While no significant differences in TSH, FT4, or total T4 levels were observed between overweight and normal-weight individuals [106] [107], obese individuals were found to have normal [106] or elevated FT3 levels [108]. Similarly, another investigation reported that although FT3 and the FT3/FT4 ratio were not associated with visceral fat, elevated FT3 levels were independently correlated with abdominal subcutaneous fat [109]. Earlier research suggested that the elevation in

FT3 observed in obesity might represent an adaptive response, as FT3 levels do not directly correlate with body weight but can be increased by higher caloric intake [110]. A recent study further investigating the relationship between thyroid hormones and body composition found that FT3 was positively associated with both fat mass and the skeletal muscle mass index, while FT4 was positively correlated with fat mass. TSH showed no significant association with any of these parameters. Upon stratifying participants with a BMI ≥ 24 kg/m^2 into high-fat/low-muscle, intermediate, and low-fat/high-muscle groups, a positive correlation was identified between FT3 and the co-occurrence of obesity and low muscle mass [111]. In summary, the relationship between thyroid hormones, adipose tissue distribution, and skeletal muscle mass remains incompletely elucidated and warrants further investigation.

7. Parathyroid Hormone [PTH]

PTH, secreted by the parathyroid glands, is a key regulator of calcium and phosphate homeostasis and bone metabolism, operating within a negative feedback loop with serum calcium levels [112]. The kidney is responsible for synthesizing 1,25-dihydroxyvitamin D (1,25(OH)2D). Consequently, in chronic kidney disease, declining serum vitamin D levels disrupt this regulation, typically leading to elevated PTH concentrations [113]. Notably, even in individuals with normal renal function, those with SO present with lower vitamin D levels and concomitantly higher PTH levels compared to healthy controls [17]. Interestingly, a large-scale Korean study found that although both sarcopenia and SO were significantly inversely correlated with vitamin D levels, the association between serum PTH and sarcopenia lost statistical significance after adjustment for BMI [24]. Other research has suggested that elevated PTH levels may be independently associated with obesity itself [114]. While it remains unclear whether alterations in PTH levels in SO are primarily a consequence of vitamin D deficiency or a direct effect of the condition, most interventional studies indicate that vitamin D supplementation in individuals with SO not only improves body composition but also effectively reduces PTH levels.

8. Conclusions

This review systematically examines the hormonal profile of SO. Multiple hormones play significant roles in the pathophysiology of SO: deficiencies in vitamin D, testosterone, IGF-1, and GH are associated with reduced muscle mass and increased adipose tissue accumulation. Conversely, elevated levels of catabolic hormones such as cortisol may exacerbate this process, while imbalances in estrogen and thyroid hormones are also linked to alterations in body composition. These hormones interact with each other, collectively forming a complex endocrine regulatory network in SO.

Regarding the management of sarcopenic obesity, current evidence supports supplementation with 25(OH)D in specific populations; however, no clear guide-

lines recommend intervention with other hormones. Beyond hormonal approaches, the benefits of nutritional interventions and enhanced physical exercise as non-hormonal therapies are well-established.

Nevertheless, most existing studies have not directly focused on individuals with SO but have instead indirectly analyzed the relationships between various hormones and body composition. Furthermore, the efficacy and safety of interventions targeting multiple hormones in SO populations remain unclear. Future research should delve deeper into the underlying mechanisms and provide a basis for personalized treatment strategies.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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