Neural oscillation

Neural oscillations, or brainwaves, are rhythmic or repetitive patterns of neural activity in the central nervous system. Neural tissue can generate oscillatory activity in many ways, driven either by mechanisms within individual neurons or by interactions between neurons.

High-Frequency Oscillations

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Brain oscillations have gained great attention in neuroscience as functional building blocks of cognitive-sensory processes. Research has shown that oscillations in “alpha,” “beta,” “gamma,” “delta,” and “theta” frequency windows are highly modified in the brain pathology, including in patients with a cognitive impairment like bipolar disorder (BD). The study of changes in brain oscillations can provide fundamental knowledge for exploring neurophysiological biomarkers in cognitive impairment. Lu et al. review findings from the role and molecular basis of abnormal neural oscillation and synchronization in the symptoms of patients with BD. An overview of the results clearly demonstrates that, in cognitive-sensory processes, resting and evoked/event-related electroencephalogram (EEG) spectra in the delta, theta, alpha, beta, and gamma bands are abnormally changed in patients with BD showing psychotic features. Abnormal oscillations have been found to be associated with several neural dysfunctions and abnormalities contributing to BD, including abnormal GABAergic neurotransmission signaling, hippocampal cell discharge, abnormal hippocampal neurogenesis, impaired cadherin, and synaptic contact-based cell adhesion processes, extended lateral ventricles, decreased prefrontal cortical gray matter, and decreased hippocampal volume. Mechanistically, impairment in calcium voltage-gated channel subunit alpha1 I, neurotrophic tyrosine receptor kinase proteins, genes involved in the brain neurogenesis and synaptogenesis like WNT3 and ACTG2, genes involved in the cell adhesion process like CDH12 and DISC1, and gamma-aminobutyric acid (GABA) signaling have been reported as the main molecular contributors to the abnormalities in resting-state low-frequency oscillations in BD patients. Findings also showed the association of impaired synaptic connections and disrupted membrane potential with abnormal beta/gamma oscillatory activity in patients with BD. Of note, the synaptic GABA neurotransmitter has been found to be a fundamental requirement for the occurrence of long-distance synchronous gamma oscillations necessary for coordinating the activity of neural networks between various brain regions [1)].

In individual neurons, oscillations can appear either as oscillations in membrane potential or as rhythmic patterns of action potentials, which then produce oscillatory activation of post-synaptic neurons. At the level of neural ensembles, synchronized activity of large numbers of neurons can give rise to macroscopic oscillations, which can be observed in an electroencephalogram. Oscillatory activity in groups of neurons generally arises from feedback connections between the neurons that result in the synchronization of their firing patterns. The interaction between neurons can give rise to oscillations at a different frequency than the firing frequency of individual neurons. A well-known

example of macroscopic neural oscillations is alpha activity.

Computational networks can be a powerful tool to analyze the consequences of injury. Gabrieli et al. used the Izhikevich spiking neuron model to create networks representative of cortical tissue. After an initial settling period with spike-timing-dependent plasticity (STDP), networks developed rhythmic oscillations similar to those seen in vivo. As neurons were sequentially removed from the network, population activity rate and oscillation dynamics were significantly reduced. In a successive period of network restructuring with STDP, network activity levels returned to baseline for some injury levels and oscillation dynamics significantly improved.

Gabrieli et al. explored the role that specific neurons have in the creation and termination of oscillation dynamics. They determined that oscillations initiate from activation of low firing rate neurons with limited structural inputs. To terminate oscillations, high activity excitatory neurons with strong input connectivity activate downstream inhibitory circuitry. Finally, they confirmed the excitatory neuron population role through targeted neurodegeneration. These results suggest targeted neurodegeneration can play a key role in the oscillation dynamics after injury [2].

1)
Lu Z, Wang H, Gu J, Gao F. Association between abnormal brain oscillations and cognitive performance in patients with bipolar disorder; Molecular mechanisms and clinical evidence. Synapse. 2022 Jul 18. doi: 10.1002/syn.22247. Epub ahead of print. PMID: 35849784.
2)
Gabrieli D, Schumm SN, Vigilante NF, Parvesse B, Meaney DF. Neurodegeneration exposes firing rate dependent effects on oscillation dynamics in computational neural networks. PLoS One. 2020 Sep 23;15(9):e0234749. doi: 10.1371/journal.pone.0234749. PMID: 32966291.

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