An Excitatory Neural Assembly Encodes Short-Term Memory in the Prefrontal Cortex

Highlights
- Two-photon imaging enables the monitoring of neural activities on behaving animals
- A neuronal subpopulation in the mPFC that exhibited emergent properties encodes STM
- These neuronal subpopulations exclusively comprise excitatory neurons
- This STM coding mechanism is absent in an animal model of AD

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In Brief
Using two-photon imaging on behaving mice, Tian et al. discover a functional coding mechanism that relies on the emergent behavior of a functionally defined neuronal assembly to encode short-term memory. They further demonstrate that this coding mechanism is absent in an animal model of Alzheimer’s disease (AD).
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SUMMARY

Short-term memory (STM) is crucial for animals to hold information for a small period of time. Persistent or recurrent neural activity, together with neural oscillations, is known to encode the STM at the cellular level. However, the coding mechanisms at the microcircuitry level remain a mystery. Here, we performed two-photon imaging on behaving mice to monitor the activity of neuronal microcircuitry. We discovered a neuronal subpopulation in the medial prefrontal cortex (mPFC) that exhibited emergent properties in a context-dependent manner underlying a STM-like behavior paradigm. These neuronal subpopulations exclusively comprise excitatory neurons and mainly represent a group of neurons with stronger functional connections. Microcircuitry plasticity was maintained for minutes and was absent in an animal model of Alzheimer’s disease (AD). Thus, these results point to a functional coding mechanism that relies on the emergent behavior of a functionally defined neuronal assembly to encode STM.

INTRODUCTION

Short-term memory (STM) is the capacity to hold information in the brain in an active, readily accessible state for a brief period of time, typically from seconds to minutes (Jonides et al., 2008; Richardson, 2007). Deficits in STM are associated with the pathogenesis of many psychiatric disorders, including Alzheimer’s disease (AD) and posttraumatic stress disorder (PTSD). For example, familial AD patients exhibit deficits in verbal memory about two years before an official diagnosis of AD (Fox et al., 1998), and sporadic AD patients show significant impairment in veridical recall in a semantically associated word list learning task (MacDuffie et al., 2012). People with PTSD show worse non-verbal STM on the Benton visual retention test, possibly because of dysfunction in the hippocampal (Emdad and Søndergaard, 2006).

Unlike long-term memory that depends on protein synthesis (Davis and Squire, 1984; Jarome and Helmstetter, 2014), STM seems to depend on firing patterns at the cellular level (Fuster and Alexander, 1971; Goldman-Rakic, 1995; Miller et al., 1996; Rawley and Constantinidis, 2009; Wang, 2001). The encoding mechanism of STM has been most studied in the context of working memory, which involves both STM and a central executive module. Extensive studies have shown that a persistent
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