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PEER-REVIEW REPORT

Name of journal: *World Journal of Gastroenterology*

Manuscript NO: 72700

Title: Activation of natural killer T cells contributes to Th1 bias in the murine liver after 14 days of ethinylestradiol exposure

Provenance and peer review: Invited Manuscript; Externally peer reviewed

Peer-review model: Single blind

Reviewer's code: 03252941
Position: Editorial Board
Academic degree: MD
Professional title: Doctor, Professor
Reviewer's Country/Territory: Japan
Author's Country/Territory: China

Manuscript submission date: 2021-10-26

Reviewer chosen by: AI Technique

Reviewer accepted review: 2021-11-16 23:28

Reviewer performed review: 2021-11-19 13:34

Review time: 2 Days and 14 Hours

Scientific quality	[ ] Grade A: Excellent [Y] Grade B: Very good [ ] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish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

Zou et al. investigated the role of invariant natural killer T (iNKT) cells in cholestatic liver injury caused by ethinylestradiol (EE) by using mouse model. They found that EE increased intrahepatic iNKT cells along with biased Th1 cytokine production. They also investigated the mechanism of iNKT cell-mediated cholestatic liver injury caused by EE in the mouse model. The experimental design is valid and its results are clear. The interpretation is appropriate and the text is easy to read. This is a well-done study. I list up some minor points. 1. Histopathological results: The meaning of pathological score is not clear. Please specify. In addition, the authors only describe portal/periportal inflammation, such as piecemeal necrosis and proliferation of pseudocholangiolar duct. How about lobular inflammation? By the way, “bile duct hyperplasia” that the authors say is not right. Correctly, it is proliferation of pseudocholangiolar duct. 2. Why does the fold change of control C57BL/6J in Fig. 3A and Fig. 5A, B, C not distribute around 1.0? 3. (p.16, ll.15-17) CD1d knockout mice (lacking NKT cells) exhibit enhanced (>4-fold) proinflammatory cytokine secretion and higher mRNA levels of TLR4 in a NAFLD model[44].: This should be “CD1d knockout mice (lacking NKT cells) exhibit enhanced (>4-fold) proinflammatory cytokine secretion and higher mRNA levels of TLR4 in kidney of a NAFLD model.” 4. LPS is not defined in the text.