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PEER-REVIEW REPORT

Name of journal: World Journal of Stem Cells

Manuscript NO: 47161

Title: Ameliorating liver fibrosis in an animal model using the secretome released from miR-122-transfected adipose-derived stem cells

Reviewer's code: 02566952

Reviewer's country: Romania

Science editor: Ying Dou

Reviewer accepted review: 2019-06-06 13:47

Reviewer performed review: 2019-06-06 14:53

Review time: 1 Hour

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[ ] Grade A: Excellent [Y] Grade B: Very good [ ] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish	[ ] Grade A: Priority publishing [Y] Grade B: Minor language polishing [ ] Grade C: A great deal of language polishing [ ] Grade D: Rejection	[ ] Accept (High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

SPECIFIC COMMENTS TO AUTHORS

Interesting in vitro and small animal study reporting about the potential interest in using transfected ADSC secretome as a modality to inhibit liver fibrosis. Below are point by point comments Abstract and running title Do the authors consider a more specific

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terminology can be used instead of secretome? Conditioned media? Microvesicles? The manuscript seem to remain elusive in this respect failing ot inform exactly what the authors have used for their study. Introduction I would argue the veridicity of the first phrase. I am not convinced stem cell research is the most promising branch of biomedicine (what is biomedicine by the way, do authors mean biomedical research?) Stem cell research might be promising for researchers but from the clinician s and patient perspective it has not delivered so far too much compared with nano and advanced material science which is providing increasingly performant implants, Aside of couple of approved therapies (one counts on the digits from one hand) the large majority (if we exclude hematopoetic stem cell transplantation) for otherwise untreatable diseases we do not have stem cells in the clinic as of 2019 . One of the reason is indeed highlighted by the authors themselves when they try to argument the use of cell free therapies. I don t think they are miRNAs responsible for liver fibrosis rather involved in one way or another in the process. Material and methods Please revise description of chondrogenetic assays. It is not clear how the cells were cultured (normally a mention about some form of high density culture should be there, if it was not the case please explain) Results In figure A what is the significance of “Mock” are they ADSCs transfected with vector only? In this case a control with non transfected ADSCs should have been added for comparison The subchapter “Determination of the antifibrotic effects of the secretome released from miR-122-transfected ASCs in an in vitro model of liver fibrosis: remains esoteric as there is no description of how this has been performed. On what kind of samples and using what methods. Please resolve this as it is important to understand what kind of secretome the author are referring to. Is it the conditioned media? Have the microvesicles have been extracted or not. This important aspect in the context of this paper should be very clearly described. Same remark about the affirmation “we treated LX2 cells” how were the cells treated (methods,

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doses, timing) and most of all exactly what were they treated with (conditioned media? MV?) For the histological evaluation of rat liver fibrosis how was the collagen content quantitatively determined? Figure 3 and 4 legend inform the graphs below the pictures show the relsitve density of the markers. How was this assessed quantitatively? Discussion chapter is well written. Resulting arguments collected from the study seem to supporting the use of transfected ADSCs and antifibrotic agents with improved potential compared to native ADSCs.

INITIAL REVIEW OF THE MANUSCRIPT

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