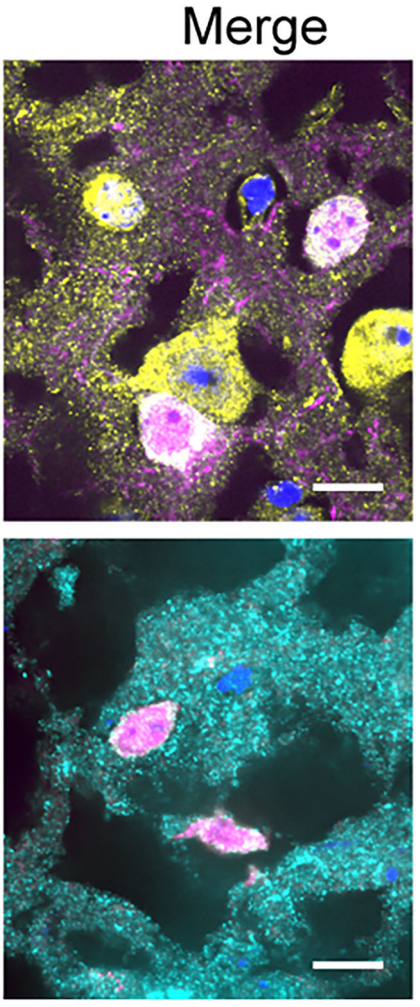
This Week in The Journal

Alpha Wave Direction Differentially Impacts Working Memory

Yifan Zeng, Paul Sauseng, and Andrea Alamia

(see article e0532242024)

Research consistently links alpha waves to working memory, revealing that these waves become more powerful when there is a higher working memory load or when distractors are anticipated. Previous work also suggests that inhibition from alpha waves shields working memory from competing stimuli, but how alpha waves do this remains unclear. To explore this, Zeng et al. assessed the spatiotemporal dynamics of alpha oscillations, which, according to the authors, researchers have not done before. By reanalyzing two open-access EEG datasets of 180 people performing visual working memory tasks, the authors discovered that the direction alpha waves move plays different roles in working memory retention. As distractor load increased, forward-moving alpha power increased. Additionally, as memory set size increased, forward-moving alpha power increased, but backward-moving alpha power decreased. The authors also observed a lateralization effect, whereby the increases in forward-moving alpha power occurred in the brain hemisphere contralateral to the competing stimulus, while the decrease in backward-moving alpha power occurred in the brain hemisphere contralateral to the target stimulus. Altogether, forward-moving waves appear to be regulated by distractor load, while backward-moving waves are inversely modulated by memory targets.



Dopamine 1–expressing MSNs (top), visualized by immunostaining for substance P, and dopamine 2–expressing MSNs (bottom), visualized by immunostaining for A2A receptors. See Serranilla et al. for more information.

New Insights on Neuron Loss in a Huntington's Disease Mouse Model

Melissa Anne Serranilla, Jessica Pressey, and Melanie A. Woodin

(see article e0215242024)

Huntington's disease is characterized by a mutation in the Huntingtin gene that leads to the loss of medium spiny neurons (MSNs) expressing dopamine 1 and 2 receptors in the striatum. Researchers have not yet uncovered the mechanisms that drive degeneration of these MSN subpopulations in Huntington's disease. To explore mechanisms of disease-relevant MSN subpopulation degeneration, Serranilla and colleagues used a mouse model for the disease (R6/2 mice). Because Huntington's disease alters GABAergic signaling to MSNs and functional GABAergic transmission relies on proper chloride regulation, Serranilla et al. investigated whether MSN subpopulations have distinct impairment of chloride homeostasis and how this impacts degeneration of each subpopulation. The authors used electrophysiology, biochemistry methods, and fluorescence imaging to discover that chloride was dysregulated in dopamine 2–expressing MSNs, but not MSNs expressing dopamine 1 receptors. Correcting this dysregulation in dopamine 2–expressing MSNs delayed motor impairment onset in the mice. Serranilla et al. also found that chloride regulation was hindered in the globus pallidus externa, which led to excitatory GABA release. These mechanistic insights may inform treatment development strategies in more advanced animal models.

This Week in The Journal was written by Paige McKeon
https://doi.org/10.1523/JNEUROSCI.twij.44.50.2024