## Meeting Program

Tuesday (9/3)							
15:00	-	18:30	Arrival and Hotel Check-In				
18:30	-	20:30	Welcome Dinner @ Fuji (reception 18:00-)				
Wednesday (9/4)							
Welcome ar	Welcome and Introduction						
9:00	-	9:10	Masanori Murayama, on behalf of meeting organizers Scott Soderling, on behalf of meeting organizers				
Keynote Leo	ture	•					
9:15	-	10:00	Kaoru Inokuchi (Introduction: Masanori Murayama)				
New Molece	ules	in Synaptic pl	asticity (Chair: Elly Nedivi)				
10:00	-	10:30	Takeshi Imai (J)				
10:30	-	11:00	Susumu Tomita (US)				
11:00	-	11:20	Coffee/Tea break				
11:20	-	11:50	Mineko Kengaku (J)				
11:50	-	12:20	Scott Soderling (US)				
Luncheon so	emir	ar (Sponsor:	NIKON INSTECH CO., LTD.)				
12:40	-	13:40	Keisuke Ota (J)				
			Masako Ino (NIKON INSTECH CO., LTD.)				
New concep	ots o	f synaptic pla	sticity in neural circuit function (Chair: Takeshi Imai)				
14:00	-	14:30	Schuichi Koizumi (J)				
14:30	-	15:00	Tetsuya Takano (US)				
15:00	-	15:30	Haruhiko Bito (J)				
15:30	-	15:50	Coffee/Tea break				
15:50	-	16:20	Thomas Blanpied (US)				
16:20	-	16:50	Masanori Murayama (J)				
17:00	-	18:20	Poster Session and Discussion @ Kaede				
18:40	-	20:00	Dinner @ Mugibatake restaurant				
20:10	-	22:00	Poster Session and Discussion (Optional)				

Thursday (9/5)							
8:45	-	8:55	Welcome and solicitation of feedback for next day				
			Masanori Murayama and Scott Soderling				
Keynote Lecture							
9:00	-	9:45	Richard Mooney (Introduction: Scott Soderling)				
New concepts of synaptic plasticity in disease (Chair: Matthew Kennedy)							
9:45	-	10:15	Akiko Hayashi-Takagi (J)				
10:15	-	10:45	Gavin Rumbaugh (US)				
10:45	-	11:05	Coffee/Tea break				
Novel and en	merg	ing imaging m	ethods for synaptic analysis (Chair: Akiko Hayashi-Takagi)				
11:05	-	11:35	Hideji Murakoshi (J)				
11:35	-	12:05	Makoto Higuchi (J)				
12:05	-	12:35	Takuya Takahashi (J)				
Luncheon se	emir	ar (Sponsor: C	Dlympus Corporation)				
12:55	-	13:55	Shinichiro Tsutsumi (J)				
			Kazuhiko Hosono (Olympus Corporation)				
New ideas in	n dyr	namics of synap	otic signaling and structure (Chair: Hideji Murakoshi)				
14:00	-	14:30	Elly Nedivi (US)				
14:30	-	15:00	Shigeo Okabe (J)				
15:00	-	15:30	Lin Tian (US)				
15:30	-	16:00	Yukiko Gotoh (J)				
16:00		16:20	Coffee/Tea break				
New faces in	n syn	aptic neuroscie	nce: Trainee talks (Chair: Masanori Murayama)				
16:20	-	16:35	Kareem Abdou (J)				
16:35	-	16:50	Dalila Ordonez (US)				
16:50	-	17:05	PinWu Liu (J)				
17:05	-	17:20	Austin M Ramsey (US)				
17:20	-	17:35	Daichi Kawaguchi (J)				
17:35	-	17:50	Nerea Llamosas (US)				

18:00	-	18:55	Poster Session and Discussion @ Kaede			
19:00	-	21:00	Dinner @ Fuji (PIs vote for poster awards)			
21:10	-	22:00	Poster Session and Discussion (Optional)			
Friday (S	9/6)					
New ideas in pre and postsynaptic function (Chair: Lin Tian)						
9:00	-	9:30	Yasunori Hayashi (J)			
9:30	-	10:00	Jason Shepherd (US)			
10:00	-	10:30	Takeshi Sakaba (J)			
10:30	-	10:50	Coffee/Tea break			
10:50	-	11:20	Michisuke Yuzaki (J)			
11:20	-	11:50	Matthew Kennedy (US)			
Luncheon seminar (Sponsor: Thorlabs Japan Inc.)						
12:00	-	13:00	Henry Haeberle (Thorlabs, Inc.)			
13:10	-	13:25	Poster Award (3 persons)			
13:25	-		General discussion and organization of collaborations			
			Departure of participants			
			Organizer meeting to draft summary document			

