Obstructive Sleep Apnea and Cognitive Decline in Older Adults

To the Editor:

Gosselin and colleagues conducted a systematic review of the association between obstructive sleep apnea (OSA) and cognitive decline in older adults with special reference to aging, neurodegenerative mechanism in the brain, treatment effect, and future perspective (1). Although the content of this summary report is valid, I would like to add serum insulin and insulin activity in the brain, which seems indispensable for understanding the biological mechanism of cognitive decline in older adults.

Kullmann and colleagues investigated the effect of three doses of insulin as nasal sprays on the central and autonomous nervous systems (2). Although high-dose nasal insulin showed spillover into the bloodstream, nasal insulin dose-dependently modulated regional brain activity and normalized the high-frequency component of heart rate variability. Insulin activity in the brain is inversely related to serum insulin levels (3), and metabolic disorders caused by insulin resistance with hyperinsulinemia would be indirectly related to OSA and cognitive decline.

Rodriguez-Flores and colleagues investigated the association between obesity and the breath-holding index, which was measured by transcranial Doppler as an indicator of vasomotor reactivity of the brain (4). Subjects without diabetes mellitus and hypertension were selected, and there was a linear negative association between obesity and the breath-holding index, which was adjusted by insulin resistance. The authors concluded that insulin resistance made no contribution to the association between obesity and vasomotor reactivity of the brain, although obesity was closely associated with insulin resistance. There is a significant relationship between OSA and vasomotor reactivity of the brain, and the interrelationship among OSA, cognitive impairment, and depression should be comprehensively evaluated (5).

Lal and colleagues explored biomarkers of cognitive impairment in female patients with OSA who were 45–60 years of age (6). Pathway analysis showed that serum insulin was a prominent protein for regulating other significant biomarkers. In addition, advanced stages of cognitive impairment, such as Alzheimer’s disease, presented similar findings. Taken together, serum insulin and insulin in the brain might be one of the predictors of cognitive decline.

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In summary, although OSA is a risk factor for IR/T2DM in middle-aged individuals, which in turn could increase the risks of cognitive decline and dementia, there is currently not enough evidence to conclude that IR/T2DM might be one of the mechanisms that explain these associations. That said, OSA and IR/T2DM present similar pathological mechanisms, and thus the concomitant presence of these conditions may lead to an additive insult to the brain. However, given the interaction between OSA and IR, insulin regulation may be an interesting avenue to explore when investigating how OSA increases the risk of dementia and cognitive decline. Because OSA and IR/T2DM are treatable, clarifying these associations has the potential to lead to possible interventions to reduce or even prevent neurodegeneration; however, the current state of the literature lacks the appropriate evidence.

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