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E-mail: bpgoffice@wjgnet.com http://www.wjgnet.com

ESPS PEER-REVIEW REPORT

Name of journal: World Journal of Gastroenterology
ESPS manuscript NO: 24250
Title: Expression of B7-H4 and HBx in HBV-related hepatocellular carcinoma
Reviewer's code: 00013065
Reviewer's country: Germany
Science editor: Ze-Mao Gong
Date sent for review: 2016-01-16 18:00
Date reviewed: 2016-01-20 23:50

CLASSIFICATION	LANGUAGE EVALUATION	SCIENTIFIC MISCONDUCT	CONCLUSION
[ ] Grade A: Excellent	[ ] Grade A: Priority publishing	Google Search:	[ ] Accept
[Y] 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
[ ] 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

Dr. Hong and colleagues have presented an interesting study in which they aimed to investigate the expression and clinical significance in terms of HCC-development of B7-H4 in dependency of HBx in cell culture and HBV-positive patient samples. The colleagues found that B7-H4 up-regulation in HBx-positive samples were correlated with hepato-carcinogenesis in cell culture and patients. The authors conclude that B7-H4 may be involved in facilitating HBV-related HCC-development. Overall, the manuscript is well written and concise in its content and the methodical design is well performed. However, there are some comments which should be addressed. 1) There are some minor spelling and typing errors which should be polished, e.g., in the Abstract, Conclusion first sentence it should read "was" correlated instead of "were". 2) Abstract section, Results; the correct abbreviation for the hepatoma cell line is HepG2.2.15 3) Material and Methods, Cell lines; the HepG2.2.15 cells are stably HBV-expressing hepatoma cells derived from HepG2 cells and were firstly described by Sells et a 1987 and 1988 (PNAS 84:1005-1009 and J Virol 62:2836-2844). 4) The Introduction section is informative but much too long and reads like a review article. Please shorten it and focus on the essentials. 5) Table 1 and 2. There are some cryptic signs for Tumor grade and TNM stage. This

might be due to the word file; however, I can't read it. 6) Fig. 1a; In my opinion and as a suggestion the density data should be presented as a graph rather than a table. 7) Fig. 1 legend; the last sentence of the figure legend is incomplete **, vs HepG2 cells should read HepG2.2.15 vs HepG2 8) Fig.2; it is hard to identify the intracellular localisation of B7-H4 due to the minor quality of the figures. Please enhance the quality of the figures by, e.g., enhancing the magnification. 9) Fig. 3. The staining of HBx and also B7-H4 is not really convincing. Please present figures with higher magnification showing clearly B7-H4 and HBx expression and staining. 10) There are two recent reports on B7-H1 expression showing that HBeAg supresses the cellular immunity by Han et al (Cell Immunol 2013 283:25-30) and especially of Xu et al (Clin Immunol 2010 136:30-41) showing that B7-H4 expression attenuates con-A induced hepatic injury. These reports could be discussed in the context of the findings of the authors.

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ESPS PEER-REVIEW REPORT

Name of journal: World Journal of Gastroenterology
ESPS manuscript NO: 24250
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Reviewer's country: South Korea
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CLASSIFICATION	LANGUAGE EVALUATION	SCIENTIFIC MISCONDUCT	CONCLUSION
[ ] Grade A: Excellent	[ ] Grade A: Priority publishing	Google Search:	[ ] Accept
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[ ] Grade E: Poor		[Y] No	[ ] Major revision
		BPG Search:	
		[ ] The same title	
		[ ] Duplicate publication	
		[ ] Plagiarism	
		[Y] No	

COMMENTS TO AUTHORS

The authors reported that the elevated expression of B7-H2, known as a co-stimulatory molecule, was detected not only HepG2.215 cell lines (transfected with HBV DNA) but also HBV-related hepatocellular carcinoma clinical tissues in this manuscript. Although they showed a positive correlation between HBx and B7-H4 expression in hepatocellular carcinoma of HBV positive patients, they provide a mere phenomenon. They did not show a role of B7-H4 in this manuscript. Even though they showed a role of B7-H4 in pancreatic carcinoma cells through their previous study, we would like to urge roles of B7-H4 in hepatic cancel cell. For instance, During B7-H4 suppression with its siRNA in HepG2.215 cells, roles of B7-H4 including cell to cell interaction, cell growth, and apoptosis are examined.