# Abstracts

## Oral Presentation

### Cell ensemble mechanisms underlying memory formation

#### K. Inokuchi

Memories are not stored in isolation from other memories but are integrated into associative networks. At the same time, each memory has its own identity. Because association of related memories, with keeping the identity of each memory, is the fundamentals of knowledge formation, it is important to understand the underlying mechanisms. In this seminar, I will show that sharing memory engram cells underlies the linkage between memories (1), while synapse-specific plasticity guarantees the identity and storage of individual memories (2). In addition, I will suggest that engram cells in the hippocampus are organized into sub-ensembles representing distinct pieces of information, which are then orchestrated to constitute an entire memory (3).

#### References

1. Yokose et al, Overlapping memory trace indispensable for linking, but not recalling, individual memories. *Science*, 355: 398-403 (2017). doi:10.1126/science.aal2690

2. Abdou et al, Synapse-specific representation of the identity of overlapping memory engrams. *Science*, 360: 1227-1231 (2018). doi:10.1126/science.aat3810

3. Ghandour et al, Orchestrated ensemble activities constitute a hippocampal memory engram. *Nature Communications*, 10: 2637 (2019). doi:10.1038/s41467-019-10683-2

# Abstracts

## Poster Presentation

### Synapse-specific plasticity governs the identity of overlapping memory traces

K. Abdou, M. Shehata, K. Choko, H. Nishizono, M. Matsuo, S. Muramatsu, and K. Inokuchi

Memories are encoded and stored in specific neuronal ensemble, called engram cells. Some of these memories are associated and stored in shared ensemble. However, brain machinery that underlies memory storage and defines certain memory identity amidst numerous number of memories stored in the same ensemble is poorly understood.

Here we show that when two associative memories are encoded in shared ensemble, engram-specific synaptic plasticity delineates specific memory entity and that specific plasticity 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 memory engram no longer exists in that circuit. This result was correlated with the resetting of plasticity and functional connectivity between the engram assemblies. Furthermore, potentiating or depotentiating the plasticity at synapses specific to a given memory did not affect the linked memory that is encoded in the same ensemble, suggesting that memories are stored in specific synapses.

These findings unravel how the brain organizes and stores multiple associative memories in shared ensemble, underpinning a causal relationship between synaptic input-specific plasticity and memory identity and storage. Moreover, our study sheds light on the capability of selective and integral erasure of memory trace from the engram network, suggesting a potential way to treat post-traumatic stress disorder (PTSD).

### Manipulation of fear memory association by posterior parietal cortex

<u>A. Suzuki</u>, S. Kosugi, E. Murayama, N. Ohkawa, M. Matsuo, H. Nishizono, and K. Inokuchi

The association of fear memory occurs when a conditioned stimulus (CS) is paired with an unconditioned stimulus (US). Although previous studies suggested that some of brain regions responded to CS and US signals, it is still unclear how the association regulates.

Here we show that the cellular ensemble in Posterior Parietal Cortex (PPC) specifically modulates CS-US association without the processing of CS and US information. In the modified context-preexposure facilitation effect (CPFE) paradigm, optical silencing of PPC neurons which responded to context exposure (CS), when mice received footshock (US) in the same context, failed to associate the context (CS) and the shock (US). On the other hand, optical activation of PPC neurons which responded to context exposure, when mice received footshock in a different context, generated an artificial CS-US associative memory, in which mice showed a freezing response in the initial context where mice did not received footshock, but not in the neutral context. Furthermore, 15 min optically silencing of PPC neurons that responded during reactivation of CS-US associate memory that has been once formed immediately after CS exposure 1 day after reactivation suppressed fear memory when mice were tested 1 day later without optical silencing. Thus, manipulating the PPC activity diassociate CS-US associative memory.