

## ESPS PEER-REVIEW REPORT

**Name of journal:** World Journal of Hepatology

**ESPS manuscript NO:** 16480

**Title:** Ethanol-induced hepatic autophagy: Friend or foe?

**Reviewer's code:** 02861252

**Reviewer's country:** Turkey

**Science editor:** Xue-Mei Gong

**Date sent for review:** 2015-01-20 14:11

**Date reviewed:** 2015-01-22 07:25

CLASSIFICATION	LANGUAGE EVALUATION	SCIENTIFIC MISCONDUCT	CONCLUSION
<input type="checkbox"/> Grade A: Excellent	<input checked="" type="checkbox"/> Grade A: Priority publishing	PubMed Search:	<input checked="" type="checkbox"/> Accept
<input checked="" type="checkbox"/> Grade B: Very good	<input type="checkbox"/> Grade B: Minor language polishing	<input type="checkbox"/> The same title	<input type="checkbox"/> High priority for publication
<input type="checkbox"/> Grade C: Good	<input type="checkbox"/> Grade C: A great deal of language polishing	<input type="checkbox"/> Duplicate publication	<input type="checkbox"/> Rejection
<input type="checkbox"/> Grade D: Fair	<input type="checkbox"/> Grade D: Rejected	<input checked="" type="checkbox"/> Plagiarism	<input type="checkbox"/> Minor revision
<input type="checkbox"/> Grade E: Poor		[ Y] No	<input type="checkbox"/> Major revision
		BPG Search:	
		<input type="checkbox"/> The same title	
		<input type="checkbox"/> Duplicate publication	
		<input type="checkbox"/> Plagiarism	
		[ Y] No	

## COMMENTS TO AUTHORS

Very interesting editorial manuscript. Thank you for this new information.

## ESPS PEER-REVIEW REPORT

**Name of journal:** World Journal of Hepatology

**ESPS manuscript NO:** 16480

**Title:** Ethanol-induced hepatic autophagy: Friend or foe?

**Reviewer's code:** 01407353

**Reviewer's country:** Italy

**Science editor:** Xue-Mei Gong

**Date sent for review:** 2015-01-20 14:11

**Date reviewed:** 2015-02-04 01:29

CLASSIFICATION	LANGUAGE EVALUATION	SCIENTIFIC MISCONDUCT	CONCLUSION
<input type="checkbox"/> Grade A: Excellent	<input checked="" type="checkbox"/> Grade A: Priority publishing	PubMed Search:	<input checked="" type="checkbox"/> Accept
<input checked="" type="checkbox"/> Grade B: Very good	<input type="checkbox"/> Grade B: Minor language polishing	<input type="checkbox"/> The same title	<input type="checkbox"/> High priority for publication
<input type="checkbox"/> Grade C: Good	<input type="checkbox"/> Grade C: A great deal of language polishing	<input type="checkbox"/> Duplicate publication	<input type="checkbox"/> Rejection
<input type="checkbox"/> Grade D: Fair	<input type="checkbox"/> Grade D: Rejected	<input checked="" type="checkbox"/> Plagiarism	<input type="checkbox"/> Minor revision
<input type="checkbox"/> Grade E: Poor		[ Y] No	<input type="checkbox"/> Major revision
		BPG Search:	
		<input type="checkbox"/> The same title	
		<input type="checkbox"/> Duplicate publication	
		<input type="checkbox"/> Plagiarism	
		[ Y] No	

## COMMENTS TO AUTHORS

I have no comments.

