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Presentation Abstract

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Presentation Title: Optogenetic perturbation of motor preparation in primate dorsal premotor cortex

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Abstract: Unlike reflexes, voluntary movements are thought to be prepared before they are executed. Neuronal activity in several brain areas is modulated during motor preparation and execution, including primary motor (M1) and dorsal premotor (PMd) cortex. Recent experimental evidence supports the idea that motor preparation brings neural activity to a beneficial initial state for producing the desired movement. However, the ability to perturb motor cortical activity during motor preparation while simultaneously observing cortical spiking activity would provide causal insights into the neural mechanisms underlying movement preparation. While electrical microstimulation delivered to dorsal premotor cortex (PMd) has been previously shown to disrupt this preparatory process (Churchland and Shenoy, 2007), the observation of neural activity during and immediately after the perturbation is difficult because of electrical interference at the electrode recording surface. Optogenetic techniques recently developed in primates facilitate temporally precise perturbations of genetically-defined neural populations, while allowing for simultaneous electrical recording of spiking activity. Here, two rhesus macaque monkeys (Q,O), injected with AAV5-CaMKIIα-C1V1(T/T)-ts-eYFP in the upper arm region of PMd, were trained on an instructed-delay center-out reaching task. Reach target presentation was followed by a variable delay period before movement initiation was permitted. In both monkeys, we found that when optical stimulation (561 nm, 3 mW) was delivered to PMd near the time of the go

cue, reaction times (RTs) were slower (~12 ms on average in O, $p < 10^{-14}$) compared to non-stimulated trials, demonstrating that optical stimulation can disrupt movement preparation. However, when optical stimulation was delivered > 150 ms in advance of the go cue, RTs were not significantly affected (~-3.5 ms on average in O, $p = 0.99$), suggesting that PMd can recover from the effects of optogenetic perturbation on a rapid timescale. We then sought to assess the effect of optical stimulation on motor cortex in order to gain insight into the dynamics of this disruption and recovery. We utilized multiple electrodes inserted around the optical fiber to record spiking activity and LFP, which allowed us to sample the network response to optogenetic perturbation delivered at various times relative to motor preparation and execution. These experiments highlight the potential for targeted optogenetic perturbation to elucidate causal links between neural dynamics and behavior in primate models.

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MOTOR CORTEX

MULTIELECTRODE

Support: DARPA REPAIR

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NIH

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