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PEER-REVIEW REPORT

Name of journal: World Journal of Gastroenterology

Manuscript NO: 31467

Title: Naringenin prevents experimental liver fibrosis by blocking TGFβ-Smad3 and JNK-Smad3 pathways

Reviewer's code: 03576374

Reviewer's country: South Korea

Science editor: Ze-Mao Gong

Date sent for review: 2017-02-23

Date reviewed: 2017-02-24

CLASSIFICATION	LANGUAGE EVALUATION	SCIENTIFIC MISCONDUCT	CONCLUSION
<input type="checkbox"/> Grade A: Excellent	<input checked="" type="checkbox"/> Grade A: Priority publishing	Google 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		<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 by Hernández-Aquino et al investigated the hepato-protective and anti-fibrotic effects of naringenin (NAR) using a carbon-tetrachloride (CCl4)-induced liver fibrosis model. The authors found NAR protects liver functions in the CCl4-treated livers, and reduces levels of oxidative stress, fibrosis, and inflammation. This is an interesting paper which has implications for possible mechanisms underlying hepato-protective and anti-fibrotic effects of NAR. The manuscript was well written and the data support their claims. No further requests are required.



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PEER-REVIEW REPORT

Name of journal: World Journal of Gastroenterology

Manuscript NO: 31467

Title: Naringenin prevents experimental liver fibrosis by blocking TGF β -Smad3 and JNK-Smad3 pathways

Reviewer's code: 03656599

Reviewer's country: China

Science editor: Ze-Mao Gong

Date sent for review: 2017-02-23

Date reviewed: 2017-02-27

CLASSIFICATION	LANGUAGE EVALUATION	SCIENTIFIC MISCONDUCT	CONCLUSION
<input type="checkbox"/> Grade A: Excellent	<input checked="" type="checkbox"/> Grade A: Priority publishing	Google 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"/> No	<input type="checkbox"/> Minor revision
<input type="checkbox"/> Grade E: Poor		BPG Search:	<input type="checkbox"/> Major revision
		<input type="checkbox"/> The same title	
		<input type="checkbox"/> Duplicate publication	
		<input type="checkbox"/> Plagiarism	
		<input checked="" type="checkbox"/> No	

COMMENTS TO AUTHORS

In this study, authors have shown that the molecular mechanisms involved in the hepatoprotective effects of naringenin (NAR) on carbon tetrachloride (CCl₄) induced liver fibrosis. NAR prevents CCl₄ induced liver inflammation, necrosis and fibrosis, due to its antioxidant capacity as free radical inhibitor and by inhibiting the NF- κ B, TGF- β -Smad3 and JNK-Smad3 pathways. So NAR may be utilized in human fibrosis previous clinical and toxicological evaluation. Technically, this approach is very interesting. The experiment design is reasonable, the figures are clear and the evidence is sufficient. This article complies with published requirements of the magazine.