



## PEER-REVIEW REPORT

**Name of journal:** World Journal of Gastroenterology

**Manuscript NO:** 48566

**Title:** Towards a standard diet-induced and biopsy-confirmed mouse model of non-alcoholic steatohepatitis: Impact of dietary fat source

**Reviewer's code:** 03647890

**Reviewer's country:** China

**Science editor:** Jia-Ping Yan

**Reviewer accepted review:** 2019-04-26 02:11

**Reviewer performed review:** 2019-04-28 01:25

**Review time:** 1 Day and 23 Hours

SCIENTIFIC QUALITY	LANGUAGE QUALITY	CONCLUSION	PEER-REVIEWER STATEMENTS
<input type="checkbox"/> Grade A: Excellent	<input type="checkbox"/> Grade A: Priority publishing	<input type="checkbox"/> Accept	Peer-Review:
<input type="checkbox"/> Grade B: Very good	<input type="checkbox"/> Grade B: Minor language	(High priority)	<input type="checkbox"/> Anonymous
<input type="checkbox"/> Grade C: Good	polishing	<input type="checkbox"/> Accept	<input type="checkbox"/> Onymous
<input type="checkbox"/> Grade D: Fair	<input type="checkbox"/> Grade C: A great deal of	(General priority)	Peer-reviewer's expertise on the
<input type="checkbox"/> Grade E: Do not	language polishing	<input type="checkbox"/> Minor revision	topic of the manuscript:
publish	<input type="checkbox"/> Grade D: Rejection	<input type="checkbox"/> Major revision	<input type="checkbox"/> Advanced
		<input type="checkbox"/> Rejection	<input type="checkbox"/> General
			<input type="checkbox"/> No expertise
			Conflicts-of-Interest:
			<input type="checkbox"/> Yes
			<input type="checkbox"/> No

### SPECIFIC COMMENTS TO AUTHORS

The authors try to design a new formula to establish animal model of non-alcoholic steatohepatitis in the manuscript. Authors analyze several critical the histopathologic characteristics by IHC and biochemical markers among of new model and referenced



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models. This model is very important in NASH studies. In this manuscript, the data could support authors's conclusion, and the new model could be comparable to both models of ob/ob and trans-fat feeding. The manuscript orgnization is good.

#### INITIAL REVIEW OF THE MANUSCRIPT

##### *Google Search:*

- The same title
- Duplicate publication
- Plagiarism
- No

##### *BPG Search:*

- The same title
- Duplicate publication
- Plagiarism
- No



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**Manuscript NO:** 48566

**Title:** Towards a standard diet-induced and biopsy-confirmed mouse model of non-alcoholic steatohepatitis: Impact of dietary fat source

**Reviewer's code:** 03699961

**Reviewer's country:** Japan

**Science editor:** Jia-Ping Yan

**Reviewer accepted review:** 2019-04-26 15:09

**Reviewer performed review:** 2019-05-07 01:01

**Review time:** 10 Days and 9 Hours

SCIENTIFIC QUALITY	LANGUAGE QUALITY	CONCLUSION	PEER-REVIEWER STATEMENTS
<input type="checkbox"/> Grade A: Excellent	<input type="checkbox"/> Grade A: Priority publishing	<input type="checkbox"/> Accept	Peer-Review:
<input type="checkbox"/> Grade B: Very good	<input checked="" type="checkbox"/> Grade B: Minor language	(High priority)	<input checked="" type="checkbox"/> Anonymous
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			<input checked="" type="checkbox"/> No

### SPECIFIC COMMENTS TO AUTHORS

**Title:** Towards a standard diet-induced and biopsy-confirmed mouse model of non-alcoholic steatohepatitis: Impact of dietary fat source Michelle L. Boland, Denise Oró, Kirstine S. Tølbøl, et al. 1) General Comments In this manuscript, the authors



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reported a benefit of mice that fed a high-fat/fructose diet, in which trans-fat was substituted by palm oil, as a nonalcoholic fatty liver disease (NAFLD) model. In addition to the histological confirmation of fat deposit, active inflammation, and fiber accumulation in the liver, characteristic gene expression profiles and gut bacterial taxonomic shifts were presented in association with the disease development. The strategy is straightforward, and the results are clear. An unfocused presentation, however, diminishes the value of this study. The following are concerns that the authors may wish to consider:

2) Specific comments Major concerns: 1. Although the authors presented the data of gene expression profiles and gut bacterial taxonomic shifts, there are not insightful interpretation nor discussion. If these data were included in this manuscript, the authors should thoroughly evaluate the results and provide the data in a comprehensive way. 2. The authors describe in the introduction that a glucose intolerance is a hallmark of NAFLD. Although there is a clear difference in terms of a glucose intolerance between ob/ob mice that fed AMLN and GAN diets, histopathological findings of the liver are similar between these two groups. Please explain the reasons why the metabolic difference did not lead to either a phenotypic difference or gene expression profile in the liver.? 3. I believe that the aim of this study is to emphasize the benefit of a GAN diet in a NAFLD animal model comparing with an original AMLN diet. In this context, many aspects should be compared between two diets in the same genetical background. However, the authors performed several comparisons such as the clusters of transcriptomes between ob/ob mice and C57 controls. If this article focuses on the effect of different diets to promote NAFLD, the control against a GAN diet should be an original AMLN diet in mice with the same background. Minor concerns: 1. Do not use an abbreviation such as AMLN from the beginning.



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