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PEER-REVIEW REPORT

Name of journal: World Journal of Gastroenterology

Manuscript NO: 44541

Title: Anti-tumor necrosis factor α therapy associates to type 17 helper T lymphocytes immunological shift and significant microbial changes in dextran sodium sulphate colitis

Reviewer's code: 01557050

Reviewer's country: Japan

Science editor: Ruo-Yu Ma

Date sent for review: 2018-12-21

Date reviewed: 2018-12-29

Review time: 2 Hours, 8 Days

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[ ] Grade E: Do not publish		[ ] Rejection	[ ] Advanced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

SPECIFIC COMMENTS TO AUTHORS

1) General comments Dr. Petito and Lopetuso, et al. investigated 'Anti-TNFα therapy associates to Th17 immunological shift and significant microbial changes in dextran sodium sulphate colitis. The article is informative and well-presented. The reviewer has

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some comments. Comments 1) The reviewer could not understand well about T0 and T2. Please describe the definition of T0 and T2 in Methods.

INITIAL REVIEW OF THE MANUSCRIPT

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PEER-REVIEW REPORT

Name of journal: World Journal of Gastroenterology

Manuscript NO: 44541

Title: Anti-tumor necrosis factor α therapy associates to type 17 helper T lymphocytes immunological shift and significant microbial changes in dextran sodium sulphate colitis

Reviewer's code: 04091933

Reviewer's country: Russia

Science editor: Ruo-Yu Ma

Date sent for review: 2018-12-27

Date reviewed: 2018-12-31

Review time: 19 Hours, 4 Days

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

SPECIFIC COMMENTS TO AUTHORS

This is indeed a high-quality study that demonstrates the viability of murine DSS-induced colitis model for studying the effects of anti-TNF-α (Infliximab, IFX) on the immune system and gut microbiota. The authors have previously shown that IFX

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showed a good affinity both for human-TNF-α and murine-TNF-α, and it ameliorated the severity of DSS colitis in mice [Lopetuso L.R. et al., 2013]. The new study clearly demonstrated that the anti-inflammatory effect of IFX is associated with an increase in Th17 pathway, along with gut microbiota alteration. An important clinical consequence, if it will be confirmed in humans, is the identification of a potential dysbiotic effect of IFX. This anti-TNF-α-induced dysbiosis should be taken into consideration, and not only in non-responders. Perhaps, some IBD patients with dysbiosis on anti-TNF-α will require therapeutic modulation of gut microbiota. Furthermore, anti-TNF-α-induced shift toward Th17 pathway could be considered when deciding to switch/change therapy in non-responders. The limitations are described in detail by the authors and do not affect the quality of the article. In my opinion, the results of this study will lead to a significant breakthrough in future clinically important studies of the interaction between biological therapy, the immune system and gut microbiota. In turn, mouse models will help develop effective methods for the therapeutic correction of dysbiotic microbiota in IBD.

INITIAL REVIEW OF THE MANUSCRIPT

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