PLASTICITY IN LATERAL AMYGDALA AFTER PAVLOVIAN FEAR CONDITIONING – A Computational Study

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ABSTRACT

One of the main contributions of the dissertation is an explanation of how and why certain neurons are recruited into a memory trace. For this, we developed a biophysical model of the rodent lateral amygdala (LA) and then examined how particular LA neurons are assigned to the fear memory trace. The model revealed that neurons with high intrinsic excitability are more likely to be integrated into the memory trace but that competitive synaptic interactions also play a critical role. We also examined the relative contributions of plasticity in auditory afferent (thalamic, cortical) neurons vs. within LA. This revealed that plasticity in afferent pathways to LA is required for fear memory formation, but that once formed, the plasticity in afferent pathways was not needed. The model then provided insights into how 'competition' was implemented at the single cell level, including the role of excitatory connections among neurons, of disynaptic inhibition, and of neuromodulation. These principles should also apply to other forms of memory in brains. We then investigated another related concept of specificity of memory. Analysis showed that formation of memory involves plasticity in the connections within LAd and this plasticity also ensures specificity for that memory.

We report a procedure to develop a reduced order model matching passive properties, current injection traces, and preserving some synaptic integration features.