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PEER-REVIEW REPORT

Name of journal: *World Journal of Clinical Cases*

Manuscript NO: 91841

Title: Exploring the Pathogenesis from autophagy and Screening Natural Drugs for Therapeutic Potentials of active Ulcerative Colitis

Provenance and peer review: Unsolicited Manuscript; Externally peer reviewed

Peer-review model: Single blind

Reviewer's code: 03837089
Position: Peer Reviewer
Academic degree: PhD

Professional title: Professor

Reviewer's Country/Territory: Taiwan

Author's Country/Territory: China

Manuscript submission date: 2024-01-07

Reviewer chosen by: AI Technique

Reviewer accepted review: 2024-01-07 15:09

Reviewer performed review: 2024-01-10 14:19

Review time: 2 Days and 23 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Scientific quality	[ ] Grade A: Excellent [ ] Grade B: Very good [Y] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish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 [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) [ ] Minor revision [Y] Major revision [ ] Rejection
Re-review	[ ] Yes [Y] No
Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous
	Conflicts-of-Interest: [ ] Yes [Y] No

SPECIFIC COMMENTS TO AUTHORS

After reviewing this study, I strongly suggest the authors splitting this article into two. Despite the current article shows some novelty to UC pathology and treatment, the novelty of this article is minor. If the authors can split this article into two, the novelty will be greater. Second, the authors use autophagy as a target for UC treatment. However, the pathological role of autophagy in UC progression is not clear, at least in the introduction of this article. Therefore, the reader cannot understand the rationale of targeting autophagy for UC treatment. Continuous from the aforementioned suggestion, autophagy in disease progression can be therapeutic or pathologic. For diminishing pathologic autophagy, inhibitors are necessary; otherwise, activators would be a choice. However, the molecular docking can just present the simulation of binding rather than promotion or inhibition of the autophagy. If the authors want to claim TCMSP benefit in UC treatment, wet lab data for assessing the influence of the autophagy activity is necessary. Continuous from the aforementioned suggestion. The authors found several autophagy-related genes in UC specimen. However, such genes are upstream or downstream mediators of UC progression is not evaluated in this study. This limitation

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diminishs the novelty of this study.