Assessment Of GPR12 Receptors Expression In Acute Traumatic Brain Injury Mouse Model.

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INTRODUCTION

Traumatic brain injury (TBI) is a leading cause of global mortality and morbidity worldwide[1] which leads to multiple abnormalities in brain structure and dysfunction of the blood vessels, thus resulting in necrosis and intracranial hemorrhage[2]. G-protein-coupled receptors (GPCRs) are the most targeted group for treating neurological disorders; one of those receptors is GPR12 which is found to be expressed in the central nervous system (CNS), with a high expression level in the brain's limbic areas[3]. Previous studies have demonstrated that GPR12 plays a role in the enhancement of cell survival and promoting neurotic outgrowth[4]. Given the importance of GPCRs in brain diseases and their imperative for treating neurological disorders, further investigation of GPR12 role in the pathophysiology of TBI is deemed necessary[5]. Therefore, our aim is to investigate the role of GPR12 in the pathophysiology of TBI in mice animal models.

OBJECTIVES

1. Measuring the mice recovery time after TBI induction.
2. Evaluating the impairment in locomotion in study groups.
3. Measuring the changes in the protein expression level of GPR12 and Tumor necrosis factor alpha (TNFα).

METHODS

Animal model:

Twenty-two C57BL/6 male mice with weight of 18-27g were divided equally into control and TBI groups. TBI was induced using the weight-drop method in TBI group after anesthesia with isoflurane and the control group was exposed to the same anesthesia without induction of TBI.

Behavioral studies:

Latency time and open field tests were performed for both groups to evaluate the general exploratory activity in the mice.

Biochemical studies:

The mice's brains were collected to perform western blot analysis.

Statistical Analysis:

The data were analyzed using a student's t-test (Prism 9). A p-value < 0.05 was considered statistically significant.

RESULTS

In the TBI group latency time was increased (p=0.0019) (Fig.1) and based on the open field test locomotor activity was decreased (P=0.0036) compared to the control group (Fig.2). Furthermore, tumor necrosis factorα (TNFα) was increased in TBI group (p<0.0001) (Fig.3), although no difference in GPR12 protein receptor expression was observed between the two groups (Fig.4).

Figure 1

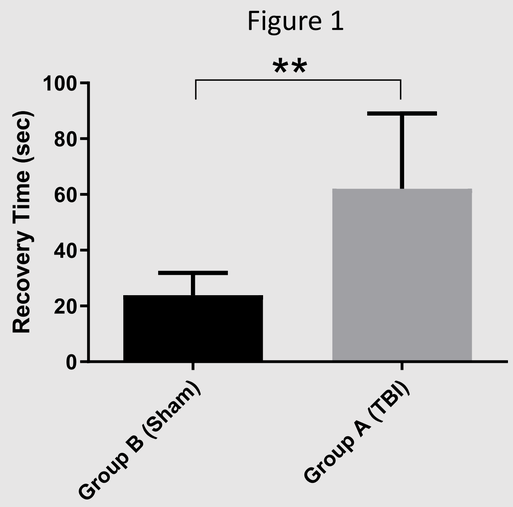


Figure 2

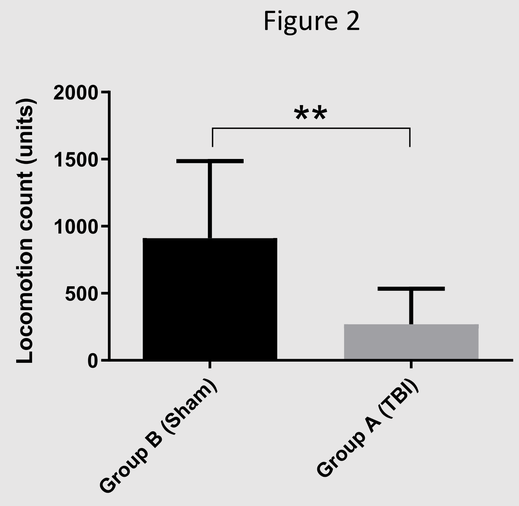


Figure 3

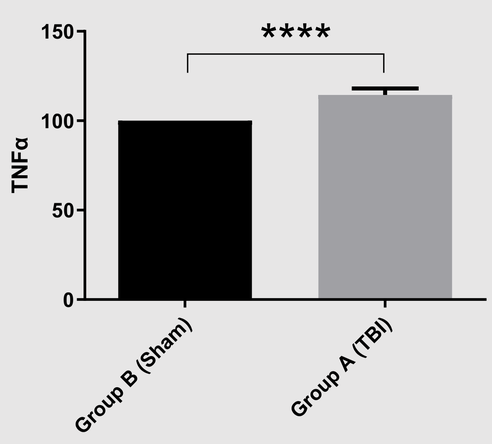
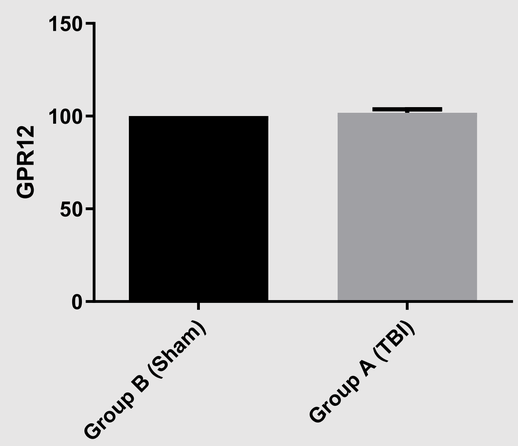


Figure 4



DISCUSSION & CONCLUSION

GPR12 plays a role in the enhancement of cell survival and promoting neurotic outgrowth. However, based on our results, further studies are needed to address the direct or indirect role of GPR12 in TBI.

REFERENCES

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