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PEER-REVIEW REPORT

Name of journal: *World Journal of Gastroenterology*

Manuscript NO: 72735

Title: Long noncoding RNA TNFRSF10A-A promotes colorectal cancer through upregulation of HuR

Provenance and peer review: Unsolicited Manuscript; Externally peer reviewed

Peer-review model: Single blind

Reviewer's code: 05776245
Position: Peer Reviewer
Academic degree: BSc, MSc

Professional title: Academic Research, Research Scientist, Teaching Assistant

Reviewer's Country/Territory: Poland

Author's Country/Territory: China

Manuscript submission date: 2021-10-26

Reviewer chosen by: AI Technique

Reviewer accepted review: 2021-11-14 09:17

Reviewer performed review: 2021-11-17 21:12

Review time: 3 Days and 11 Hours

Scientific quality	[ ] Grade A: Excellent [Y] Grade B: Very good [ ] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish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

Dear Authors, a very solid original article. You identified the TNFRSF10A-AS1/miR-3121- 3p/HuR axis that could be of therapeutic importance for CRC and should be further investigated. The preparation of the manuscript is of high quality and conclusions appropriately summarize the data from this study. I do have some suggestions for further improvement (see below), but overall I am satisfied with the presentation and study insights. 1. In the Introduction section, I would like to see more information about TNFRSF10A-AS1 in CRC, to underline that this direction was indeed relevant. One of examples would be to describe the "inconsistency of its role", as you mentioned in the sentence "However, the role of TNFRSF10A-AS1 in tumors is inconsistent and the exact mechanism is unclear" - I would like to know more details regarding this aspect. Another one could be some additional literature data of TNFRSF10A-AS1 role in CRC (currently, it is only about association of this lncRNA with autophagy in CRC - or maybe this is the only available data?). Also, I would limit the last sentences of Introduction about what was done and what are the findings (this could fit well in Abstract or Conclusions), and focus more on clear rationale and study aim. 2. Why did you use 6 colon cancer cell lines to conclude about CRC and not e.g. three representing rectal carcinoma, and the next three representing colon cancer? I was quite sure that there are some established cell lines of rectal carcinoma that could be used (see Cellosaurus for example) 3. At the beginning of Results, you mentioned about using GEPIA online tool - I would add short mention about it also in Materials and Methods. Through that, it will be possible for you to include information like p-value threshold, usage of log-scale and also some small but important detail that the box-plots present

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TCGA cancer data but in terms of normal tissues - there are TCGA matched samples as well as GTEx data. 4. In section "miR-3121-3p is downregulated in CRC" I would change "procancer" to "procancerous". 5. The next suggestion is about figures - although they are of high quality and the presentation is on point, the referencing in text makes it sometimes difficult to switch between figures. This is visible mainly in Figure 3 and 4, where in text you mention about e.g. Figure 3A-D, then about 4C, then again 3E, then 4A, then 3F etc. I understand that you made you figures taking into account the aesthetics (and this is highly welcomed) but it could be hard for reader to follow the switching of figures several times. Please consider improving this aspect. 6. In terms of the Figure 1, I believe that in-text mention of Fig1C and 1D should be switched ("TNFRSF10A-AS1 was also significantly upregulated in colon cancer cell lines compared with FHC cells (Fig. 1D). These results were confirmed by RT-PCR (Fig. 1C)"). The data on Fig1C does not represent expression determined through RT-PCR, while the quoted part states this. 7. Why DLD1 and HCT116 were used to show localization in nucleus/cytoplasm in Fig1E? I also wonder if some similarities between cell lines determined their grouping in pairs during performance of other assays (e.g. DLD1 and HT29 together, and HCT116 and SW480 together, as visible on Fig2A)? In addition, why HT29 is lacking on Figure 2G?

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Reviewer's code: 05119548
Position: Editorial Board
Academic degree: MD, PhD

Professional title: Professor

Reviewer's Country/Territory: Japan

Author's Country/Territory: China

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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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SPECIFIC COMMENTS TO AUTHORS

Authors performed well planned experiments and obtained huge amount of data. They concluded TNFRSF10A-AS1 exerts a tumor-promoting function through the miR-3121-3p/HurTNFRSF10A-AS1 axis in CRC and thus may be a potential therapeutic for CRC. It should be considered the concept of this manuscript will be more confirmed, if the overexpression of Hur n CRC tissues compared with adjacent normal mucosa would be shown ias well as TNFRSF10A-AS.