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Molecular and Systemic Awarness into Sarcopenic Obesity: The Inflammatory Connection

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Description

Sarcopenic obesity is a complex condition characterized by the coexistence of reduced muscle mass and function (sarcopenia) and excessive fat accumulation (obesity). Chronic low-grade inflammation, often associated with aging and obesity, has emerged as a central factor in the pathogenesis of sarcopenic obesity. The role of chronic inflammation in the development and progression of sarcopenic obesity, highlighting molecular mechanisms, clinical implications and potential therapeutic interventions.

Sarcopenic obesity represents a dual burden on health, as it combines the detrimental effects of sarcopenia and obesity. Sarcopenia impairs physical function and increases frailty, while obesity contributes to metabolic disorders and chronic diseases. The overlap of these conditions increases health risks, particularly in older adults.

Chronic low-grade inflammation, also known as "inflammaging," is a sign of aging and obesity. This state of persistent, systemic inflammation is characterized by elevated levels of pro-inflammatory cytokines, which negatively impact muscle and fat tissues. Understanding the role of chronic inflammation in sarcopenic obesity is for developing targeted strategies to utilizing its adverse effects. Obesity is associated with an increased accumulation of adipose tissue, particularly visceral fat, which is metabolically active and produces pro-inflammatory cytokines such as Tumor Necrosis Factor-alpha (TNF), InterLeukin-6 (IL-6) and Monocyte Chemoattractant Protein-1 (MCP-1). These cytokines promote systemic inflammation and recruit immune cells, such as macrophages, to adipose tissue.

Adipocyte hypertrophy leads to hypoxia, triggering the production of inflammatory mediators. Necrotic adipocytes attract macrophages, forming crown-like structures that further amplify inflammation. Obesity-induced insulin resistance increases inflammatory signaling, perpetuating a cycle of inflammation and metabolic dysfunction. Chronic inflammation adversely affects muscle homeostasis, contributing to sarcopenia in the context of obesity. Pro-inflammatory cytokines, such as TNF and IL-6, inhibit the Akt/mTOR pathway, reducing muscle protein synthesis.

Inflammation activates the ubiquitin-proteasome and autophagy-lysosome pathways, accelerating muscle protein degradation. Chronic inflammation induces mitochondrial dysfunction in muscle cells, impairing energy production and promoting oxidative stress. This further exacerbates muscle atrophy and weakness. Inflammatory cytokines disrupt the function of satellite cells, the precursors of muscle regeneration, reducing the capacity for muscle repair and growth.

The interconnection between adipose and muscle tissues is mediated by adipokines and myokines, which are signaling molecules produced by fat and muscle, respectively. Leptin and resistin promote inflammation and insulin resistance, negatively affecting muscle metabolism. Adiponectin, an anti-inflammatory adipokine, is often reduced in obesity, diminishing its protective effects on muscle health. Myokines such as InterLeukin-15 (IL-15) and irisin have anti-inflammatory properties and promote fat oxidation.

Chronic inflammation disrupts the balance of myokines, impairing their protective roles in muscle and adipose tissue. Chronic inflammation in sarcopenic obesity exacerbates insulin resistance, dyslipidemia and hypertension, increasing the risk of type 2 diabetes and cardiovascular diseases. The combined effects of muscle loss and fat accumulation, driven by inflammation, lead to reduced strength, mobility and quality of life, particularly in older adults. Inflammation accelerates biological aging processes, contributing to frailty and increased susceptibility to age-related diseases.

Conclusion

Chronic inflammation plays a major role in the pathogenesis of sarcopenic obesity, linking muscle loss and fat accumulation through complex molecular and systemic interactions. Addressing chronic inflammation through dietary, physical, pharmacological and lifestyle interventions offers a promising avenue for reducing the health risks associated with sarcopenic obesity. As research advances, a deeper understanding of these mechanisms will prepare for personalized and effective therapeutic strategies, ultimately improving outcomes for individuals affected by this multifaceted condition.

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