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Dynamical emergence of fear and extinction cells in the amygdala – a computational model

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During a typical fear conditioning experiment a neutral stimulus is paired with a fearful one and after multiple trials the former acquires aversive properties. This learning can be suppressed by repeated presentations of the initially neutral stimulus alone (fear extinction). The major brain structure involved in these fear related processes is the amygdaloid complex, but the exact functional mechanisms are not well understood. A recent article reported that two distinct fear and extinction cell populations within the basal nucleus of the amygdala (BA) are exclusively activated during fear conditioning and extinction respectively [1].

The aim of this project is to explain how these two neural subpopulations arise and what their causal role is in the acquisition and suppression of fear memories. For this purpose we built a spiking neuron network model using the NEST simulator [2]. We modeled the BA based on known anatomical data as a recurrent network consisting of excitatory and inhibitory neurons. The excitatory neurons received sensory input from the adjacent lateral nucleus (LA) and contextual input from the hippocampus, and in turn excited the surrounding inhibitory neurons. We propose short-term plasticity at the sensory afferents of the excitatory neurons as one of the main mechanism underlying the formation of the experimentally observed neuron subgroups within the BA. This type of plasticity is governed by contextual inputs and regulated by neuromodulators.

Endowed with this plasticity mechanism the recurrent network model is able to replicate three key experimental findings: (i) Emergence of fear cells during fear conditioning in one context; (ii) Emergence of extinction cells during extinction training in a different context; (iii) Post-extinction activation of the same fear cells in the original conditioning context. These results are evidently based on the assumption that fear extinction is a new learning rather than unlearning, that is fear and extinction memories coexist and compete with each other. The experimental findings reported a distinct functional connectivity of fear and extinction cells to hippocampus and pre-frontal cortex, which suggests a rather rigid organization of those cells. In contrast, our model shows that fear and extinction cells can emerge dynamically as a result of the identical learning mechanism being applied to the population as a whole.

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