

PEER-REVIEW REPORT

Name of journal: World Journal of Gastroenterology

Manuscript NO: 65462

Title: EZH2 contributes to apoptosis and inflammatory response by inactivating Janus kinase 2/signal transducers and activators of transcription signaling in inflammatory bowel disease

Reviewer's code: 05843492

Position: Peer Reviewer

Academic degree: MD

Professional title: Doctor

Reviewer's Country/Territory: China

Author's Country/Territory: China

Manuscript submission date: 2021-03-07

Reviewer chosen by: AI Technique

Reviewer accepted review: 2021-03-10 03:31

Reviewer performed review: 2021-03-11 10:22

Review time: 1 Day and 6 Hours

Scientific quality	<input type="checkbox"/> Grade A: Excellent <input type="checkbox"/> Grade B: Very good <input checked="" type="checkbox"/> Grade C: Good <input type="checkbox"/> Grade D: Fair <input type="checkbox"/> Grade E: Do not publish
Language quality	<input type="checkbox"/> Grade A: Priority publishing <input checked="" type="checkbox"/> Grade B: Minor language polishing <input type="checkbox"/> Grade C: A great deal of language polishing <input type="checkbox"/> Grade D: Rejection
Conclusion	<input type="checkbox"/> Accept (High priority) <input type="checkbox"/> Accept (General priority) <input type="checkbox"/> Minor revision <input checked="" type="checkbox"/> Major revision <input type="checkbox"/> Rejection
Re-review	<input checked="" type="checkbox"/> Yes <input type="checkbox"/> No
Peer-reviewer	Peer-Review: <input checked="" type="checkbox"/> Anonymous <input type="checkbox"/> Onymous

statementsConflicts-of-Interest: [] Yes [**Y**] No**SPECIFIC COMMENTS TO AUTHORS**

1.DSS is often used to induce colitis in mice, but there seems to be no evidence for treating cells?It is reported that EZH2 inhibits TNFalpha-regulated apoptosis and inflammation in IBD, cells should be stimulated with TNF-a. 2.Since IBD is not a neoplastic disease, the use of normal intestinal epithelial cells (such as NCM460, FHC) can better reflect the pathogenesis of IBD. Please add the relevant experiments of normal intestinal epithelial cell lines. 3.How about the expressions of EZH2 in patients with IBD and in colitis mice?

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Title: EZH2 contributes to apoptosis and inflammatory response by inactivating Janus kinase 2/signal transducers and activators of transcription signaling in inflammatory bowel disease

Reviewer's code: 03321798

Position: Editorial Board

Academic degree: MD, PhD

Professional title: Consultant Physician-Scientist

Reviewer's Country/Territory: Italy

Author's Country/Territory: China

Manuscript submission date: 2021-03-07

Reviewer chosen by: Man Liu

Reviewer accepted review: 2021-03-12 08:54

Reviewer performed review: 2021-03-18 15:19

Review time: 6 Days and 6 Hours

Scientific quality	<input type="checkbox"/> Grade A: Excellent <input checked="" type="checkbox"/> Grade B: Very good <input type="checkbox"/> Grade C: Good <input type="checkbox"/> Grade D: Fair <input type="checkbox"/> Grade E: Do not publish
Language quality	<input type="checkbox"/> Grade A: Priority publishing <input checked="" type="checkbox"/> Grade B: Minor language polishing <input type="checkbox"/> Grade C: A great deal of language polishing <input type="checkbox"/> Grade D: Rejection
Conclusion	<input type="checkbox"/> Accept (High priority) <input type="checkbox"/> Accept (General priority) <input checked="" type="checkbox"/> Minor revision <input type="checkbox"/> Major revision <input type="checkbox"/> Rejection
Re-review	<input type="checkbox"/> Yes <input checked="" type="checkbox"/> No
Peer-reviewer	Peer-Review: <input checked="" type="checkbox"/> Anonymous <input type="checkbox"/> Onymous

statementsConflicts-of-Interest: [☐] Yes [☒] No**SPECIFIC COMMENTS TO AUTHORS**

This study analyzes the role of EZH2 in the inflammatory response and the underlying mechanisms in inflammatory bowel disease. The topic is important as epigenetic mechanisms have an emerging role in pathogenesis and are involved as therapeutic targets in IBD. The specific role of EZH2 has been rarely investigated in IBD with contrasting results. The authors have managed to perform a rigorous lab based study and to clearly present their results in this paper. Studies like this are needed to identify new potential therapeutic molecules in IBD. I recommend the paper for publication in view of the relevance of its topic and the solid experimental design.

Minor comments: Overall, the paper reads well and it is easy for the reader to follow its flow. I would suggest to review the grammar and I indicate some minor language changes, prior to publication (see the attached file).

RE-REVIEW REPORT OF REVISED MANUSCRIPT

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Reviewer's code: 05843492

Position: Peer Reviewer

Academic degree: MD

Professional title: Doctor

Reviewer's Country/Territory: China

Author's Country/Territory: China

Manuscript submission date: 2021-03-07

Reviewer chosen by: Jia-Ru Fan

Reviewer accepted review: 2021-04-12 09:16

Reviewer performed review: 2021-04-12 09:43

Review time: 1 Hour

Scientific quality	<input type="checkbox"/> Grade A: Excellent <input checked="" type="checkbox"/> Grade B: Very good <input type="checkbox"/> Grade C: Good <input type="checkbox"/> Grade D: Fair <input type="checkbox"/> Grade E: Do not publish
Language quality	<input type="checkbox"/> Grade A: Priority publishing <input checked="" type="checkbox"/> Grade B: Minor language polishing <input type="checkbox"/> Grade C: A great deal of language polishing <input type="checkbox"/> Grade D: Rejection
Conclusion	<input type="checkbox"/> Accept (High priority) <input checked="" type="checkbox"/> Accept (General priority) <input type="checkbox"/> Minor revision <input type="checkbox"/> Major revision <input type="checkbox"/> Rejection
Peer-reviewer statements	Peer-Review: <input checked="" type="checkbox"/> Anonymous <input type="checkbox"/> Onymous Conflicts-of-Interest: <input type="checkbox"/> Yes <input checked="" type="checkbox"/> No



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SPECIFIC COMMENTS TO AUTHORS

The article is well written with key changes, and the figures show the ideas well. I recommend the paper for publication.