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The Complex Relationship Between Obstructive Sleep Apnoea and Metabolic Syndrome

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Introduction

Obstructive Sleep Apnoea (OSA) and Metabolic Syndrome (MetS) are two prevalent health conditions that have garnered significant attention due to their adverse effects on individual health and well-being. OSA is characterized by repetitive episodes of complete or partial upper airway obstruction during sleep, leading to intermittent hypoxia, sleep fragmentation and subsequent daytime sleepiness. On the other hand, MetS encompasses a cluster of interconnected metabolic abnormalities, including central obesity, dyslipidaemia, hypertension and insulin resistance, predisposing individuals to cardiovascular diseases and type-2 diabetes [1].

OSA disrupts the balance of various hormones involved in metabolic homeostasis, such as leptin, ghrelin, adiponectin and cortisol, contributing to appetite dysregulation, insulin resistance and dyslipidaemia. Similarly, MetS-related hormonal imbalances, including elevated leptin and reduced adiponectin levels, exacerbate OSA severity and promote a pro-inflammatory and pro-thrombotic state.

While these conditions may seem distinct, emerging evidence suggests intricate bidirectional relationships between OSA and MetS, shaping a complex metabolic crossroads. This article aims to delve into the multifaceted interactions between OSA and MetS, exploring their shared pathophysiological mechanisms, clinical implications and therapeutic considerations. Both OSA and MetS are associated with heightened sympathetic nervous system activity and chronic low-grade inflammation. Intermittent hypoxia and sleep fragmentation in OSA trigger sympathetic overactivation and release of pro-inflammatory cytokines, contributing to insulin resistance, endothelial dysfunction and systemic inflammation. Similarly, adipose tissue dysfunction in MetS releases adipokines and inflammatory mediators, exacerbating oxidative stress and sympathetic tone, further exacerbating OSA severity. Intermittent hypoxia-reoxygenation cycles in OSA induce oxidative stress, mitochondrial dysfunction and endothelial dysfunction, promoting insulin resistance and dyslipidaemia. Moreover, chronic hypoxia stimulates adipose tissue hypoxia-inducible factor-1α (HIF-1α) expression, leading to adipose tissue inflammation and dysfunction, perpetuating metabolic dysregulation [2,3].

Description

The coexistence of OSA and MetS synergistically increases the risk of cardiovascular diseases, including hypertension, coronary artery disease and stroke, owing to shared pathophysiological mechanisms such as endothelial dysfunction, oxidative stress and systemic inflammation. Individuals with both conditions exhibit a higher prevalence of adverse cardiovascular outcomes and mortality compared to those with either condition alone. OSA exacerbates metabolic dysregulation in MetS by impairing glucose metabolism, lipid profile and blood pressure control. Conversely, MetS-related obesity exacerbates upper airway collapsibility and worsens OSA severity, forming a vicious cycle of metabolic and sleep disturbances. Moreover, untreated OSA in MetS patients attenuates the efficacy of lifestyle interventions and pharmacotherapy targeting metabolic abnormalities [4].

OSA and MetS independently contribute to neurocognitive impairment, including impaired attention, memory and executive function, due to chronic sleep fragmentation, hypoxia-induced neuronal damage and neuroinflammation. The co-occurrence of OSA and MetS exacerbates cognitive dysfunction, heightening the risk of dementia and cognitive decline in affected individuals. Lifestyle interventions targeting weight loss, dietary modification, regular exercise and alcohol cessation remain cornerstone strategies for managing both OSA and MetS. Weight reduction improves OSA severity, metabolic parameters and cardiovascular risk factors, highlighting the importance of multidisciplinary approaches in managing these interconnected conditions [5].

Continuous Positive Airway Pressure (CPAP) therapy is the primary treatment for moderate to severe OSA, improving nocturnal oxygenation, sleep architecture and daytime symptoms. CPAP therapy also exerts beneficial effects on metabolic parameters, including insulin sensitivity, lipid profile and blood pressure, particularly in individuals with concurrent MetS. Pharmacological agents targeting metabolic abnormalities, such as statins, antihypertensives and insulin sensitizers, may complement CPAP therapy in managing OSA-MetS overlap syndrome. Novel therapeutic agents modulating inflammatory pathways, oxidative stress and adipose tissue function hold promise in mitigating the bidirectional impact of OSA and MetS on metabolic health.

Conclusion

OSA and MetS represent intersecting axes of metabolic dysregulation, sharing common pathophysiological mechanisms and clinical consequences. Understanding the intricate interactions between OSA and MetS is paramount in guiding comprehensive management strategies aimed at improving cardiovascular outcomes, metabolic health and quality of life in affected individuals. Multidisciplinary approaches integrating lifestyle modifications, CPAP therapy and pharmacological interventions offer promising avenues for addressing the complex metabolic crossroads at the nexus of OSA and MetS. Further research elucidating the underlying molecular pathways and longitudinal studies assessing the impact of integrated therapeutic approaches are warranted to optimize the management of this clinically significant overlap syndrome.

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Conflict of Interest

None.

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