

[Print this Page](#)

Presentation Abstract

Program#/Poster#: 750.13/YY3

Presentation Title: Dynamics of primate premotor cortical recovery following optogenetic disruption of motor preparation

Location: Halls B-H

Presentation time: Wednesday, Nov 13, 2013, 8:00 AM - 9:00 AM

Authors: ***D. J. O'SHEA**¹, W. GOO², P. KALANITHI⁴, I. DIESTER⁵, I. OZDEN⁶, J. WANG⁷, A. V. NURMIKKO⁶, K. DEISSEROTH², K. V. SHENOY³; ²Bioengineering, ³Electrical Engin., ¹Stanford Univ., Stanford, CA; ⁴Neurosurg., Stanford Hosp. and Clinics, Stanford, CA; ⁵ESI, Max Planck, Frankfurt, Germany; ⁶Engin., ⁷Physics, Brown Univ., Providence, RI

Abstract: The ability to perturb neural activity and measure the behavioral consequences is critical to understanding how this activity contributes to controlling behavior. Optogenetics enables targeted perturbation and simultaneous recording of neural populations in behaving primates. Here, we explore population responses in primate dorsal premotor (PMd) and primary motor (M1) cortex following optogenetic disruption of motor preparation during a delayed-reaching task. In this task, reaction times (RTs) are faster when subjects prepare before moving. We reported previously (O'Shea, Goo et al., 2011) that optogenetic stimulation (AAV5-CaMKII-C1V1T/T, 3 mW, 561 nm) in PMd disrupts preparatory activity and can partially erase the RT benefit achieved via planning, similar to electrical microstimulation (Churchland & Shenoy, 2007). However, the neural mechanisms which enable recovery from perturbation and orchestrate movement initiation remain opaque. We asked whether motor cortex "replans"--does the perturbed neural population return to the pre-stimulation plan state before movement is initiated? At each stimulation site, we formed trial-averaged neural trajectories across the population of recorded single units and determined when stimulated trajectories were significantly distinct from equivalent non-stimulated trajectories. At 42/44 sites, stimulated trajectories returned to the plan state before movement

initiation when stimulation ends 80 ms before the go cue, which fails to affect RTs. When stimulation occurs nearer the go cue, stimulated trajectories at 29/44 sites did not return to the plan state, but instead proceeded along a distinct path until after movement initiation, which may underlie the observed RT penalty. We then asked how light-evoked activity interacts with task-related activity in motor cortical neurons. We modeled the response of each neuron in each reach direction during the delay period with optical stimulation (optical+task) as a weighted combination of the neuron's delay period activity without stimulation (task) and the light-evoked response during passive rest (optical). This model predicted the average optical+task responses well ($r^2 > 0.97$ in monkeys O and Q). Combined responses were primarily driven by the optically-evoked activity (coeff=0.98, 95% C.I.=[0.97-0.99] in O; 0.99 [0.97-1.01] in Q), but stimulation did not suppress task-related activity (coeff=0.68 [0.65-0.70] in O; 0.67 [0.60-0.74] in Q), in contrast to prior findings by Jazayeri et al. in macaque V1. These results demonstrate the utility of optogenetic perturbation to probe the precise role of neural activity in controlling behavior.

Disclosures: **D.J. O'Shea:** None. **W. Goo:** None. **P. Kalanithi:** None. **I. Diester:** None. **I. Ozden:** None. **J. Wang:** None. **A.V. Nurmikko:** None. **K. Deisseroth:** None. **K.V. Shenoy:** None.

Keyword(s): MOTOR CORTEX
MOTOR CONTROL
PREMOTOR

Support: DARPA REPAIR
HHMI
NIH
NIH Pioneer
CIRM