

PEER-REVIEW REPORT

Name of journal: *World Journal of Gastroenterology*

Manuscript NO: 70678

Title: Fibrinogen-like protein 2 deficiency inhibits virus-induced fulminant hepatitis through abrogating inflammatory macrophage activation

Provenance and peer review: Unsolicited manuscript; Externally peer reviewed

Peer-review model: Single blind

Reviewer's code: 02446498

Position: Peer Reviewer

Academic degree: MD, PhD

Professional title: Professor

Reviewer's Country/Territory: Japan

Author's Country/Territory: China

Manuscript submission date: 2021-08-18

Reviewer chosen by: AI Technique

Reviewer accepted review: 2021-09-15 01:35

Reviewer performed review: 2021-09-27 09:36

Review time: 12 Days and 8 Hours

Scientific quality	<input type="checkbox"/> Grade A: Excellent <input checked="" type="checkbox"/> Grade B: Very good <input type="checkbox"/> Grade C: Good <input type="checkbox"/> Grade D: Fair <input type="checkbox"/> Grade E: Do not publish
Language quality	<input checked="" type="checkbox"/> Grade A: Priority publishing <input type="checkbox"/> Grade B: Minor language polishing <input type="checkbox"/> Grade C: A great deal of language polishing <input type="checkbox"/> Grade D: Rejection
Conclusion	<input type="checkbox"/> Accept (High priority) <input type="checkbox"/> Accept (General priority) <input checked="" type="checkbox"/> Minor revision <input type="checkbox"/> Major revision <input type="checkbox"/> Rejection
Re-review	<input checked="" type="checkbox"/> Yes <input type="checkbox"/> No

Peer-reviewer statements	Peer-Review: [<input checked="" type="radio"/>] Anonymous [<input type="radio"/>] Onymous Conflicts-of-Interest: [<input type="radio"/>] Yes [<input checked="" type="radio"/>] No
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SPECIFIC COMMENTS TO AUTHORS

In this manuscript, the authors examined the role of macrophage Fgl2 in a mouse model of virus-induced fulminant hepatitis. Fgl2 expression was increased in Kupffer cells and MoMFs with proinflammatory phenotypes after viral infection. Fgl2 deletion in bone marrow cells attenuated virus-induced hepatitis, associated with decreased inflammation. In addition, Fgl2 deletion increased M2 phenotypes in macrophages. This is a well-written paper. I have minor comments. 1. The effect of Fgl2 deletion on antibacterial immunity was examined. How about that on antiviral immunity? Please examine the effect of Fgl2 deletion on cellular antiviral responses and viral loads. 2. The authors used a mouse model of fulminant hepatitis. Does Fgl2 deletion prolong mouse survival?