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PEER-REVIEW REPORT

Name of journal: *World Journal of Gastroenterology*

Manuscript NO: 83909

Title: Mechanism of annexin A1/N-formylpeptide receptor regulation of macrophage function to inhibit hepatic stellate cell activation through Wnt/β-catenin pathway

Provenance and peer review: Unsolicited manuscript; Externally peer reviewed

Peer-review model: Single blind

Reviewer's code: 05345731
Position: Peer Reviewer
Academic degree: BSc, MD, MSc

Professional title: Doctor

Reviewer's Country/Territory: Kazakhstan

Author's Country/Territory: China

Manuscript submission date: 2023-02-15

Reviewer chosen by: AI Technique

Reviewer accepted review: 2023-02-22 01:16

Reviewer performed review: 2023-02-23 22:52

Review time: 1 Day and 21 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Scientific quality	[ ] Grade A: Excellent [ ] Grade B: Very good [Y] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish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) [Y] Accept (General priority) [ ] Minor revision [ ] Major revision [ ] Rejection
Re-review	[Y] Yes [ ] No
Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous
	Conflicts-of-Interest: [ ] Yes [Y] No

SPECIFIC COMMENTS TO AUTHORS

Based on the information presented in this abstract, it appears that the study is investigating the potential role of Annexin A1 (AnxA1) in liver fibrosis, which is a common complication of chronic liver disease. The abstract suggests that hepatic stellate cells (HSCs) are activated by cytokines released by Kupffer cells, leading to the deposition of extracellular matrix and the development of liver fibrosis. The idea of investigating the potential role of AnxA1 in liver fibrosis is interesting, as AnxA1 has been shown to have anti-inflammatory effects and to regulate various cellular processes. However, the study can benefit from less invasive methods as well as using bioinformatic tools can be preferable to animal experiments. In general, the abstract could benefit from additional information on the study design, such as the sample size and motivation for animal experiments. Additionally, it would be helpful to know the specific mechanisms through which AnxA1 may be involved in liver fibrosis and how this could be targeted therapeutically. Overall, while the abstract raises an interesting question, further details are needed to fully evaluate the significance and potential impact of the study and its applicability in human studies.

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Peer-review model: Single blind

Reviewer's code: 03257320
Position: Peer Reviewer
Academic degree: MD

Professional title: Lecturer

Reviewer's Country/Territory: Japan

Author's Country/Territory: China

Manuscript submission date: 2023-02-15

Reviewer chosen by: Geng-Long Liu

Reviewer accepted review: 2023-03-20 00:11

Reviewer performed review: 2023-03-24 06:08

Review time: 4 Days and 5 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 [ ] Grade B: Good [Y] Grade C: Fair [ ] Grade D: No novelty
Creativity or innovation of this manuscript	[ ] Grade A: Excellent [ ] Grade B: Good [Y] Grade C: Fair [ ] Grade D: No creativity or innovation

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Re-review	[Y] Yes [ ] No
Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous
	Conflicts-of-Interest: [ ] Yes [Y] No

SPECIFIC COMMENTS TO AUTHORS

1) General comments This article is focusing on the mechanism of annexin A1(AnxA1) in liver fibrosis induced by CCl4. The authors showed that CCL4-induced liver injury were more severe in AnxA1 KO mice than those in wild type mice. The authors also showed that AnxA1 attenuated CCL4-induced liver inflammation and fibrosis and furthermore, Wnt/β-catenin signaling pathway are involved in the mechanism of AnxA1 induced inhibition of hepatic fibrosis. Several point remains to be obscure and complicated, and the reviewer has a comments as follows. Major comment 1. The authors mentioned that compared with the control group, AnxA1 expression was significantly increased in the CCl4 model group, and was higher at week 8 than week 4, suggesting that AnxA1 was related to the development of liver fibrosis. In contrast, the authors showed that the lesions in the AnxA1 / group were more severe than in the wild-type mice. If AnxA1 can attenuate the progression of liver injury, the reviewer thinks that AnxA1 expression will be lower at week 8 than week 4. The authors should show the reason why AnxA1 was higher at week 8 than week 4, suggesting that AnxA1 was related to the development of liver fibrosis. Furthermore, it is unclear if all data were significantly

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different between CCL4 4W group and CCL4 8W group, and between wild type and AnxA1 KO mice. Please show more minutely differences between these groups. 2. The authors also mentioned that the collagen deposition in the liver of AnxA1 / mice was significantly aggravated by CCl4, and the degree of liver fibrosis was even more severe. However the authors did not show the degree of the collagen deposition in the liver by 8week CCl4 was more severe in AnxA1 / mice than those in wild type mice. Minor comment 1. Although the authors showed that representative histological findings in each groups, it was unclear whether the differences of inflammation and fibrosis between wild type groups and AnxA1 / mice.

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Provenance and peer review: Unsolicited manuscript; Externally peer reviewed

Peer-review model: Single blind

Reviewer's code: 05085435
Position: Peer Reviewer
Academic degree: MD

Professional title: Assistant Professor

Reviewer's Country/Territory: Egypt

Author's Country/Territory: China

Manuscript submission date: 2023-02-15

Reviewer chosen by: Geng-Long Liu

Reviewer accepted review: 2023-03-18 15:13

Reviewer performed review: 2023-03-26 22:22

Review time: 8 Days and 7 Hours

Scientific quality	[ ] Grade A: Excellent [Y] Grade B: Very good [ ] 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
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Re-review	[ ] Yes [Y] No
Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous
	Conflicts-of-Interest: [ ] Yes [Y] No

SPECIFIC COMMENTS TO AUTHORS

In this study the aim was to evaluate the effect and mechanism of action annexin A1 in liver fibrosis. CCl4 (20%) and Ac2-26 and Boc2 were injected intraperitoneally to induce liver fibrosis in wild-type mice/Anxa1 knockout mice and detect the expression of inflammatory factors, collagen deposition, and the role of the Wnt/β-catenin pathway in hepatic fibrosis. The authors concluded that AnxA1 inhibited liver fibrosis in mice, and its mechanism may be related to inhibition of HSC Wnt/β-catenin pathway activation by targeting formylpeptide receptors to regulate the function of Macrophage.