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LETTERS TO THE EDITOR

Why Should We Care About Selenium in Obstructive Sleep Apnea?

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This year marks the 200th anniversary of the discovery of selenium by the Swedish chemist Jöns Jacob Berzelius. However, this micronutrient has only recently gained the attention it deserves. The human genome presents 25 encoding genes for selenium-containing proteins, called selenoproteins. Selenoenzymes belong to this group and require one selenium atom at their active site, usually obtained from the amino acid selenocysteine, to perform catalytic activities. Selenoenzymes have two main functions: to protect cell components against oxidation and to inhibit proinflammatory cell metabolism.[1] Glutathione-peroxidase (GPx), thioredoxin reductase (TrxR), and iodothyronine deiodinase are examples of selenoenzymes, and in situations of selenium deficiency, their activities are drastically reduced. Vegetables are the main dietary source of selenium; its concentration is determined by soil characteristics and bioavailability.[2] Insufficient intake of selenium has been associated with diabetes, inflammatory disorders, and cardiovascular outcomes. In the heart, GPx enzymes regulate redox balance of cardiac tissue, playing a critical role in the prevention of ischemia/reperfusion and inhibiting the oxidation of plasma low-density lipoprotein, thereby preventing vascular inflammation and atherogenesis.[3] TrxR prevents oxidative stress and regulates myocardial remodeling by reducing cardiac hypertrophy, and iodothyronine deiodinase is fundamental to contractile function.[3]

Cardiovascular comorbidities are prevalent in individuals with obstructive sleep apnea (OSA). The most accepted hypothesis is that intermittent hypoxia leads to oxidative stress contributing to proatherogenic effects: lipid peroxidation, endothelial dysfunction, and systemic inflammation. The literature is scarce regarding studies investigating the role of selenoenzymes/selenium in OSA pathophysiology. It has been demonstrated that serum GPx concentrations are lower in individuals with OSA, and are inversely correlated with mean oxygen saturation and directly correlated with apnea-hypopnea index (AHI).[4] This could characterize a compensatory mechanism, in which there is an overproduction of GPx to minimize the oxidative stress generated by hypoxemia. Chen and colleagues[5] showed that patients with moderate OSA had lower activity of antioxidant enzymes, including erythrocyte GPx, and lower concentrations of erythrocyte selenium when compared to controls. In this study, AHI presented a significantly inverse correlation with erythrocyte selenium so that when the AHI was higher, the concentration of selenium was lower. Thioredoxin (Trx), a small class of proteins that constitute the thioredoxin redox system together with TrxR, has also been investigated. Serum Trx was increased in individuals with OSA and in individuals with OSA and hypertension, and higher OSA severity was associated with higher Trx concentrations.[6]

The role of selenium in OSA progression and associated cardiovascular comorbidities is not well elucidated and needs to be further investigated. However, its importance is clear, given its role as a fundamental cofactor of antioxidant enzymes. Although the recommend daily intake of selenium is 45 µg/d, which seems a very small amount, one in seven people have an inadequate dietary intake, and it is estimated that selenium deficiency risk will increase in the near future.[2] Considering the antioxidant capacity of this micronutrient, ensuring its adequate dietary intake could have a beneficial effect on the response to oxidative stress observed in patients with OSA, reducing the potentially critical cardiovascular and metabolic consequences.

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