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PEER-REVIEW REPORT

Name of journal: *World Journal of Psychiatry*

Manuscript NO: 85267

Title: Hippocampus Protection from Apoptosis by Baicalin in a LiCl-Pilocarpine-Induced Rat Status Epilepticus Model Through Autophagy Activation

Provenance and peer review: Unsolicited Manuscript; Externally peer reviewed

Peer-review model: Single blind

Reviewer's code: 06521484
Position: Peer Reviewer
Academic degree: MD
Professional title: Doctor
Reviewer's Country/Territory: Ghana
Author's Country/Territory: China

Manuscript submission date: 2023-05-19

Reviewer chosen by: AI Technique

Reviewer accepted review: 2023-05-21 02:13

Reviewer performed review: 2023-05-31 02:01

Review time: 9 Days and 23 Hours

Scientific quality	[ ] Grade A: Excellent [Y] Grade B: Very good [ ] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish
Novelty of this manuscript	[ ] Grade A: Excellent [Y] Grade B: Good [ ] Grade C: Fair [ ] Grade D: No novelty
Creativity or innovation of this manuscript	[ ] Grade A: Excellent [Y] Grade B: Good [ ] Grade C: Fair [ ] Grade D: No creativity or innovation

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Scientific significance of the conclusion in this manuscript	[ ] Grade A: Excellent [Y] Grade B: Good [ ] Grade C: Fair [ ] Grade D: No scientific significance
Language quality	[ ] Grade A: Priority publishing [Y] Grade B: Minor language polishing [ ] Grade C: A great deal of language polishing [ ] Grade D: Rejection
Conclusion	[ ] Accept (High priority) [ ] Accept (General priority) [Y] Minor revision [ ] Major revision [ ] Rejection
Re-review	[Y] Yes [ ] No
Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous
	Conflicts-of-Interest: [ ] Yes [Y] No

SPECIFIC COMMENTS TO AUTHORS

Status epilepticus (SE) is a complicated pathophysiological process, involving many mechanisms and lacking of effective therapy. In this study, the authors aimed at exploring the protective value of Baicalin in treating hippocampus apoptosis caused by SE. The authors used animal models, Nissl staining, TUNEL staining, Western Blotting, and immunofluorescent labelling to verify their hypothesis. The results showed that LiCl-Pilocarpine Induced rat SE successfully, and Baicalin protected nerve cells from apoptosis by increasing autophagy. So, in my opinion, this paper is well-written. The experimental design is reasonable, and the results reflects the conclusion as well. I recommend its acceptance after the minor revision. The detailed comments are: 1. The authors have reviewed that indications of autophagy variation are observed in several neuroprotective drugs. What are these drugs? Compared with these drugs, what is the key advantage of Baicalin? 2. In the section of Introduction, the authors indicated that elimination of apoptotic organelles by autophagy is potentially the mechanism of anti-epilepsy activity of Baicalin; however, the precise mechanism remains largely unknown. Since this paper did not discuss the underlying mechanism of Baicalin for

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inducing autophagy, the above expression is not appropriate here. 3. Why the authors only used male rats in this study?

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Reviewer's code: 06520997
Position: Peer Reviewer
Academic degree: MD, PhD
Professional title: Assistant Professor, Doctor
Reviewer's Country/Territory: Poland
Author's Country/Territory: China

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Reviewer performed review: 2023-05-31 09:03

Review time: 7 Days and 8 Hours

Scientific quality	[ ] Grade A: Excellent [ ] Grade B: Very good [Y] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish
Novelty of this manuscript	[ ] Grade A: Excellent [ ] Grade B: Good [Y] Grade C: Fair [ ] Grade D: No novelty
Creativity or innovation of this manuscript	[ ] Grade A: Excellent [Y] Grade B: Good [ ] Grade C: Fair [ ] Grade D: No creativity or innovation

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Conclusion	[ ] Accept (High priority) [ ] Accept (General priority) [Y] Minor revision [ ] Major revision [ ] Rejection
Re-review	[Y] Yes [ ] No
Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous
	Conflicts-of-Interest: [ ] Yes [Y] No

SPECIFIC COMMENTS TO AUTHORS

The authors build a rat model of status epilepticus via LiCl-pilocarpine induction and use it to investigate the protective role of possibility of Baicalin in the progress of SE. After reasonable grouping the rat models, the authors showcased that Baicalin can effectively decrease the apoptosis of hippocampus by inducing autophagy signaling, and such a protective role can be inhibited using 3-Methyladenine. This result also draws a conclusion that Baicalin is a potential drug for SE treatment. In short, the topic of this manuscript is timely and interesting. The authors have organized the manuscript rationally, with good methodology and well-written English. However, some important editing needs to be done before publication: 1) In part 2.2, the rats are randomly divided into four groups: control, SE, SE + B100, and SE + B200. However, rats in groups SE + B100 and SE + B200 are intraperitoneally injected with 90 mg/kg and 180 mg/kg of Baicalin, respectively. So, the names of SE + B100 and SE + B200 are very confusing for the readers. 2) After testing the protective role of Baicalin for decreasing the apoptosis of hippocampus, the authors divided the rats into for groups once again, including control, SE, SE+Baicalin, and SE+Baicalin+3-MA. What is the treatment on the rats in group

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SE+Baicalin? Why using such a concentration of Baicalin for treating rats in this group?