第44回 最先端 脳科学セミナ-

Synaptic and cellular representation of the identity of overlapping memory engrams



Memories are formed through long-term changes in synaptic efficacy, a process known as synaptic plasticity, and are stored in the brain in specific neuronal ensembles called engram cells, which are activated during corresponding events. When two memories are associated, cell ensembles corresponding to each memory overlap. However, each memory has its own identity. How the brain stores and defines a specific memory identity when two memories interact and are encoded in the shared ensemble have remained elusive. Here, we show that sy s napse-specific plasticity represents specific memory entities, and that synaptic plasticity between specific engram assemblies is both sufficient and crucial for information storage. Using auditory fear conditioning and c-fos-TetTag system, optogenetic stimulation of the activated ensemble terminals of auditory cortex (AC) and medial geniculate nucleus (MGm) in lateral amygdala (LA) after complete retrograde amnesia -accomplished by autophagy induction with protein synthesis inhibition- failed to induce memory recall at recent and remote time points, indicating that the memory engram no longer existed in that circuit. This result was correlated with the resetting of plasticity and functional connectivity between the engram assemblies. Complete retrograde amnesia of a given fear memory did not affect the linked fear memory encoded in the shared ensemble. Furthermore, potentiation or depotentiation of the plasticity at synapses specific to one memory affected the recall of only that memory without influencing the linked memory. Thus, sharing of engram cells underlies the linkage between memories, while synapse-specific plasticity guarantees the identity and storage of individual memories. Our study gives insight into therapeutic approaches to treating post-traumatic stress disorder (PTSD).

Featured publication:

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