Review of: "Impending role of hippocampal neurogenesis in the development of chronic epilepsy following seizures after Kainic acid and Pentylenetetrazol treatment"

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Potential competing interests: No potential competing interests to declare.

The article shows in a very simple and didactic way the organization of the experiments, the groups, and protocols carried out.

It is evident that the protocols using BrDU and immunostaining attempt to elucidate the processes that occur in the acute and chronic phases in the animal models used.

The figure 7 shows the results of immunocytochemical localization and immunoblotting of NGF and BDNF, but in the results, the authors used: “expression BDNF, and NGF were also investigated,” that can cause some confusion. I suggest that they change the word “expression.”

Additionally, the figure of GABA immunostaining is missing in the paper.

The results of GABA immunopositive neurons state: "After 48 hr of SE, a decline in GABA immunostaining was observed in the hippocampus of PTZ-treated brains, while it remained unchanged in KA-treated brains compared with the control brain."

Could it be because a negative feedback in GABA expression was caused by PTZ (antagonist action)?

“Interestingly, divergent outcomes were observed in GABA immunostaining between the two groups after a duration of 8 weeks. The brain subjected to PTZ treatment exhibited higher intensity compared to the rats treated with KA.” In this last afirmation, how much is the diference? This could be the cause of the absence of spontaneous recurrent motor seizures in PTZ animals.

The work is very interesting, and I hope to be able to read it soon. Congratulations to the authors involved.