The neuroplasticity – neuropathology continuum: an alternative view on learning and memory formation

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Abstract

Neuroscientists describe learning at the neuronal level as a process of long-lasting enhancement in signal transmission between neurons causing modification of cellular responses specific to external stimulation. It is widely accepted that long-term potentiation – the synaptic strengthening resulting from a synchronous stimulation of neurons (Hebbian theory) – is the mechanism underlying these modifications. On the ontogenetic level, these induced, physical changes of intrinsic brain activity are believed to be the basis for memory formation. Extending this line of arguments, the concept of neuronal memory allocation suggests that distinct brain processes specifically determine which neurons and synapses encode certain memory traces, hinting towards a Kantian view of knowledge encoding.
In this talk, I will argue that comparable neuronal modifications that underlie long-term potentiation are also involved in processes causing long-term depression or pathological and neurodegenerative conditions. Thus, molecular mechanisms ascribed to memory formation seem to represent only a narrow section of a neuroplasticity-neuropathology continuum (McEachern and Shaw, 1999).
I will substantiate these arguments with results from my own work as well as using data from other molecular neuroscientists and electrophysiologists. This alternative view of neuroplasticity aims to trigger a discussion about the congruity between synaptic mechanisms and knowledge encoding/storing and to fuel a debate about neurodiversity and its contribution to our "knowledge society".

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