

Evaluation of *kalrn* transcript levels in zebrafish seizure model

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Abstract

Temporal transcript profile of *kalrn* gene in zebrafish larvae brain after pentylentetrazole (PTZ)-induced seizure.

Key words: zebrafish, *kalrn*, seizure.

Introduction

Kalirin (*kalrn*) regulates neuronal shape and spine growth playing an important role in plasticity [1]. Besides, it has been implicated in some neurological diseases [2]. Today, there is no study associating the *kalrn* gene and seizures. We have been using the zebrafish seizure model for genetic investigations because its advantages in genetics studies. The main aim of this study was to investigate the *kalrn* mRNA levels in the zebrafish pentylentetrazole (PTZ)-seizure model.

Results and Discussion

Our results showed no statistical difference in the *kalrn* mRNA levels between seizure group (SG) and control group (CG) after 24 and 48 hours of PTZ-induced seizure (images 1 and 2). The mean \pm SEM of *karln* mRNA comparing SG and CG groups were the following: CG24h 0.85 ± 0.14 ; SG24h 0.92 ± 0.1 ($p=0.5$) and CG48h 1.00 ± 0.03 ; SG48h 0.93 ± 0.03 ($p=0.1$).

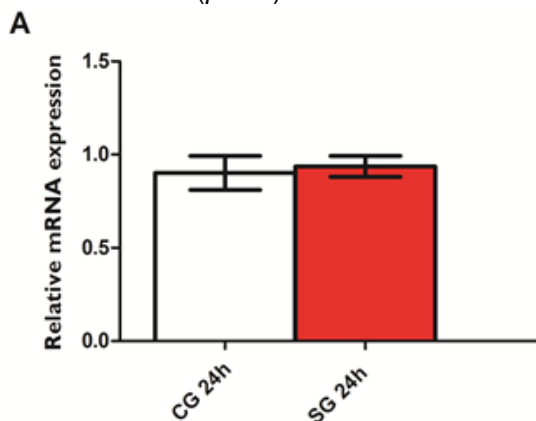


Image 1. Relative quantification of *kalrn* mRNA in zebrafish larvae brain after 24 hours after pentylentetrazole-evoked seizure.

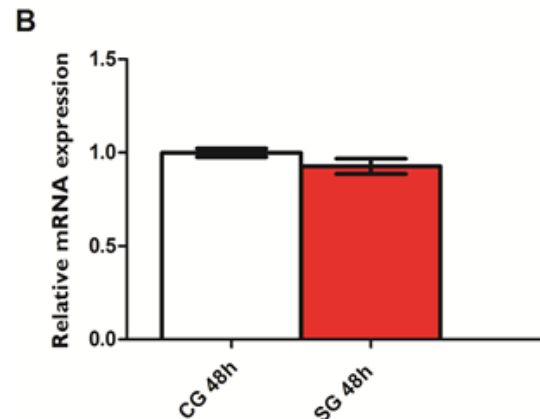


Image 2. Relative quantification of *kalrn* mRNA in zebrafish larvae brain after 48 hours after pentylentetrazole-evoked seizure.

The *kalrn* gene has been associated with human neurological disorders, such as schizophrenia, Alzheimer disease, Huntington's disease and ischemic stroke[2]; nevertheless, there is no information about this gene and epilepsy. Although no differences were found in *karln* transcripts at 24h and 48h after PTZ, we cannot conclude that *karln* does not play a role after seizures.

Conclusions

By investigating other time points of *karln* mRNA expression we may shed some light into the role of this gene and its relation with the mechanisms underlying plasticity in epilepsy.

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¹ Ma XM et al. Kalirin-7 is required for synaptic structure and function. *J Neurosci* **2008**, 28, 12368–12382.

² Penzes P, Remmers C. Kalirin Signaling: Implications for Synaptic Pathology. *Mol Neurobiol* **2012**, 45(1), 109–118.