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PEER-REVIEW REPORT

Name of journal: *World Journal of Gastroenterology*

Manuscript NO: 72524

Title: CFTR prevents ischemia/reperfusion induced intestinal apoptosis via inhibiting PI3K/AKT/NF-κB pathway

Provenance and peer review: Unsolicited Manuscript; Externally peer reviewed

Peer-review model: Single blind

Reviewer's code: 06143430
Position: Peer Reviewer
Academic degree: MD

Professional title: Doctor

Reviewer's Country/Territory: Australia

Author's Country/Territory: China

Manuscript submission date: 2021-11-10

Reviewer chosen by: AI Technique

Reviewer accepted review: 2021-11-16 10:19

Reviewer performed review: 2021-11-29 12:05

Review time: 13 Days and 1 Hour

Scientific quality	[ ] Grade A: Excellent [Y] Grade B: Very good [ ] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish
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

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Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous Conflicts-of-Interest: [ ] Yes [Y] No

SPECIFIC COMMENTS TO AUTHORS

The innate and adaptive apoptosis resulting from intracellular damage and the translocation of gut pathogens play a crucial role in the pathological process of intestinal ischemia/reperfusion. Extensive researches for the role of CFTR focus on epithelial cell function, while its role in other types of cells, such as endothelial cells, is largely unclear. Many studies showed that cystic fibrosis patients had endothelial perturbation and microvascular dysfunction, suggesting that CFTR deficiency contributes to endothelial dysfunction. The apoptosis of intestinal cells caused by ischemia can cause cell damage and provoke epithelial barrier dysfunction during reperfusion, resulting in system dysfunction. However, the mechanism of apoptosis induced by the hypoxia/reoxygenation remains unclear. This study is designed to investigate the effects of CFTR on ischemia/reperfusion-induced intestinal apoptosis and its underlying molecular mechanisms. Overall, the study is well designed. The methods are reasonable and described in detail. The Western blotting of the proteins are good. The results are very interesting. Those results were discussed well, and the references are updated. Comments: 1. Some minor language polishing should be corrected. 2. The images are too small. Please make an update. 3. Please list the limit of the study.

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Provenance and peer review: Unsolicited Manuscript; Externally peer reviewed

Peer-review model: Single blind

Reviewer's code: 06143437
Position: Peer Reviewer
Academic degree: MD
Professional title: Doctor
Reviewer's Country/Territory: United Kingdom
Author's Country/Territory: China

Manuscript submission date: 2021-11-10

Reviewer chosen by: AI Technique

Reviewer accepted review: 2021-11-16 10:27

Reviewer performed review: 2021-11-29 12:06

Review time: 13 Days and 1 Hour

Scientific quality	[ ] Grade A: Excellent [Y] Grade B: Very good [ ] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish
Language quality	[ ] Grade A: Priority publishing [Y] Grade B: Minor language polishing [ ] Grade C: A great deal of language polishing [ ] Grade D: Rejection
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Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous Conflicts-of-Interest: [ ] Yes [Y] No

SPECIFIC COMMENTS TO AUTHORS

This study is interesting. I have some comments about the manuscript: Please consider the consistencies of terms, manufacturers, and SI units that you use throughout the manuscript for better viewing and understanding. Is it II/R or I/R? Caco2 or Caco-2 or CaCo2 cells? What is LV in CFTR? LV was not introduced previously, was it abbreviated from Lentivirus? What is MOI 20 in line 142? please explain in brief. If you are using abbreviated terms, please describe it at the beginning before using the abbreviation. Please refer to the guidelines for authors (for basic research) when constructing the manuscript. There are many mistyped words and poorly arranged spacings in this manuscript. Please refer the previous study that you mention in the text in line 159. Please explain about the in-vitro sample size or replication. In the results section, please give the results data as stated in the statistical analysis (data provided in mean +/- SEM). Please re-arrange the figures regarding the sizes and sequences, shorten and wrap the figure explanation and merge them in the results section.

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Provenance and peer review: Unsolicited Manuscript; Externally peer reviewed

Peer-review model: Single blind

Reviewer's code: 06143360

Position: Peer Reviewer

Academic degree: MD

Professional title: Doctor

Reviewer's Country/Territory: Italy

Author's Country/Territory: China

Manuscript submission date: 2021-11-10

Reviewer chosen by: AI Technique

Reviewer accepted review: 2021-11-16 10:19

Reviewer performed review: 2021-11-29 12:08

Review time: 13 Days and 1 Hour

Scientific quality	[Y] Grade A: Excellent [ ] Grade B: Very good [ ] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish
Language quality	[Y] Grade A: Priority publishing [ ] Grade B: Minor language polishing [ ] Grade C: A great deal of language polishing [ ] Grade D: Rejection
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Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous Conflicts-of-Interest: [ ] Yes [Y] No

SPECIFIC COMMENTS TO AUTHORS

This experimental study indicates that the overexpression of CFTR protects CaCo2 cells from H/R-induced apoptosis through the PI3K/AKT/NF-κB signaling pathway. The study is very interesting, and worthy for publication.