

## ESPS Peer-review Report

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

**Ms:** 3031

**Title:** Human platelets inhibit liver fibrosis in SCID mice

**Reviewer code:** 00005388

**Science editor:** j.l.wang@wjgnet.com

**Date sent for review:** 2013-04-04 23:40

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

## COMMENTS

### COMMENTS TO AUTHORS:

The work described in this manuscript show the effects of human platelet transfusion in SCID mouse models of liver fibrosis. The antifibrotic effect of platelet was supported by a significant decrease in the amount of fibrosis area and in the liver hydroxyproline content. Accordingly, the amount of TGF-beta and HGF in platelet-treated mice are significantly diminished and reduced, respectively. While these data are in accordance with the antifibrotic effects of platelet previously reported in the literature, many data of this study need to be confirmed. The authors argued for an increase of hepatocyte apoptosis in treated mice but there are no statistical analyses for TUNEL staining, MMP9 and BCL2 expression and the amount of cleaved caspase 3 might be compared to the non cleaved form. In these figures, the quality of western blot pictures needs to be improved. The increase of p-MET and p-SMAD3 are convincing but the quantification of several independent experiments is required. The discussion is interesting but quite too speculative about the direct implication of Kupffer cells which are not formally characterized in this study. An important concern is the absence of significant difference in the sensitive markers of hepatic dysfunctions that include serum AST and ALT. How the authors explain changes in the fibrotic index without changes of these parameters?

## ESPS Peer-review Report

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

**Ms:** 3031

**Title:** Human platelets inhibit liver fibrosis in SCID mice

**Reviewer code:** 00054597

**Science editor:** j.l.wang@wjgnet.com

**Date sent for review:** 2013-04-04 23:40

**Date reviewed:** 2013-04-30 04:44

CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
<input type="checkbox"/> Grade A (Excellent)	<input type="checkbox"/> Grade A: Priority Publishing	Google Search:	<input type="checkbox"/> Accept
<input type="checkbox"/> Grade B (Very good)	<input checked="" type="checkbox"/> Grade B: minor language polishing	<input type="checkbox"/> Existed	<input type="checkbox"/> High priority for publication
<input checked="" type="checkbox"/> Grade C (Good)	<input type="checkbox"/> Grade C: a great deal of language polishing	<input type="checkbox"/> No records	<input type="checkbox"/> Rejection
<input type="checkbox"/> Grade D (Fair)		BPG Search:	<input type="checkbox"/> Minor revision
<input type="checkbox"/> Grade E (Poor)	<input type="checkbox"/> Grade D: rejected	<input type="checkbox"/> Existed	<input type="checkbox"/> Major revision
		<input type="checkbox"/> No records	

## COMMENTS

### COMMENTS TO AUTHORS:

In this manuscript by Takahashi K et al, the authors speculate the protective role of platelets in inhibiting liver fibrosis. The authors determine that platelet transfusion results in increased HGF and MMP9 with decreased TGF $\beta$  that result in decreased liver fibrosis. There are some concerns which need clarification: 1. Platelets themselves are an important source of HGF. Hence, it is unclear why the authors speculate a role for kupffer cells as the major producers of HGF. This needs to be explained considering that the authors demonstrate increased platelet concentrations in the liver. 2. MMP-9 would result in increased inflammation and extracellular matrix turnover. Thus suggest that this may lead increased fibrosis. Also previous reports have in fact associated MMP-9 with liver fibrosis. Hence the author's findings needs explanation in this context since they note increased MMP levels with decreased fibrosis.