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ESPS PEER-REVIEW REPORT

Name of journal: World Journal of Gastroenterology
ESPS manuscript NO: 14561
Title: SGK1 inhibits cellular apoptosis and promotes proliferation via MEK/ERK/p53 pathway in Crohn's disease
Reviewer's code: 00069964
Reviewer's country: China
Science editor: Yuan Qi
Date sent for review: 2014-10-13 20:47
Date reviewed: 2014-10-29 09:15

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

COMMENTS TO AUTHORS

General comment: The paper entitled "SGK1 inhibits cellular apoptosis and promotes proliferation via MEK/ERK/p53 pathway in Crohn's disease" established a mouse model of CD and investigated its relationship with SGK1. This paper found that SGK1 expression was increased, silencing of SGK1 inhibited the phosphorylation of MEK1 and downstream molecular ERK1/2 which induced the up-regulation of p53 and Bax, which induced IECs' apoptosis and proliferation. In general, this paper is worth publishing in WJG. But it is better if the paper further address the following questions. 1) SGK1 is ubiquitously expressed in almost all tissues of digestive tract, such as esophagus, stomach, liver, intestine, and pancreas. This paper did not offer evidence of abnormal expression of SGK1 in Crohn's disease (CD), so its hypothesis that SGK1 inhibitors may be potentially therapeutics in the treatment of CD is not reasonable. Because this a model study, so it can not reflect the real CD. Thus, it is better to offer this evidence literally in the introduction or experimentally in CD samples. Otherwise, the results in this paper can not conclude that GK1 inhibits cellular apoptosis and promotes proliferation via MEK/ERK/p53 pathway in Crohn's disease 2) The figure2 and figure 4

showing apoptosis is not specific and the apoptosis cells can not be seem, it is better give another images. 3) This paper concluded that SGK1 inhibits cellular apoptosis and promotes proliferation via MEK/ERK/p53 pathway by interfering of SGK1, this is not a direct result. Thus it is better to give the direct results (for example, overexpression cause apoptosis and inhibition of proliferation) if consider acceptation. 4) This sentence "For further investigation, MEK1 inhibitor (U0126) was used to prove the MEK/ERK-dependent reaction" is not a result. Many English errors are found in the paper.

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ESPS PEER-REVIEW REPORT

Name of journal: World Journal of Gastroenterology
ESPS manuscript NO: 14561
Title: SGK1 inhibits cellular apoptosis and promotes proliferation via MEK/ERK/p53 pathway in Crohn's disease
Reviewer's code: 00058696
Reviewer's country: United States
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CLASSIFICATION	LANGUAGE EVALUATION	SCIENTIFIC MISCONDUCT	CONCLUSION
[ ] Grade A: Excellent	[ ] Grade A: Priority publishing	PubMed Search:	[ ] Accept
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		[ ] Plagiarism	
		[Y] No	

COMMENTS TO AUTHORS

We have carefully examined this new manuscript. Our major concerns are summarized below: 1) The title needs to be rewritten since the authors are not studying Crohn's disease. In fact, the authors have used the model BALB/c mice for their experimentation, with TNBS as the chemical to induce colitis. 2) The manuscript does have many grammatical mistakes, and many sentences need reframing. 3) A drawback of the study is that the authors have failed to discuss the possible confounding factors and limitations of the study in their discussion. 4) Should not the legend for figure 3 C, D be that SGK1 expressions at different times and doses on TNF treated IEC-6 cells (rather than HCT-116 cells)?