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ESPS Peer-review Report
Name of Journal: World Journal of Gastroenterology
ESPS Manuscript NO: 4106
Title: Deletion of Gpr128 results in weight loss and increased intestinal contraction frequency
Reviewer code: 00038529
Science editor: Gou, Su-Xin
Date sent for review: 2013-06-14 16:49
Date reviewed: 2013-06-27 10:16

CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[ ] Grade A (Excellent)	[ ] Grade A: Priority Publishing	Google Search:	[ ] Accept
[ ] Grade B (Very good)	[Y] Grade B: minor language polishing	[ ] Existed	[ ] High priority for publication
[Y] Grade C (Good)	[ ] Grade C: a great deal of language polishing	[ ] No records	
[ ] Grade D (Fair)		BPG Search:	[ ]Rejection
[ ] Grade E (Poor)	[ ] Grade D: rejected	[ ] Existed	[ ] Minor revision
		[ ] No records	[Y] Major revision

COMMENTS TO AUTHORS

This paper (Manuscript Number:4106) shows deletion of Gpr128 in mice results in weight loss and increased intestinal contraction frequency. The authors attempt to demonstrate the relationship between weight loss and intestinal motility. However, they only enumerate bits of information and they did not do the important part of experiments. Although they mentioned that further studies with regard to the relationship between peristalsis and nerve plexus, slow waves and ICC should be conducted in the next paper and I also expect those studies in the next paper, the authors should identify the intracellular localization of epithelial cells of intestine at least. Furthermore, it is better to conduct time course study of intestinal motility, because if the weight loss is the effect of hyper motility, intestinal motility must be observed at not only 32 weeks but also from around 20~24 weeks. Major Comments This paper shows deletion of Gpr128 in mice results in weight loss and increased intestinal contraction frequency. Although Gpr128 is one of the Adhesion-GPCR families, it may be mainly expressed on the basolateral membrane of epithelial cells of intestine. However, as with the cases of GPR40, which is the receptor for fatty acid and GPR116, these are also GPCR and expressed on the apical membrane of gut epithelial cells and alveolar epithelial cells, respectively, the authors should confirm the intracellular localization of Gpr128. I hope this result facilitates their research. Furthermore, the authors examined intestinal motility study at only 32 weeks. It is better to do time course change of motility study, if the authors would mention the relationship between weight loss and hyper motility of intestine. Minor Comments The author used primer R1 (5′-GATTCCAACTTCATTACTCTG-3′) and primer R2 (5′-GGTCCATATCTGCC CACTG-3′), however I could not find the result using these primers. Did the authors do the nested PCR? The authors used terms R1, R2, P1, P2,….N1, N2. I think odd numbers such as R1, P1 and N1 indicate

forward primers and even numbers R2, P2 and N2 indicate reverse primers. I was confused that which primer was for forward, and which one was for reverse. It may be better to describe the details in the sentence. For example, forward primer (or sense primer) R1,…reverse primer (or anti sense primer) N2.

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ESPS Peer-review Report
Name of Journal: World Journal of Gastroenterology
ESPS Manuscript NO: 4106
Title: Deletion of Gpr128 results in weight loss and increased intestinal contraction frequency
Reviewer code: 02276563
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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[ ] Grade A (Excellent)	[ ] Grade A: Priority Publishing	Google Search:	[ ] Accept
[Y] Grade B (Very good)	[Y] Grade B: minor language polishing	[ ] Existed	[ ] High priority for publication
[ ] Grade C (Good)	[ ] Grade C: a great deal of language polishing	[ ] No records	
[ ] Grade D (Fair)		BPG Search:	[ ]Rejection
[ ] Grade E (Poor)	[ ] Grade D: rejected	[ ] Existed	[Y] Minor revision
		[ ] No records	[ ] Major revision

COMMENTS TO AUTHORS

In a manuscript entitled "Deletion of Gpr128 results in weight loss and increased intestinal contraction frequency" authors Ying-Yin Ni et al. generated a Gpr128 gene knockout mouse model and investigated its phenotypes and biological function. Overall, this study fits nicely within the scope of the journal. The data are generally clean and could potentially uncover the physiological roles of Gpr128, which is of value to the field. This reviewer has only minor comments. 1. It would be helpful to provide data showing intracellular localization of Gpr128, which could help to explain the potential roles of Gpr128. 2. Data shown in Fig. 3D indicated that there were no differences in the epididymal and uterine fat pads, brown fat, liver, heart, spleen, lung, and kidney weights between the Gpr128+/+ and Gpr128-/- mice. However, in Figure 3 legend, it stated that organs were weighed and correlated to body weight. Could this correction eliminate the difference in organ weight between the Gpr128+/+ and Gpr128-/- mice? 3. The interesting results described beg the question concerning the mechanism(s) underlying Gpr128 knockout mouse phenotypes. It is clearly follow-up work. If the authors wish, please comment on the possible mechanism(s). 4. Figure 1D in Page 7 should be Figure 2D. Figure 1A, Figure 1D and 1E in page 15 should be Figure 2A, 2D and 2E.

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ESPS Peer-review Report
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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[ ] Grade A (Excellent)	[ ] Grade A: Priority Publishing	Google Search:	[ ] Accept
[ ] Grade B (Very good)	[Y] Grade B: minor language polishing	[ ] Existed	[ ] High priority for publication
[ ] Grade C (Good)	[ ] Grade C: a great deal of language polishing	[ ] No records	[Y]Rejection
[ ] Grade D (Fair)	[ ] Grade D: rejected	BPG Search:	[ ] Minor revision
[Y] Grade E (Poor)		[ ] Existed	[ ] Major revision
		[ ] No records	

COMMENTS TO AUTHORS

1. In the abstract, I suggest that the authors should reduce the discussion of methods because in the body, authors said the same thing. 2. Also, the authors reduce the sentences of results in the abstract. 3. The background and aim in the abstract should be written more clearly and explained better. 4. Authors should send a clear copy of the manuscript. Not one filled with corrections and suggestions. 5. Authors described the importance of Gpr128 in introduction, but it is not clear why authors need to study the role of Gpr128 in the intestine. I suggest that authors should explain clearly in the aim of the study in introduction. 6. I think the study design of this manuscript is not enough, and authors need to study the importance of Gpr128 on digestive tissues. 7. I suggest that authors need to add their thoughts in the discussion and conclusion, not only repeat the results.