## Interhemispheric serial dependence in working memory in prefrontal cortex

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Working memory (WM) content is mostly stored in neurons preferring contralateral items in prefrontal cortex (PFC) [1], but representations can travel between hemispheres [2] creating full-field spatial WM continuity. Recently, temporal continuity in WM has been linked to reactivations of activity-silent memory traces in PFC supporting behavioral serial dependence (SD) [3], an attraction of the current report towards the previous stimulus. However, it is unclear how active and activity-silent WM interact with the anatomical lateralization to ensure both WM spatial and temporal continuity.

To answer this question, we test the lateralization of SD using human and monkey behavioral responses with and without TMS in a spatial WM task. Further, we analyze simultaneous bilateral PFC multiunit recordings in three monkeys to assess interhemispheric transfer of reactivations. We find that SD is equally strong when two consecutive stimuli are presented in the same or opposite hemifields. Additionally, we find equally strong neural reactivations coding for the previous trial's stimulus in both hemispheres. Both of these findings indicate spatial continuity of SD. Conversely, we find causal evidence for localized reactivations that cannot travel between hemispheres when inducing reactivations through TMS in humans. We find that increasing reactivations unilaterally, exclusively leads to an increase in SD when the consecutive target is stored in the TMS-affected hemisphere.

To understand how these seemingly inconsistent findings could be reconciled, we perform single-trial decoder correlations of the neural activity between hemispheres. We find stronger single-trial correlations during the delay than during reactivations, indicating a coordination of trial-by-trial memory representations across hemispheres during active memory maintenance but not during reactivations. This suggests that reactivations occur in both hemispheres equally but privately, explaining both the spatial continuity of SD in regular WM trials, as well as the lateralized increase of SD during unilateral reactivation enhancement.

- [1] Funahashi, S. *et al.*, *J Neurophysiol* 61:2, 331-349 (1989)
- [2] Brincat, S. L. et al., Neuron 109:6, 1055-1066.e4 (2021)
- [3] Barbosa, J., Stein, H., Martinez, R.L. et al., Nat Neurosci 23, 1016–1024 (2020)