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ESPS Peer-review Report

Name of Journal: World Journal of Gastroenterology

ESPS Manuscript NO: 3857

Title: MicroRNA-155 regulates intestinal epithelial apical junctional complex in severe acute pancreatitis

Reviewer code: 00503609

Science editor: Wang, Jin-Lei

Date sent for review: 2013-05-29 18:35

Date reviewed: 2013-07-10 04:27

CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
<input type="checkbox"/> Grade A (Excellent)	<input type="checkbox"/> Grade A: Priority Publishing	Google Search:	<input type="checkbox"/> Accept
<input type="checkbox"/> Grade B (Very good)	<input type="checkbox"/> Grade B: minor language polishing	<input type="checkbox"/> Existed	<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"/> No records	<input type="checkbox"/> Rejection
<input type="checkbox"/> Grade D (Fair)	<input type="checkbox"/> Grade D: rejected	BPG Search:	<input type="checkbox"/> Minor revision
<input type="checkbox"/> Grade E (Poor)		<input type="checkbox"/> Existed	<input type="checkbox"/> Major revision
		<input type="checkbox"/> No records	

COMMENTS TO AUTHORS

Rui Tian and co-workers present a study examining how MicroRNA-155 (miR-155) regulates epithelial apical junctional complex in severe acute pancreatitis. The study was carried out in a caerulein/LPS-induced mouse model of acute pancreatitis. Samples were collected 3 hours after the last injection. The authors demonstrated evidence of severe acute pancreatitis. miR-155 expression increased compared to controls and was associated with a decrease in ZO-1, E-cadherin, and RhoA expression. The authors conclude that these results suggest that severe acute pancreatitis decreases intestinal barrier function by decreasing synthesis key junctional complex proteins via mi-R-155 signaling. Critique 1. The question that arises is whether the findings are specific for severe acute pancreatitis? The model uses two substances to induce severe pancreatitis. Does either of these substances directly induce the findings in intestine? Did you perform controls in which cerulean alone and LPS alone are given without induction of pancreatitis? Is there evidence that pancreatitis caused by other mechanisms result in these changes? 2. It is difficult to understand how the mi-R-155 target genes were predicted using miRTarBase, RNA22, and PicTar. This explanation needs to be expanded. 3. The intestine when examined showed necrosis of cells and shedding of mucosal cells in the intestine of the pancreatitis mice. How does this loss of cells translate in measurement of the proteins in question? Was it down regulated or lost during intestinal damage? 4. Minor: The manuscript is easy to understand, but the abstract has abbreviations that are not defined. For example, the abbreviated term for MicroRNA-155 should be placed in parenthesis after it is first mentioned. Also, it would be best to define the measured proteins in the abstract as those important for intestinal barrier function. The manuscript and study is straight forward and, except for the



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prediction of target genes, easy to follow. Like all pancreatitis models, there is question about the correlation of the findings in the specific pancreatitis model with pancreatitis in humans. In this case, one must determine if the effects studied in the mice were due to severe acute pancreatitis or the agents used to induce it. The findings are interesting, but what is the significance of these findings to the prevention or treatment of pancreatitis? ?



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ESPS Peer-review Report

Name of Journal: World Journal of Gastroenterology

ESPS Manuscript NO: 3857

Title: MicroRNA-155 regulates intestinal epithelial apical junctional complex in severe acute pancreatitis

Reviewer code: 00058401

Science editor: Wang, Jin-Lei

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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
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<input type="checkbox"/> Grade B (Very good)	<input type="checkbox"/> Grade B: minor language polishing	<input type="checkbox"/> Existed	<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"/> No records	<input type="checkbox"/> Rejection
<input type="checkbox"/> Grade D (Fair)		BPG Search:	<input type="checkbox"/> Minor revision
<input type="checkbox"/> Grade E (Poor)	<input type="checkbox"/> Grade D: rejected	<input type="checkbox"/> Existed	<input type="checkbox"/> Major revision
		<input type="checkbox"/> No records	

COMMENTS TO AUTHORS

To the authors: Congratulations for the excellent work-liberatocaboclo@gmail.com



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ESPS Peer-review Report

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ESPS Manuscript NO: 3857

Title: MicroRNA-155 regulates intestinal epithelial apical junctional complex in severe acute pancreatitis

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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
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<input type="checkbox"/> Grade B (Very good)	<input type="checkbox"/> Grade B: minor language polishing	<input type="checkbox"/> Existed	<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"/> No records	<input type="checkbox"/> Rejection
<input type="checkbox"/> Grade D (Fair)		BPG Search:	<input type="checkbox"/> Minor revision
<input type="checkbox"/> Grade E (Poor)	<input type="checkbox"/> Grade D: rejected	<input type="checkbox"/> Existed	<input type="checkbox"/> Major revision
		<input type="checkbox"/> No records	

COMMENTS TO AUTHORS

ESPS Manuscript NO: 3857 MicroRNA-155 regulates intestinal epithelial apical junctional complex in severe acute pancreatitis It is well designed study with new interesting findings. There are few typing errors. The close correlation between intestinal barrier dysfunction and poor prognosis of SAP is well known. The authors reported interesting points 'for the first time': 1) The contribution of miR-155 in intestinal barrier dysfunction in SAP and the participation of TNF- α in the early inflammatory responses in SAP with AJC structure damage via miR-155 pathway. 2) miR-155 acts on the target gene of RhoA and inhibits protein synthesis of ZO-1 and E-cadherin, which are the major components for TJs and AJs, respectively. 3) TNF- α -miR-155-RhoA pathway may contribute on to a better understanding of etiology and the mechanisms of intestinal barrier dysfunction in SAP