

The Role of Inflammation and Oxidative Stress in Sleep Apnea Pathophysiology

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DESCRIPTION

Sleep apnea has emerged as one of the most significant yet underappreciated sleep-related disorders of modern times, with far-reaching implications that extend well beyond disturbed sleep. Characterized by repeated interruptions in breathing during sleep, this condition disrupts the very essence of restorative rest, fragmenting sleep cycles and depriving the body of oxygen. Though it often manifests as loud snoring, restless nights, and daytime fatigue, sleep apnea is not simply a matter of discomfort; it is a complex disorder with profound physiological, psychological, and societal consequences. As awareness of sleep health grows, understanding sleep apnea as a serious medical condition rather than a benign nighttime nuisance becomes imperative.

The impact of sleep apnea extends far beyond poor sleep quality. Oxygen desaturation and fragmented sleep trigger systemic consequences that place enormous strain on multiple organs. Cardiovascular complications are perhaps the most widely recognized. Each apnea episode activates the sympathetic nervous system, leading to spikes in blood pressure and heart rate. Over time, this constant strain contributes to chronic hypertension, arrhythmias, stroke, and heart failure. The repeated surges of stress hormones such as adrenaline and cortisol perpetuate vascular inflammation, further escalating cardiovascular risk. Numerous studies have demonstrated that untreated sleep apnea significantly increases the risk of myocardial infarction and sudden cardiac death, making it one of the strongest modifiable risk factors in cardiology today.

Metabolic health is also profoundly influenced by sleep apnea. Intermittent hypoxia alters glucose metabolism, impairs insulin sensitivity, and contributes to obesity a condition that both results from and worsens apnea. This bidirectional relationship creates a vicious cycle, where obesity narrows airway structures

and increases resistance to airflow, while untreated apnea exacerbates weight gain and metabolic dysfunction. The association between sleep apnea and type 2 diabetes has been well established, raising concerns about the broader role of disordered sleep in fueling the global epidemic of metabolic disease. Furthermore, sleep apnea has been implicated in non-alcoholic fatty liver disease, linking disrupted sleep to yet another domain of systemic illness.

Cognitive and psychological consequences cannot be ignored. Sleep apnea deprives the brain of oxygen and restorative rest, leading to daytime sleepiness, impaired concentration, memory deficits, and reduced alertness. These impairments have real-world consequences, contributing to workplace accidents, poor academic performance, and most notably, motor vehicle crashes. Excessive daytime sleepiness caused by untreated apnea has been compared to the impairments seen in alcohol intoxication, underscoring the public safety hazards associated with the disorder. Beyond cognitive dysfunction, sleep apnea has also been strongly associated with depression, anxiety, and reduced quality of life. Patients often report irritability, social withdrawal, and diminished motivation, all of which compound the challenges of living with a chronic, underdiagnosed condition.

Technology is playing a growing role in reshaping the landscape of diagnosis and treatment. Home sleep apnea testing devices have expanded access to evaluation, reducing the reliance on overnight laboratory-based polysomnography. Wearable devices and smartphone applications now offer continuous monitoring of sleep patterns, oxygen levels, and snoring, providing both patients and clinicians with valuable data. While these technologies hold promise, they also raise questions about accuracy, data privacy, and the risk of self-diagnosis without proper medical oversight. Still, they represent an important step toward democratizing access to care in a field where underdiagnosis remains a major barrier.

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