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Dear Editor,

Thank you for your help and your advice. And thank you very much for your decision letter and advice on our manuscript (Manuscript 31173). We also thank the reviewer for the comments and suggestions. These comments and suggestions are very helpful for us. We have revised the manuscript accordingly.

We hope the revision can be improved to be acceptable for the publication in your journal.

Thank you very much for your help to us again!

Look forward to hearing from you soon.

With best wishes,

Yours sincerely,

Dr Chaochao Qin

## ESPS PEER-REVIEW REPORT

**Name of journal:** World Journal of Gastroenterology
**ESPS manuscript NO:** 31173
**Title:** Macrophage inflammatory protein-2 (MIP-2) as mediator of inflammation in acute liver injury
**Reviewer's code:** 03087967
**Reviewer's country:** Turkey
**Science editor:** Jing Yu
**Date sent for review:** 2016-11-03 11:13
**Date reviewed:** 2016-11-15 15:23

| CLASSIFICATION | LANGUAGE EVALUATION | SCIENTIFIC MISCONDUCT | CONCLUSION |
|---|---|---|---|

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|---|---|---|---|
| [ ] Grade A: Excellent | [ ] Grade A: Priority publishing | Google Search: | [ Y] Accept |
| [ Y] Grade B: Very good | [ ] Grade B: Minor language polishing | [ ] The same title | [ ] High priority for publication |
| [ ] Grade C: Good | [ Y] Grade C: A great deal of language polishing | [ ] Duplicate publication | [ ] Rejection |
| [ ] Grade D: Fair | [ ] Grade D: Rejected | [ ] Plagiarism | [ ] Minor revision |
| [ ] Grade E: Poor | | [ ] No | [ ] Major revision |
| | | BPG Search: | |
| | | [ ] The same title | |
| | | [ ] Duplicate publication | |
| | | [ ] Plagiarism | |
| | | [ ] No | |

## COMMENTS TO AUTHORS

It is an extensive review on MIp-2 and liver toxicity and regeneration. There are however multiple mistakes in language. Some of them were given below. Abtract There are numerous language errors in the abstract. Some of them are: MIP-2 effect on neutrophil recruitment and activation through p38 MAPK-dependent signal pathway, by binding to its specific receptors, CXC chemokine receptor 1 (CXCR1) and CXCR2. Then the MIP-2-recruited and activated neutrophils can accelerate liver inflammation by releasing various kinds of inflammatory mediators. There are also many errors in the main text, some of the sentences that require corrections are give below. MIP-2 production can be effectively inhibited in lipopolysaccharide (LPS)-stimulated mouse peritoneal macrophage cell line, RAW 264.7, though down-regulating mRNA accumulation and protein expression of membarne TLR4/mCD14. The results indicated that upstream inhibition of TLR4/CD14-mediated inflammation pathway may be an effective therapeutic approach for attenuating damaging immune activation [24]. So et al. found that Scutellariae Radix and Liriopis Tuber (SL) could significantly inhibit the release of MIP-2 in LPS-induced RAW 264.7 cells [25]. When injected in vivo as recombinant chemokines KC and MIP-2 in models of inflammation, each can cause neutrophil influx [33]- order of words is inappropiate. In liver injury, neutralizing KC and MIP-2 would result in less neutrophil extravasation and reduce neutrophil-induced injury in a mouse model of cholestatic liver damage [35] One is the production of reactive oxygen intermediates may directly induce hepatic endothelium damage either or indirectly induce tissue injury by triggering other inflammatory mediators [47, 48]. Neutrophil-derived proteases facilitate extravasation and are involved in the regulation of inflammatory mediator production. However, there are few clinical reports about the effect of MIP-2 in acute liver injury.-what are they Further study showed that the LPS-induced MIP-2 production was dependent on NF-κB activation via inhibition of nuclear factor kappa-B kinase (IKK) pathway [79-81]. Single dose γ-irradiation (25 Gy) that was focused on the liver could recruite neutrophils attached to the portal vessels and to portal (myo)fibroblasts in the liver, and several chemokines may be necessary in its recruitment, adhesion, and transmigration. A fast and early induction of gene expression of several chemokines and the chemokines receptor CXCR2 gene expression in irradiated liver tissue and portal area were

observed.- expressions While, the later phase of I/R injury is dependent upon hepatic neutrophil sequestration, and the subsequent increased adherence between neutrophil-endothelial- in complete sentence. Excessive alcohol exposure leads to alcohol liver disease (ALD), a major cause of morbidity and mortality worldwide. Alcohol abuse also causes hepatic stestosis.- should be steatosis he decreased very low-density lipoprotein (VLDL) secretion, and the increasing levels of chemokine secretion and adhesion molecule expression [97- 99].- increased In alcohol-fed male Sprague-Dawley (SD) rats, the alcohol intoxication-induced hepatic injury through endotoxin influx in the circulation, stimulated the Kupffer cells to produce MIP2 and upregulated the expression of adhesion molecules on hepatic cells, then resulted in altered hepatic function and hepatotoxicity by hepatic neutrophils recruitment [100]- rephrase Further mechanism study by using inhibitors of signaling kinases showed that the induction of MIP-2 was correlated with p42/p44 and PI3 kinase but not p38 kinase signaling in hypoxia [105]. Further study showed that the properties in liver regeneration of signaling glutamic acid-lysine-arginine-CXC (ELR-CXC) chemokines, such as MIP-2, in acetaminophen challenge are attributed mainly to its ELR m

**Responses to comments**

Dear editor:

Thank you for your comments. Our responses are following:

The English language errors of manuscript were corrected one by one.

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# ESPS PEER-REVIEW REPORT

**Name of journal:** World Journal of Gastroenterology
**ESPS manuscript NO:** 31173
**Title:** Macrophage inflammatory protein-2 (MIP-2) as mediator of inflammation in acute liver injury
**Reviewer's code:** 03678235
**Reviewer's country:** United States
**Science editor:** Jing Yu
**Date sent for review:** 2016-11-03 11:13
**Date reviewed:** 2016-12-01 00:40

| CLASSIFICATION | LANGUAGE EVALUATION | SCIENTIFIC MISCONDUCT | CONCLUSION |
|---|---|---|---|
| [ Y] Grade A: Excellent | [ Y] Grade A: Priority publishing | Google Search: | [ ] Accept |
| [ ] Grade B: Very good | [ ] Grade B: Minor language polishing | [ ] The same title | [ Y] High priority for publication |
| [ ] Grade C: Good | [ ] Grade C: A great deal of language polishing | [ ] Duplicate publication | [ ] Rejection |
| [ ] Grade D: Fair | [ ] Grade D: Rejected | [ ] Plagiarism | [ ] Minor revision |
| [ ] Grade E: Poor | | [ ] No | [ ] Major revision |
| | | BPG Search: | |
| | | [ ] The same title | |
| | | [ ] Duplicate publication | |
| | | [ ] Plagiarism | |
| | | [ ] No | |

## COMMENTS TO AUTHORS

In this review article Qin et al., have provided a welll written account on the role of MIP-2 in the role of acute liver injury. The article is really well written and fit for publication. The only item that I would address is the quality of figure 1 that could be improved.

**Responses to comments**

Dear editor:

Thank you for your comments. We improved the figure 1.