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جامعة الملك سعود King Saud University

Effect of Complex Febrile Seizure on the Expression of GPR12 in the Brain of Neonatal Rats

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INTRODUCTION

Febrile seizure (FS) is a type of convulsion that typically appears in childhood. This type of seizure causes abnormal patterns of electrical activity in the brain resulting in neuronal remodelling that could lead to the development of mesial temporal lobe epilepsy (mTLE). GPR12 is a G protein-coupled orphan receptor that plays a role in brain development and plasticity and promotes neurite outgrowth. Since neuronal remodelling results from febrile seizures and as GPR12 is involved in neurite outgrowth and axonal regeneration, this project aims to study the effect of complex febrile seizures on the expression of GPR12 in the brain of neonatal rats.

OBJECTIVES

- Assessing the effect of febrile seizure on neonatal rat brain tissue morphology.
- Assessing the effect of febrile seizure on the expression of GPR12 on protein level.
- Measuring the changes in the protein levels of Glial fibrillary acidic protein (GFAP).

METHODS

Animal model:
Twenty-five P10 old male Sprague-Dawley rat pups were divided into two groups. The FS group was exposed to heated steam air for 30 min after the onset of the seizure, and the control group was exposed to the normal temperature. The next day, the pups were sacrificed, and brains were collected.

Histological evaluation:
Hematoxylin and eosin histological staining for FFPE brain sections was carried out as Berger et al. (2019) described.

Immunohistochemistry:
The expression of GPR12 and GFAP were assessed in FFPE as described by Kozielewicz et al. (2017).

Statistical analysis:
The difference between the groups was analysed using Student's T-test, performed using Prism 8. P-value equal to ≤ 0.05 is considered significant.

CONCLOSIONS

A febrile seizure is associated with astrocytosis and upregulation of GPR12, which normally plays a role in axonal regeneration. Since FS causes neuronal remodelling leading to mTLE; therefore, GPR12 could be involved in the pathogenesis of neuronal rewiring observed in mTLE.

RESULTS

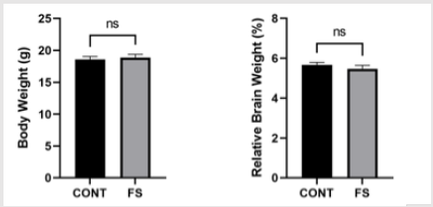

Fig 1: The body weight and the relative changes in the brain weight between febrile seizure and sham groups. ***A:*** body weight, ***B:*** relative brain weight. The data represented as mean ± SEM (n = 12, *p < 0.05)

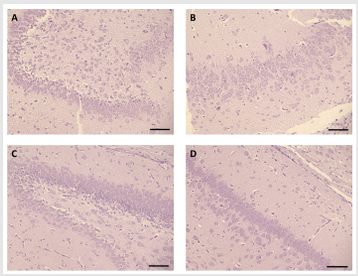

Fig 2: Unremarkable histological alterations in the Hippocampus of febrile seizure rat brain. ***A:*** Control DG and CA4, ***B:*** control CA1, ***C:*** FS DG and CA4 , ***D:*** FS CA1. (n = 6, scale bar = 20 um).

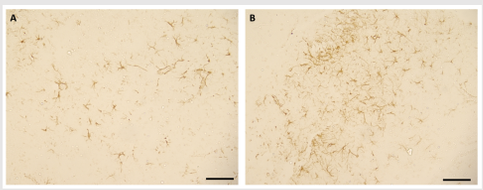

Fig 3: Immunoreactivity signals of GFAP protein in the cortex of neonatal rat brains. Upregulation of GFAP signals in the FS group indicates active astrocytosis. ***A:*** control group, ***B:*** FS group. (n = 6, scale bar = 20 um).

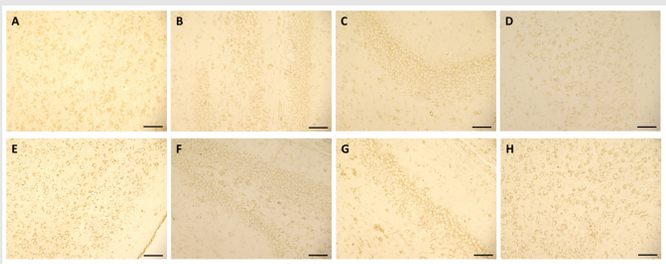

Fig 4: Immunoreactivity signals of GPR12 protein in the brain of febrile seizure neonatal rat. Moderate to high overexpression of GPR12 IR in the disease group. ***A:*** control cortex, ***B:*** control DG and CA4, ***C:*** control CA1, ***D:*** control entorhinal cortex. ***E:*** FS cortex, ***F:*** FS DG and CA4, ***G:*** FS CA1, ***H:*** FS entorhinal cortex. (n = 6, scale bar = 20 um).

REFERENCES