**COX-1 expression is up-regulated after seizure in the zebrafish seizure model**

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**Introduction:** Experimental data have shown that microglia and astrocytes are activated in response to epileptic activity1. Microglia and astrocytes activation promotes the release of pro-inflammatory mediators such as prostaglandins, which are major players in the neuroinflammation process1. Cyclooxygenases (COX) -1 and 2 are enzymes responsible for the conversion of arachidonic acid into prostaglandins1. Traditionally, COX-1 and COX-2 isoforms have been considered constitutive and inducible expression, respectively. Therefore, most reports have focused on the role of COX-2 in the neuroinflammatory response in neurodegenerative diseases, and the contribution of COX-1 remains poorly investigated. Previous studies have demonstrated that COX-1 is inducible expressed in Alzheimer Disease, Multiple Sclerosis and following traumatic brain injury2,3,4. Moreover, it has been shown that *Cox-1* was increased with kindling progression, as well as in chronic upregulation of *Il-1β* expression in the hippocampus during brain aging in mouse hippocampus1,5,6. The main aim of this study was to evaluated the *cox-1* expression after PTZ-induced seizure in zebrafish brain.

**Materials and Methods:** All experimental protocols used in this study were reviewed and approved by the Ethical Committee for Animal Research of the University of Campinas (protocol number 3098-1). Seven days post fertilization (dpf) larvae were placed in a 24-well plate (one larvae per well) containing 15 mM PTZ (seizure group; SG) or PTZ-free water (control group; CG) for 60 min. Following this time, animals were cryoanaesthetized and their heads were isolated, quickly frozen in liquid nitrogen, and stored at −80°C until further processing. A total of five samples (n=5) were used for each group, control (CG) or seizure (SG), and each sample was composed by pooling five larval heads. Total RNA was extracted by standard TRIzol® method and its concentration and quality were determined by EpochTM spectrophotometer and electrophoresis using agarose gels. cDNA was generated using the High Capacity first-strand synthesis system for RT-PCR. Relative mRNA quantification was performed using the ABI 7500 Real Time PCR system with LuminoCt® qPCR ReadyMix, and TaqMan® Gene Expression Assay. The housekeeping gene *eef1a1l1* was used to normalize the mRNA level of *ptgs1*. Data were analyzed using the SDS 7500 software to estimate qPCR efficiency and quantify the relative gene expression.

**Results:** Our results showed an inducible *cox1* expression following 60 minutes of PTZ exposure. *Cox1* mRNA levels were up-regulated compared to control group (p = 0.004). The mean ± SEM of CG and SG were 1.1 ± 0.07 and 1.5 ± 0.07, respectively.

**Discussion:** The present study showed that the expression of COX-1 significantly increased following PTZ-induced seizure in the zebrafish model.

**Conclusion:** Our findings support evidence that COX-1 might play an important role in the neuroinflammatory response after seizure; thus, it may represent a possible therapeutic target to treat neuroinflammation in seizures. Because zebrafish seizure model is very willing to anti-epileptic drugs discovery and screening, this results can bring new opportunities to evaluate the effect of anti-inflammatory compounds on seizure suppression. Supported by FAPESP #2014/15640-8, #2013/19151-9, CEPID-BRAIN #2013/07559-3.

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