Dear Editor,

Thank you for considering the manuscript entitled "Identification of marker genes associated with m6A and autophagy in ulcerative colitis" (91644). We really appreciate all the valuable comments and constructive suggestions from reviewers. We have revised the manuscript and a point-by-point response was enclosed. We would like to re-submit the revised manuscript to the World Journal of Clinical Cases, and hope it is acceptable for publication in the journal. Please do not hesitate to contact us for any question or concern.

We look forward to your final decision.

Sincerely yours

Yan-Cheng Dai

Responses to reviewer:

Reviewer #1: It is known that methylation of m6A and autophagy plays a vital role in the pathogenesis of UC. However, there is no combination study for both of these factors. This study enlightens the pathogenesis of UC and might imply the clinical setting. They found that mRNA levels of BAG3 and P4HB are higher, and FMRI and TP531NP2 are lower in the DSS-induced colitis group in a fundamental study. However, there are some concerns about this article. 1. Please explain precisely what three machine learning is. 2. Please briefly summarize the procedure of the experiment as a figure. 3. The authors proposed the need for more experimentation and clinical application studies. What are these?

Response: We have revised the manuscript. 1. The "three machine learning" is wrong, we have revised "three machine learning" to "the machine learning". 2. We have added Figure 8 to summarize the experimental procedure. 3. We proposed the need for more experimentation and clinical application studies. we speculate that Fmr1 could affect the occurrence and development of colitis through the intestinal flora and its metabolites; BAG3 is involved in the signalling pathway related to cell proliferation, migration, invasion and

chemical resistance control in colitis; BAG3, P4HB and TP53INP2-related NF-κB/TNF-α signalling pathways have curative potential in clinical UC treatment; targeted therapy that promotes macrophage polarisation by regulating TP53INP2 can reconstruct the homeostasis of intestinal immune microenvironment and restore post-inflammatory tissue homeostasis, which is a new focus of UC therapy. Therefore, m6A and ARGs how to participate in the occurrence and development of UC, as well as the genes identification are as possible markers for assessing UC severity and developing innovative UC targeted therapeutic approaches. The specific regulatory mechanisms of these genes need further experimental research and clinical application research.

Thank you for your valuable advice.