

Poster presentation

cGMP-dependent kinase I supports formation of associative fear memory and long-term potentiation in the lateral amygdala

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Nitric oxide (NO) and cGMP have been reported to modulate memory consolidation of Pavlovian fear conditioning and long-term potentiation (LTP) of synaptic transmission in the amygdala. The cGMP-dependent protein kinases, cGKI and cGKII, representing universal effectors in the NO/cGMP signalling cascade are both expressed in the amygdala and their function for amygdala-dependent fear conditioning and synaptic plasticity remains to be elucidated. Associative fear learning is thought to rely on synaptic plasticity in various inputs to the lateral amygdala. We initially examined LTP in the amygdala of wild type and cGKI^{-/-} mice using extracellular recordings. Mutant mice showed reduced LTP in the cortical input to the lateral amygdala (see Figure 1). Behavioural analysis in cGKI null mutants (cGKI^{-/-}) is obstructed by their premature death resulting from severe smooth muscle dysfunction. Differently, cGK-deficient mice live to adulthood when the expression of cGKI is rescued selectively in smooth muscle cells. We used this mouse model (cGKI rescue) to examine whether the impairment of synaptic plasticity due to the lack of cGKI is paralleled by a defect of amygdala-dependent learning. Therefore, we studied amygdala-dependent cued fear and contextual fear behaviour in wild type and rescue mice. Freezing to a tone previously conditioned with a foot shock was significantly reduced in cGKI rescue mice compared with control suggesting an impairment of the tone-shock association (see Figure 2). Freezing in the condi-

tioning environment was not different in control and cGKI rescue mice indicating that contextual fear memory was normal.

Conclusion

Inferior performance in fear conditioning of cGKI-deficient mice demonstrate a critical role of the cGKI isoform

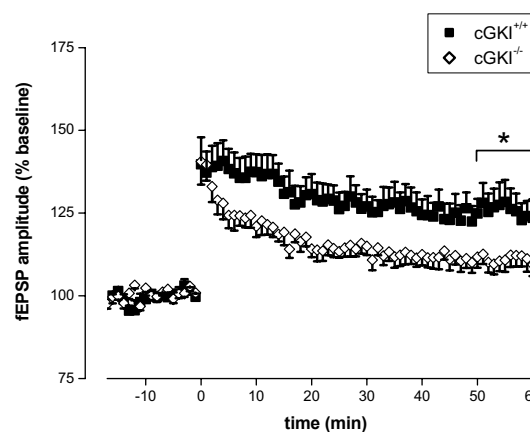


Figure 1
cGKI-deficient mice show reduced long-term potentiation in the cortical input of the lateral amygdala.

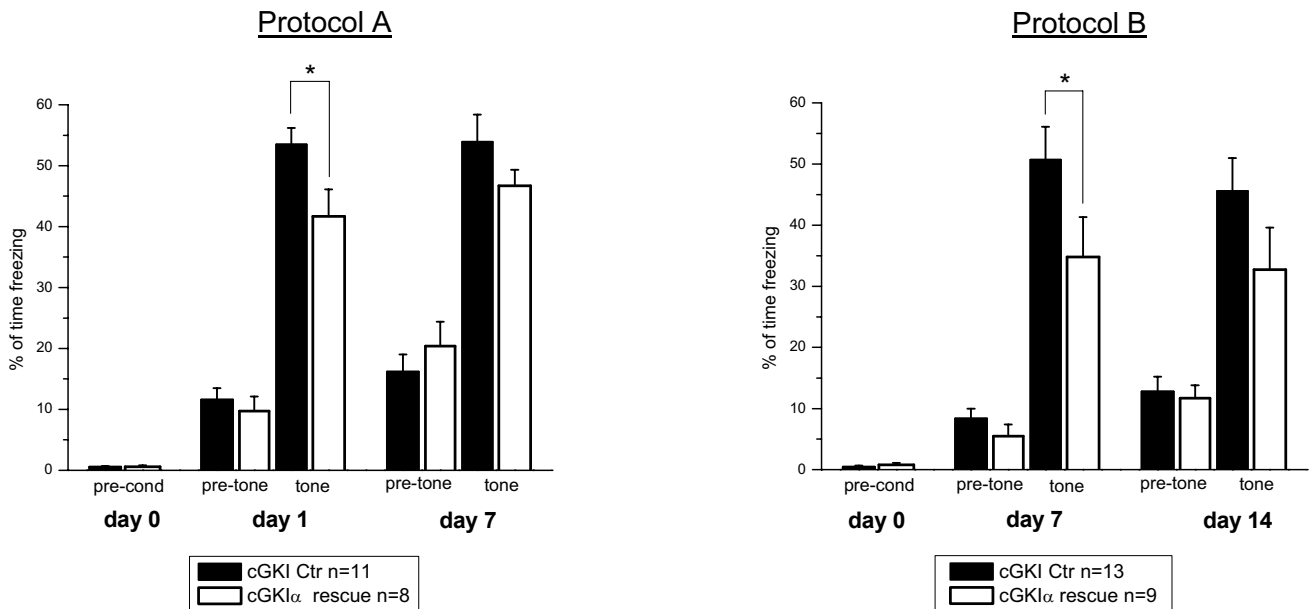


Figure 2

Freezing to the conditioned tone is reduced in cGKI-deficient mice compared to control on day 1 with protocol A (day 0 → day 1 → day 7) and also on day 7 with protocol B (day 0 → day 7 → day 14).

for associative learning likely related to mechanisms supporting memory consolidation. Moreover, reduced LTP of cGKI^{-/-} mice show that synaptic plasticity in the lateral amygdala is supported by cGKI.

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