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Molecular basis of GABAergic circuit development and neural allocation mechanisms of recent and remote memories

Normal brain function relies on the precise development of neural circuits, which begins during embryogenesis but undergoes a protracted period of refinement until adulthood. The central goal of my research has been to understand how neural networks are specifically wired in the cerebral cortex during postnatal development.

In the first part of my talk, I will present my main findings on the molecular determinants that govern GABAergic circuit formation and how abnormal integration of GABAergic sub-types – particularly interneurons impact on hippocampal function (1, 2) which guide us towards a better understanding of the pathophysiology underlying neuropsychiatric disorders (<https://authors.elsevier.com/c/1WMuH3Q9h1uv8H>).

In the second part of my talk, I will turn to adult circuits and the mechanisms supporting memory storage within neocortical networks. During the course of system's level consolidation, memories are stored and stabilized gradually within the prefrontal cortex (PFC). Previous findings suggest that an early tagging of immature memory engrams in the PFC is required for permanent storage of long-lasting memories. Although many studies support the role of synaptic plasticity as critical factor during learning for the integration of neurons into active memory engrams, the biological mechanisms responsible for the gradual consolidation of memories in the PFC remain poorly understood. It is hypothesized that intrinsic plasticity mechanisms (changes in neuronal excitability) play a profound role during memory formation. We have combined mouse genetics with behavioral as well as electrophysiological approaches, to address whether intrinsic plasticity is part of the tagging process required for the formation of enduring memories in the PFC.

1. I. Del Pino et al., Erbb4 deletion from fast-spiking interneurons causes schizophrenia-like phenotypes. *Neuron* 79, 1152-1168 (2013)
2. I. Del Pino et al., Abnormal wiring of CCK(+) basket cells disrupts spatial information coding. *Nat Neurosci* 20, 784-792 (2017)