

Interpreting changes in neural oscillatory patterns in aging populations with cognitive decline.

Lin Wei*

Department of Clinical Neurosciences, University of Milan, Italy.

*Correspondence to: Lin Wei, Department of Clinical Neurosciences, University of Milan, Italy, E-mail: l.wei@tsinghua.edu

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Introduction

Neural oscillations, or rhythmic patterns of brain activity, are fundamental to cognitive processes such as attention, memory, and executive function. As individuals age, these oscillatory patterns often exhibit characteristic alterations, some of which are associated with cognitive decline. Changes in frequency, amplitude, and synchrony of neural rhythms—particularly in the alpha (8–12 Hz), beta (13–30 Hz), and theta (4–8 Hz) bands—can serve as early indicators of deteriorating cognitive health. Studies using electroencephalography (EEG) and magnetoencephalography (MEG) have shown that healthy aging is often accompanied by a slowing of dominant frequencies, notably a shift from alpha to theta dominance. These neurophysiological signatures offer a window into the functional integrity of large-scale brain networks and may help differentiate between normal aging and pathological conditions such as mild cognitive impairment (MCI) and Alzheimer's disease (AD) [1].

One of the most consistently reported findings in aging populations with cognitive decline is a reduction in posterior alpha power and coherence, particularly during resting-state conditions. Alpha oscillations, which are associated with inhibitory control and information gating, play a critical role in

maintaining working memory and attentional focus. Their decline is often interpreted as a breakdown in the coordination of brain networks necessary for efficient cognitive processing. Simultaneously, an increase in frontal theta activity has been observed in both MCI and AD, potentially reflecting compensatory mechanisms as the brain reallocates resources to maintain performance. This frontal theta elevation has also been linked to task-related engagement and cognitive effort, suggesting a shift in neural strategies as cognitive decline progresses. However, these compensations are often insufficient to fully offset the deficits associated with disrupted alpha rhythms, leading to measurable impairments in memory consolidation and executive function [2].

Beta oscillations, particularly in the sensorimotor and frontal regions, have also been implicated in age-related cognitive changes. In older adults with cognitive decline, there is often a marked decrease in beta-band power and synchrony during both rest and cognitive tasks. Beta rhythms are essential for maintaining the current cognitive state, and their attenuation may signify a reduced ability to sustain goal-directed behavior. Moreover, reductions in beta coherence between distant cortical areas suggest a fragmentation of large-scale neural networks, which are crucial for integrating multisensory information and coordinating complex behaviors. This

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desynchronization is particularly evident in tasks that require cognitive flexibility and response inhibition, areas in which aging individuals with early dementia often struggle. Such findings indicate that beta oscillatory dynamics provide crucial insights into the neural underpinnings of executive dysfunction in aging populations [3].

Gamma oscillations, though less frequently studied in aging due to their lower signal-to-noise ratio, also exhibit important changes in populations with cognitive impairment. These high-frequency rhythms (30–100 Hz) are thought to underlie local cortical computations and are critical for processes such as perceptual binding and attentional modulation. In individuals with AD, a significant reduction in task-induced gamma power has been noted, particularly in the temporal and parietal regions. Furthermore, recent experimental therapies involving sensory stimulation at gamma frequencies have shown promise in ameliorating amyloid-beta accumulation and enhancing cognitive performance in animal models, suggesting that gamma-band deficits may not only reflect disease pathology but also serve as targets for intervention. The study of gamma oscillations in aging thus provides not only diagnostic value but also a potential therapeutic avenue [4].

Importantly, these oscillatory changes do not occur in isolation but are part of broader disruptions in brain network dynamics. Cross-frequency coupling (CFC), particularly between theta and gamma bands, is believed to support the nesting of local processing within larger cognitive frameworks, such as episodic memory encoding and retrieval. Aging and cognitive decline are associated with altered CFC patterns, especially a reduction in the strength and specificity of theta-gamma coupling in the hippocampus and prefrontal cortex. Such disruptions may impair the brain's ability to temporally coordinate memory traces and execute working memory tasks. Additionally, graph theoretical analyses of oscillatory connectivity networks have revealed a shift from efficient small-world architectures to more random and less integrated configurations in cognitively

impaired older adults. These alterations compromise the brain's ability to flexibly switch between networks required for adaptive behavior, further underscoring the significance of oscillatory biomarkers in understanding cognitive decline [5].

Conclusion

The interpretation of changes in neural oscillatory patterns offers valuable insight into the mechanisms underlying cognitive decline in aging populations. Alterations in alpha, beta, theta, and gamma rhythms reflect both the deterioration of neural integrity and the brain's attempts to compensate for functional losses. With advances in neuroimaging and computational modeling, these oscillatory markers are increasingly being used to identify at-risk individuals, differentiate stages of cognitive impairment, and evaluate the effectiveness of interventions. Understanding the temporal and spatial dynamics of brain rhythms in aging not only deepens our knowledge of the aging process but also opens pathways for developing targeted, non-invasive therapies aimed at preserving cognitive health.

References

1. Albanese A, Romito L. Deep brain stimulation for Parkinson's disease: where do we stand?. *Front Neurol.* 2011;2:33.
2. Cubo R, Åström M, Medvedev A. Optimization of lead design and electrode configuration in deep brain stimulation. *Int J Adv Life Sci.* 2016;8:76-86.
3. Galati S, Stefani A. Deep brain stimulation of the subthalamic nucleus: All that glitters isn't gold?. *Mov Disord.* 2015;30(5):632-7.
4. Skuban T, Hardenacke K, Woopen C, et al. Informed consent in deep brain stimulation—ethical considerations in a stress field of pride and prejudice. *Front Integr Neurosci.* 2011;5:7.
5. Spooner RK, Bohners BH, Schnitzler A, et al. DBS-evoked cortical responses index optimal contact orientations and motor outcomes in Parkinson's disease. *Parkinson's Disease.* 2023;9(1):37.

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