## ESPS PEER-REVIEW REPORT

**Name of journal:** World Journal of Hepatology

**ESPS manuscript NO:** 16480

**Title:** Ethanol-induced hepatic autophagy: Friend or foe?

**Reviewer's code:** 02748112

**Reviewer's country:** Netherlands

**Science editor:** Xue-Mei Gong

**Date sent for review:** 2015-01-20 14:11

**Date reviewed:** 2015-01-23 23:40

CLASSIFICATION	LANGUAGE EVALUATION	SCIENTIFIC MISCONDUCT	CONCLUSION
<input type="checkbox"/> Grade A: Excellent	<input type="checkbox"/> Grade A: Priority publishing	PubMed Search:	<input type="checkbox"/> Accept
<input type="checkbox"/> Grade B: Very good	<input type="checkbox"/> Grade B: Minor language polishing	<input type="checkbox"/> The same title	<input type="checkbox"/> High priority for publication
<input checked="" type="checkbox"/> Grade C: Good	<input type="checkbox"/> Grade C: A great deal of language polishing	<input type="checkbox"/> Duplicate publication	<input type="checkbox"/> Rejection
<input type="checkbox"/> Grade D: Fair	<input type="checkbox"/> Grade D: Rejected	<input type="checkbox"/> Plagiarism	<input type="checkbox"/> Minor revision
<input type="checkbox"/> Grade E: Poor		<input checked="" type="checkbox"/> No	<input type="checkbox"/> Major revision
		BPG Search:	
		<input type="checkbox"/> The same title	
		<input type="checkbox"/> Duplicate publication	
		<input type="checkbox"/> Plagiarism	
		<input checked="" type="checkbox"/> No	

## COMMENTS TO AUTHORS

The paper is well written and reports on an interesting mechanistic dichotomy in terms of the role of autophagy in AFLD. There are a few comments/suggestions I have for the authors. 1. The clarity of the manuscript could be improved if the authors were to add an introductory paragraph in which their aim/point is explicitly stated. Although the title is suggestive, the aim of the editorial should be better clarified. Moreover, including this paragraph will serve as a means to discern this letter from the recently published letter by the group on the same topic (J Hepatol 2013;59:398). 2. The sentence starting with "Ethanol-induced hepatocyte steatosis..." should initiate a new paragraph. 3. In the paragraph starting with "On the other hand,..." the term 'hepatic damage' following stellate cell activation is not the most suitable phrasing, as activated stellate cells do not damage hepatocytes per se but simply contribute to a fibrogenic milieu. I would use the term 'hepatopathology' or 'liver pathology.' 4. Although the dilemma surrounding the role of autophagy in KCs is well-explained, the title suggests that the letter addresses the liver as a whole in the context of the role of autophagy. So, in the end the reader is left with an unanswered question in terms of the AFLD-affected liver as a whole. Is it protective or destructive? 5. To better address point 4, I think it would be helpful to index



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the extent to which hepatocellular autophagy and stellate cell (SC) autophagy contribute to overall liver injury. In my opinion, autophagic signaling in SCs is relatively innocuous compared to that in hepatocytes, simply because the hepatocytes make up the bulk of the parenchyma and comprise the main functional element in the liver. Hence, the pro-survival signaling in hepatocytes predominates. 6. Lastly, I think that the speculation on EtOH-induced autophagy may be extended to liver-resident dendritic cells (DCs) and perhaps even endothelial cells (ECs). The DCs ameliorate the extent of pro-inflammatory KC signaling, whereas the ECs are a source of pro-inflammatory substances. The review by van Golen et al. (Free Radic Biol Med. 2012 Apr 15;52(8):1382-402) provides a good account of the cellular constituents that account for liver damage. Perhaps the authors could derive some inspiration from that review to expand the scope of their central message and drive novel research.

## ESPS PEER-REVIEW REPORT

**Name of journal:** World Journal of Hepatology

**ESPS manuscript NO:** 16480

**Title:** Ethanol-induced hepatic autophagy: Friend or foe?

**Reviewer's code:** 02992497

**Reviewer's country:** Japan

**Science editor:** Xue-Mei Gong

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**Date reviewed:** 2015-02-02 14:55

CLASSIFICATION	LANGUAGE EVALUATION	SCIENTIFIC MISCONDUCT	CONCLUSION
<input type="checkbox"/> Grade A: Excellent	<input checked="" type="checkbox"/> Grade A: Priority publishing	PubMed Search:	<input checked="" type="checkbox"/> Accept
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		[ Y] No	

## COMMENTS TO AUTHORS

The authors described the ethanol-induced hepatic autophagy, which is cytoprotective or accelerating hepatic damage, in hepatocytes, stellate cells, and Kupffer cells. This manuscript was well summarized.