



Can Tooth Loss Induce Cognitive Decline? The Chicken or the Egg Problem

Considering the rapidly aging population worldwide, the primary challenge that prosthodontists are expected to face in the near future is the treatment of patients with cognitive decline and other neurologic disorders, for which no amount of clever computer-aided dental technology is going to make any significant difference. Cognitive decline is among the most debilitating ailments—affecting ~48 million subjects worldwide—and encompasses many processes, including knowledge formation, memory, thinking, reasoning, and decision making. The impairment of these processes can negatively impact a subject's daily functions and quality of life. Since no effective medication exists to prevent or cure cognitive decline, clinicians and patients alike have been seeking alternative approaches. Consistent with Marcus Tullius Cicero's belief from 65 BC that "It is exercise alone that supports the spirits, and keeps the mind in vigor,"¹ over the past decade, a vast amount of research has done much to augment the popular belief that physical exercise is a "miracle drug" to prevent and even cure cognitive decline.

Riding on this wave of popular acclaim and mimicking those studies focusing on exercise and cognitive functions, no other topic in oral physiology has arguably generated so much interest as the role of teeth and chewing ability on cognitive functions. These studies suggest that a decreased number of teeth in elderly subjects is an epidemiologic risk factor for developing cognitive decline and even Alzheimer's disease. They also suggest that adequate chewing as physical exercise can not only promote general health, daily active living, good mood, and quality of life, but that repeated chewing in healthy subjects and improved chewing ability following oral rehabilitation can also be an effective and inexpensive miracle treatment to enhance cognitive functions and help prevent, impede, or even reverse cognitive decline. Regrettably, the conclusions of many of these studies are hindered by improper study design, such as lack of long-term follow-up, inadequate control groups, and improperly conducted statistics. Many of these studies also rely on self-reported dental status and chewing difficulty, as well as quick and easy yet outdated and incomprehensive screening tools (eg, the 1975 Mini Mental State Examination²) for diagnosing patients with cognitive decline. Moreover, the interpretation of data is often questionable. Many potential confounding factors are associated with impaired cognitive functions and cognitive decline, such as socioeconomic status, social support, education, health and health-related behaviors (eg, smoking, alcohol, drugs, and even coffee), physical activity, and mental health status. In other words, there are too many potential confounding factors to be able to control for them all.

Nevertheless, an overriding question that is raised from these studies is whether their hypotheses might make some sense. This reminds me of the perfect anecdotal evidence to illustrate the interrelations between brain degeneration and motor function: The sea squirt is a marine animal that in its young life

has a rudimentary nervous system of a cerebral ganglion (ie, its brain). This brain generates muscle contractions that enable the sea squirt to swim around the sea. But in its adult life, once the sea squirt sits and attaches itself to a rock, it never swims again. Subsequently, since it does not need to swim anymore, its brain degenerates. It also makes some sense that chewing and cognitive functions are interrelated based on what we know of the significant roles that natural teeth play in maintaining vital chewing efficiency and how the brain generates and controls both chewing and cognitive functions, their interactions, and the brain capacity to mold its neural circuitry and produce new circuits (ie, neuroplasticity) when these functions are impaired by injury (including tooth loss), disease (eg, stroke), or aging.

While there is no strong evidence to support any causal relations between the number of teeth and cognitive decline in old age, there is good evidence that tooth loss, with or without prosthetic replacement, influences food choices and may be associated with inability to sustain adequate nutrition. Improved chewing is also associated with improved nutrition and social interaction. In addition, the evidence suggests that nutrition is important for general health, since it can help reduce the risk of developing many diseases and protect and even enhance cognitive function in healthy adults, while malnutrition can disrupt cognitive functions. Nevertheless, depending on subjects' age, gender, level of education, type and severity of cognitive decline, and health-related comorbidities (such as depression and stroke), some subjects may benefit more than others from improved nutrition. Thus, it is possible that the cognitive decline observed in edentulous and partially edentulous subjects is more related to malnutrition than to the subjects' dental status or chewing capacity and that improved chewing improves nutrition intake, which in turn may improve cognitive functions.

The primary somatosensory cortex, primary motor cortex, and adjacent cortical masticatory area are the main brain regions involved in the generation and control of chewing. The premotor cortex and the supplementary motor areas are involved in the preparation and planning for ingestion, visuomotor control, and cognitive functions related to the understanding of ingestive and chewing actions. However, numerous other brain regions, including those involved in processing cognitive (eg, prefrontal cortex and association areas of the cerebral cortex) and emotive (eg, amygdala) information and memory (eg, hippocampus), also play a crucial role in chewing. All these brain regions are mutually interconnected through a rich network of neural circuits that can modulate chewing, but chewing can also influence cognitive functions and emotions, as well as memory.

A limited number of studies have revealed that impaired chewing in aging adults is associated with age-related degenerative changes within the peripheral neuromuscular system as well as within brain regions involved in the generation and control of chewing. Cells in our entire body degenerate as we age, and neurons are not an exception. In fact, it has been estimated that from around age 30, neural morphology starts to change. While the number of neurons may not change much, their dendrites and synapses start to degenerate, and as a result there is a gradual degradation of neural circuits. Therefore, it is not surprising that with aging there is a gradual decline in sensory, motor, and cognitive functions. In addition, “old brains,” while less neuroplastic than “young brains,” do retain a capacity for neuroplasticity and can thus also learn new tricks—it will just be more difficult and require more time and motivation to adapt to changes in occlusion. Arguably, in light of the vital role of chewing in sustaining life, normal aging is capable of generating compensatory mechanisms that utilize the neuroplastic capacity of the brain to recruit additional brain regions involved in processing cognitive and memory functions. Thus, chewing in the elderly becomes more reliant on these additional brain regions that provide sensory, motor, and cognitive cues to compensate for the aging-related neural, muscular, and sensory deficits and thereby facilitate chewing. However, it is also possible that the recruitment of additional brain regions reflects an inability to recruit the correct and specific brain regions necessary to efficiently execute chewing (ie, maladaptation). Moreover, these brain regions are themselves susceptible to the detrimental age-related effects manifested as clear declines in cognitive functions and memory and can thus contribute to the aging-related impairment in chewing. In addition, since these brain regions receive inputs from the sensorimotor cortex, which in turn receives inputs

from the oral region (including the teeth and masticatory muscles), any changes in the teeth and chewing can in turn influence cognitive and memory functions.

Consequently, more studies are needed to solve “the chicken and the egg problem”: Is it the chewing function alone that supports cognitive functions, or is it the cognitive performance that allows for (or not) the chewing adaptation to the age-related deteriorations in peripheral tissues, including tooth loss? These studies are crucial since cognitive functions and memory are very complex functions, and rehabilitation of these functions requires understanding and utilization of mechanisms that can drive adaptive neuroplasticity.

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References

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