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### ESPS Peer-review Report

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

**Ms:** 1727

**Title:** Resistin mediates the hepatic stellate cell phenotype

**Reviewer code:** 00000507

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

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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 checked="" 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 type="checkbox"/> Grade C (Good)	<input type="checkbox"/> Grade C: a great deal of language polishing	<input checked="" 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 checked="" type="checkbox"/> No records	

### COMMENTS

#### COMMENTS TO AUTHORS:

This study by Dong et al. reported the effects of the adipokine resistin on the biology of hepatic stellate cells and Kupffer cells. In rats subjected to bile duct ligation, resistin expression was increased in adipose tissue but not in the liver. Exposure of HSc to resistin increased expression of IL-6 and MCP-1 and mediated proliferation, migration and survival. Moreover, in KC resistin increased TGF-beta that in turn elicited profibrogenic action in HSC. The Authors conclude that resistin directly and indirectly induces profibrogenic actions in HSC. GENERAL COMMENTS The study is well conducted and the experiments carefully executed. Some of the data are not entirely new, but as a whole the paper reinforces the notion that resistin may be profibrogenic. SPECIFIC COMMENTS 1. The data on increase in cell migration, with or without anti-MCP-1 antibodies, should be confirmed in Boyden chamber experiments. 2. Figure 4C-4D shows activation of the NF-kappa B pathway but no information on the functional role of this pathway is provided. Additional experiments should be performed to assess which of the observed biologic actions depend on NF-kappaB activation in HSC or KC. 3. Figure 5 adds little to the paper and should be removed, describing the data in the text. 4. It is surprising that TGF-beta released by KC did not result in further expression in HSC. Please comment. 5. Figure 7 is very poor and should be redrawn. 6. Possible mechanisms underlying the increase in adipose tissue (and not liver) resistin after BDL. 7. Fig. 1B: please correct the legend to the y axis 8. There are several typos and syntax errors that should be corrected.