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ESPS PEER-REVIEW REPORT

Name of journal: World Journal of Hepatology
ESPS manuscript NO: 25637
Title: CD36 genetic variation, fat intake and liver fibrosis in chronic hepatitis C virus infection
Reviewer's code: 03536815
Reviewer's country: Italy
Science editor: Jing Yu
Date sent for review: 2016-03-31 08:20
Date reviewed: 2016-05-18 03:29

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

COMMENTS TO AUTHORS

To the Authors I read with great interest the manuscript titled "CD36 genetic variation, fat intake and liver fibrosis in chronic HCV infection" By Omar Ramos-Lopez et al. It explores the extremely interesting relationship between liver fibrosis, evaluated through non-invasive ultrasound based technique, and genetics. Authors aimed to determine the association of the CD36 (rs1761667) polymorphism with dietary intake and liver fibrosis in patients of West Mexico with chronic hepatitis C (CHC). Patients with the CD36 AA genotype seemed to have a higher intake of calories, total fat and saturated fatty acids (SFA) than those with the AG and GG genotypes. Moreover patients with advanced liver fibrosis had a higher frequency of the CD36 AA genotype than those with mild liver fibrosis (42.1% vs. 17.1%, p=0.002), respectively. And authors concluded that likewise, the AA genotype is associated with advanced liver fibrosis. The paper is fluent and well witten, the results clearly explained and the conclusions convincing. I have only minor concerns about it: - Please add a brief mention of the limitation of the use of three-day food record in the dietary assessment. - According to journal guidelines the conclusion must include the contributions of the conclusions to

the field and the weaknesses of the study, and provide future research directions.

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ESPS PEER-REVIEW REPORT

Name of journal: World Journal of Hepatology
ESPS manuscript NO: 25637
Title: CD36 genetic variation, fat intake and liver fibrosis in chronic hepatitis C virus infection
Reviewer's code: 02860848
Reviewer's country: Germany
Science editor: Jing Yu
Date sent for review: 2016-03-31 08:20
Date reviewed: 2016-04-03 01:12

CLASSIFICATION	LANGUAGE EVALUATION	SCIENTIFIC MISCONDUCT	CONCLUSION
[ ] Grade A: Excellent	[ ] Grade A: Priority publishing	Google Search:	[ ] Accept
[ ] Grade B: Very good	[Y] Grade B: Minor language polishing	[ ] The same title	[ ] High priority for publication
[ ] Grade C: Good	[ ] Grade C: A great deal of language polishing	[ ] Duplicate publication	[ ] Rejection
[Y] 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

In the current manuscript, the authors Ramos-Lopez et al. suggest that CD36 genetic variation (therefore presumably CD36 expression is different) correlates with amount of fat intake and liver fibrogenesis. Although much more data are definitely needed for convincingly showing that elevated amount of fat intake, due to CD36 genetic variation, enhances liver fibrogenesis in patients with chronic HCV, the current study is potentially interesting. ---------- Major concern:---------- 1. Method for dietary assessment is absolutely not clear. In a previous publication form authors (Ramos-Lopez et al., Nutrition and Food Sciences 2015) dietary assessment method is not visibly mentioned. Since the data about fat intake is a key point in the presented manuscript, the authors should provide more detailed information (e.g. as a supplementary information). Which guideline do authors follow?---------- 2. There is a huge difference on fat intake between AA and GG genotypes, but CH and protein intake (%) is nearly identical. What do patients with GG genotype eat less?---------- 3. Authors suggest that increased fat intake in patients with AA genotype show no significant difference concerning glucose, TC, TG, HDL, LDL, etc. but only AST is significantly higher in AA. It

is hard to understand how elevated fat intake for long time (presented data are from 3-day analysis but authors consider this is representative of whole time food intake behavior) does not have any metabolic parameters or obesity and only AST? Do authors have biopsy materials? Steatosis? Inflammation? Minor concern: 1. How did the authors identify exact duration of infection for each patient?---------- 2. Do patients have HCC?---------- 3. Data for ALT and AST are similar, but there is huge difference concerning significance.