

PEER-REVIEW REPORT

Name of journal: *World Journal of Gastroenterology*

Manuscript NO: 78854

Title: 3,6-dichlorobenzo[b]thiophene-2-carboxylic acid alleviates ulcerative colitis by suppressing mammalian target of rapamycin complex 1 activation and regulating intestinal microbiota

Provenance and peer review: Unsolicited Manuscript; Externally peer reviewed

Peer-review model: Single blind

Reviewer's code: 06363659

Position: Peer Reviewer

Academic degree: MD

Professional title: Doctor

Reviewer's Country/Territory: China

Author's Country/Territory: China

Manuscript submission date: 2022-07-20

Reviewer chosen by: AI Technique

Reviewer accepted review: 2022-08-29 13:13

Reviewer performed review: 2022-09-11 15:28

Review time: 13 Days and 2 Hours

Scientific quality	☐ Grade A: Excellent ☐ Grade B: Very good ☒ Grade C: Good ☐ Grade D: Fair ☐ Grade E: Do not publish
Language quality	☐ Grade A: Priority publishing ☒ Grade B: Minor language polishing ☐ Grade C: A great deal of language polishing ☐ Grade D: Rejection
Conclusion	☐ Accept (High priority) ☐ Accept (General priority) ☒ Minor revision ☐ Major revision ☐ Rejection

Re-review	[☒] Yes [☐] No
Peer-reviewer statements	Peer-Review: [☒] Anonymous [☐] Onymous Conflicts-of-Interest: [☐] Yes [☒] No

SPECIFIC COMMENTS TO AUTHORS

The article under review aims to investigate the anti-UC effect of BT2 and the underlying mechanism involved. The result indicated that BT2 significantly ameliorated DSS-induced UC, and suppressed BCAA-associated mTORC1 activation and the modulated of intestinal flora. However, the potential mechanisms of intestinal flora and BCAA-associated mTORC1 activation have not been elucidated. Here are some comments.

1. "Part 2.3. Induction of UC and experimental design: the other groups were given 3.5% DSS in drinking water Mice in the control group and DSS group were given 0.5% carboxymethylcellulose sodium." It seems that 3.5% DSS concentration a bit high, and there is no mouse death? And why mice were given 0.5% carboxymethylcellulose sodium?

2. Fig4. BT2 suppressed the activity of mTORC1 signaling pathway. The experiment can only prove that he can inhibit the mTORC1 signaling pathway, not enough to prove that bt2 is inhibiting the mTORC1 signaling pathway by inhibiting bcaa metabolic disorder, suggesting that relevant experiments could be added if it is possible.

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Reviewer's code: 05382551

Position: Editorial Board

Academic degree: PhD

Professional title: Associate Professor

Reviewer's Country/Territory: Spain

Author's Country/Territory: China

Manuscript submission date: 2022-07-20

Reviewer chosen by: Dong-Mei Wang

Reviewer accepted review: 2022-10-13 14:56

Reviewer performed review: 2022-10-13 23:17

Review time: 8 Hours

Scientific quality	☐ Grade A: Excellent ☐ Grade B: Very good ☒ Grade C: Good ☐ Grade D: Fair ☐ Grade E: Do not publish
Language quality	☐ Grade A: Priority publishing ☐ Grade B: Minor language polishing ☒ Grade C: A great deal of language polishing ☐ Grade D: Rejection
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**Baishideng
Publishing
Group**

7041 Koll Center Parkway, Suite
160, Pleasanton, CA 94566, USA
Telephone: +1-925-399-1568
E-mail: bpgoffice@wjgnet.com
<https://www.wjgnet.com>

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

SPECIFIC COMMENTS TO AUTHORS

The article is within the scope of the journal and proposes an interesting topic. It is well written and organized. His reading is fluent. Regarding the content. It is an original contribution that presents a well-designed experiment. The materials and methods used are explained and the results obtained are discussed. However, two aspects should be improved: a) The state of the art should be extended. b) A section of conclusions and future work should be included in which the scientific contribution of the work is indicated and a set of lines of future work are established.