Symposium 7: Dynamic Memory: molecular, cellular and systems approaches (Thu July 29, 16:40-18:40 JST) Chair: Bong-Kiun Kaang (Seoul National Univ, Korea)

16:40-17:04 Yi Zhong (Tsinghua University, China) Emotion-mediated switching between latent and silent memory engram

17:04-17:28 Daisuke Miyamoto (University of Toyama, Japan) In vivo imaging of cortical synaptic dynamics by learning and sleep.

17:28-17:52 Jin-Hee Han (Dept Biological Sciences, KAIST, Korea) A synaptic activity-dependent competition rule underlying memory formation

17:52-18:16 Johannes Gräff (Ecole Polytechnique Fédérale de Lausanne, Switzerland) Recent insights into remote fear memory attenuation

18:16-18:40 Denise Cai (Icahn School of Medicine at Mount Sinai, USA) The brain in motion- how ensemble fluidity supports memory updating.

Symposium 7 Speaker 1: Yi Zhong

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Emotion-mediated switching between latent and silent memory engram

In pathological or artificial conditions, memory can be formed as silenced engram that is unavailable for retrieval through presenting conditioned stimulus, but could be turned artificially into the latent state so that natural recall is allowed. However, it remains unclear whether such different states of engram bear any physiological significance and can be switched through physiological mechanisms. Here we show that an acute social reward experience rescues protein synthesis inhibition-induced amnesic memory via switching the silent memory engram into a latent state. Conversely, an acute social stress causes transient forgetting via turning latent memory engram into silent. Such emotiondriven bidirectional switching between latent and silent engrams is mediated through regulation of Rac1 activity-dependent reversible forgetting in the hippocampus, as stressactivated Rac1 suppresses retrieval while reward recovers amnesic memory by inhibiting Rac1. Thus, data presented reveal an emotion-stimulated molecular mechanism to mediate switching between latent and silenced engrams to achieve emotiondriven bidirectional fexibility.

Symposium 7 Speaker 2: Daisuke Miyamoto Associate Professor University of Toyama Toyama, Japan miyamoto@cts.u-toyama.ac.jp

In vivo imaging of cortical synaptic dynamics by learning and sleep.

Synaptic plasticity is a well-known mechanism of learning and memory. Learning during wakefulness potentiates a subset of synapses in memory-related brain regions. In reverse, sleep depresses synaptic strength on average. Although synaptic depression during sleep may support neural circuitry homeostasis, it is still unclear how learning and sleep regulates synaptic representations of memory. Here, in mice, we did in vivo two-photon imaging of spine-surface GluA1-containing AMPA receptors, as an index of excitatory synaptic strength. In the primary motor cortex, the average amount of spine AMPA receptors was upregulated by motor learning and downregulated by pre- and post-learning sleep, which was prevented by sleep deprivation. In contrast, a subset of spines which experienced maximum upregulations ("max" spines) were not affected by post-learning sleep or sleep deprivation. Motor memory performance positively correlated with downregulation of AMPA receptors during post-learning periods not in the "max" spines but in other spines (Miyamoto et al., Nat Commun, in press). These results suggest that sleep enhances signal-to-noise ratio of synaptic representations of memory through noise reduction.

Symposium 7 Speaker 3: Jin-Hee Han Associate Professor Department of Biological Sciences, KAIST han.jinhee@kaist.ac.kr

A synaptic activity-dependent competition rule underlying memory formation

Memory is supported by a sparse population of neurons distributed in broad brain areas, an engram. Despite recent advances in identifying an engram, how the engram is created during memory formation is poorly understood. Strengthening of synaptic connections between neurons that are active during an event is thought to be involved in formation of engram. However, the relation between synaptic plasticity and construction of engram is unclear. To explore this question, here we targeted a sparse random subset of neurons in the secondary auditory cortex and thalamus and found that synaptic inputs from these neurons to the lateral amygdala (LA) were not potentiated by associative fear learning. Using an optogenetic priming stimulus preceding an associative fear conditioning, we manipulated these synapses to be potentiated by the learning. In this condition, fear memory was preferentially encoded in the manipulated cell ensembles. This change, however, was abolished with optical LTD delivered to the primed synapses shortly after training. Conversely, delivering optical LTP alone shortly after fear conditioning was sufficient to induce the preferential memory encoding. These results suggest a synaptic plasticity-dependent competition rule underlying memory formation.

Symposium 7 Speaker 4 Johannes Gräff

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Recent insights into remote fear memory attenuation.

Fear and traumata generate some of the longest-lived memories, which contributes to the elevated life-time prevalence of stress and anxiety-related disorders. Despite the corresponding need to better understand how such long-lasting fear memories can be attenuated, surprisingly little is known about this process. In this talk, I will present our recent findings that help to better understand remote fear memory attenuation from a circuit, cellular and molecular perspective.

Symposium 7 Speaker 5

Denise Cai Assistant Professor Icahn School of Medicine at Mount Sinai denisecai@gmail.com

The brain in motion- how ensemble fluidity supports memory updating.

The compilation of memories, collected and aggregated across a lifetime, defines our human experience. My lab is interested in dissecting how memories are stably stored while being continuously updated across a lifetime. Our studies suggest that a shared neural ensemble may link distinct memories encoded close in time. Using in vivo calcium imaging (with open-source Miniscopes in freely behaving mice), TetTag transgenic system, chemogenetics, electrophysiology and novel behavioral designs, we tested how hippocampal networks temporally link and update memories. Multiple convergent findings suggest that contextual memories encoded close in time are linked by directing storage into overlapping hippocampal ensembles, such that the recall of one memory can trigger the recall of another temporally-related memory. Memories are continually updated after learning during an offline period, potentially during sleep. Alteration of this process (e.g. during aging, stress, etc) affects the temporal structure of memories, thus impairing efficient recall of related information.