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# Role of cGMP-dependent protein kinases for fear memory formation in the lateral amygdala

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Fear memory formation critically relies on the function of the lateral amygdala (LA) and changes in synaptic transmission of its sensory inputs. There is evidence that signaling through NO/cGMP in the amygdala contributes to these processes [1-3]. The cGMP-dependent protein kinases (cGK) type I and II serve as universal effectors of the NO/cGMP signaling cascade. Recently, we established a functional role of cGKI for synaptic plasticity and fear memory consolidation in the amygdala. First, we demonstrated that the cGKIβ isoform is highly expressed in the LA. Furthermore, we showed that cGKI-deficient mice revealed deficits in amygdala-dependent fear memory consolidation. In contrast, short-term memory and hippocampus-dependent memory was intact in these mice. In line with the behavioral phenotype of impaired auditory long-term memory, we found reduced long-term potentiation (LTP) in the LA of cGKI-deficient mice, which is considered as a cellular mechanism for learning induced synaptic plasticity. Unlike cGKI-deficient mice, cGKII-knockout mice lack these phenotypes. These results clearly suggest a crucial role of cGKI for consolidation of amygdala-dependent fear memory, a process by which newly learned information is stabilized into long-term memory (LTM). It is a well known fact that memory consolidation strictly depends on transcriptional and translational processes which is also proved essential for amygdala-dependent fear memory consolidation [4,5]. A functional link between cGKI activity and gene transcription could already be demonstrated *in vitro* [6]. The upcoming question is if cGKI-induced transcription also takes place in amygdala neurons recruited during memory formation. Preliminary data indicate a role for cGKI in cAMP-response element binding protein (CREB) signaling in the LA, a prominent transcription factor involved in learning and memory.

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