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PEER-REVIEW REPORT

Name of journal: World Journal of Gastroenterology

Manuscript NO: 46577

Title: Role and mechanism of circ-PRKCI in hepatocellular carcinoma

Reviewer's code: 03668592

Reviewer's country: Italy

Science editor: Ruo-Yu Ma

Reviewer accepted review: 2019-03-01 10:25

Reviewer performed review: 2019-03-01 11:32

Review time: 1 Hour

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[ ] Grade E: Do not publish		[ ] Rejection	[ ] Advanced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

The manuscript of "The role and mechanism of circ-PRKCI in hepatocellular carcinoma" by Qi et al try to explore the role and mechanism of circ-PRKCI in HCC, it's very interesting and useful. It may be as a new biomarker for diagnosis of HCC or other digestive system tumors. This manuscript can be accepted after some minor revision: 1

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the authors test many digestive system tumor cells, but just present some results in HCC, are there obvious difference? Why? 2 Please improve image resolution. 3 Some grammar problems need to be solved.

INITIAL REVIEW OF THE MANUSCRIPT

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PEER-REVIEW REPORT

Name of journal: World Journal of Gastroenterology

Manuscript NO: 46577

Title: Role and mechanism of circ-PRKCI in hepatocellular carcinoma

Reviewer's code: 02937396

Reviewer's country: Japan

Science editor: Ruo-Yu Ma

Reviewer accepted review: 2019-02-21 01:32

Reviewer performed review: 2019-03-05 04:00

Review time: 12 Days and 2 Hours

SCIENTIFIC QUALITY	LANGUAGE QUALITY	CONCLUSION	PEER-REVIEWER STATEMENTS
[ ] Grade A: Excellent	[ ] Grade A: Priority publishing	[ ] Accept (High priority)	Peer-Review:
[ ] Grade B: Very good	[Y] Grade B: Minor language polishing	[ ] Accept (General priority)	[Y] Anonymous
[ ] Grade C: Good	[ ] Grade C: A great deal of language polishing	[ ] Minor revision	[ ] Onymous
[Y] Grade D: Fair	[ ] Grade D: Rejection	[Y] 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

The author showed the role and mechanism of circ-PRKCI and E2E7 in HCC. The work is well written and interested. However, I think the authors should consider several revisions. Major #1. In general, lymph nodes metastases of HCC is not so much. I think these were so much in this study. Is there any relation between lymph nodes metastases

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and circ-PRKCI/E2E7 ? #2. The authors should perform MTT-assay by using circ-PRKCI knock out or down, and over expression. #3. Regarding Figure 3C, the authors showed representable photos about the apoptosis. I recommend that the authors should digitize and compare by statistics (ex: apoptotic index, etc…). #4. Regarding Figure 5, the authors should state the "reference value" of High or Low. The authors also should show relapse-free survival. Does "Survival" mean only cancer related death or overall ? It should be stated. #5. In Table 2, I recommend the authors had better to add portal venous invasion or hepatic venous invasion. Minor #1. I think the explanation about circ-PRKCI is insufficient. I recommend more detail explanation about circ-PRKCI in "Introduction".

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PEER-REVIEW REPORT

Name of journal: World Journal of Gastroenterology

Manuscript NO: 46577

Title: Role and mechanism of circ-PRKCI in hepatocellular carcinoma

Reviewer's code: 00003880

Reviewer's country: Japan

Science editor: Ruo-Yu Ma

Reviewer accepted review: 2019-02-28 22:36

Reviewer performed review: 2019-03-09 14:51

Review time: 8 Days and 16 Hours

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

The study by Qi and colleagues investigates the role of a large class of endogenous non-coding RNA, circ-PRKCI in the development of digestive cancer. Firstly, they focused on the expression of circ-PRKCI in cancer specimens. Higher expression of circ-PRKCI was found in cancerous and cancer tissues compared with adjacent tissue by

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qRT-PCR, which was most remarkable in liver tumor. Circ-PRKCI was closely associated with growth, apoptosis and invasion of hepatocellular carcinoma cell lines, which may be mediated by AKT3 activity. Moreover, the expression of Circ-PRKCI was significantly related with survival of patients with hepatocellular carcinoma, suggesting that Circ-PRKCI is suitable for a prognostic marker of gastric cancer. The experiments are properly conducted and clearly presented, but I regret to inform you that your manuscript could not be considered for publication in its present form. My comments are as follows. Major comments; 1. The main criticism is the lack of specificity in this paper. It is already demonstrated that circ-PRKCI was highly expressed in esophageal squamous cell carcinoma compared with paired adjacent normal tissues and upregulation of circ-PRKCI enhanced cell proliferation and migration of ECC cells (Shi N, et al. J Cell Biochem. 2019 Jan 18). Moreover, circ-PRKCI, as a molecular sponge of miR-3680-3p, mediates the expression of AKT. Thus, this paper shows similar results as already demonstrated data. I strongly recommend the authors emphasize that your work is novel because of the use of hepatocellular carcinoma cells and it is relevant to biology and therapy of this type of malignancy. 2. The authors mentioned circ-PRKCI silencing cell showed low levels of invasion in hepatocellular carcinoma cells. The possibility cannot be denied that this effect is induced by the inhibition of cell growth, because knockdown of circ-PRKCI expression significantly impaired cellular proliferation in the text. To address this, authors should attempt to isolate factors involving in cancer invasion accelerated by circ-PRKCI, by doing additional experiments. 3. The authors concentrated on a cell line of hepatocellular carcinoma. I think that the authors need to look at another kind of cell lines such as gastric and/or esophageal carcinoma. This would help to eliminate any bias in a particular cell in terms of technical procedure. 4. The AKT signal transduction should be further explored in the article. There is no rationale for picking only AKT for analysis. How about other pivotal

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pathways that have been described as crucial for in this phase of cell growth and invasion? 5. The authors showed association between clinicopathological characteristics and circ-PRKCI expression in hepatocellular carcinoma in Discussion section. It would be highly preferable to show the summarized table. 6. In Materials and methods Section on page 6, lines 17-18 in the manuscript, more detailed information of cell lines, such as origin species and histology, should be mentioned with references. 7. At the end of the Discussion, before the general conclusion reported, (page 11, line27-page 12, line 2), the authors should provide the specific conclusions of the work referring to the data obtained. Now these specific conclusions are lacking at the end of the paper. Minor comments; 1. Page 5 line6: Appropriate references should be shown to explain non-coding RNAs (ncRNAs) are involved in the pathogenesis of digestive system tumors. 2. There are a number of wrong PMID in Reference section of this article.

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