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PEER-REVIEW REPORT

Name of journal: World Journal of Gastroenterology

Manuscript NO: 56608

Title: Dual targeting PLK1 and BIRC5 in TP53-mutated hepatocellular carcinoma

Reviewer's code: 00504882
Position: Editorial Board
Academic degree: PhD
Professional title: Professor
Reviewer's Country/Territory: United States
Author's Country/Territory: China

Manuscript submission date: 2020-05-07

Reviewer chosen by: AI Technique

Reviewer accepted review: 2020-05-07 14:21

Reviewer performed review: 2020-05-18 15:30

Review time: 11 Days and 1 Hour

Scientific quality	[ ] Grade A: Excellent [Y] Grade B: Very good [ ] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish
Language quality	[ ] Grade A: Priority publishing [Y] Grade B: Minor language polishing [ ] Grade C: A great deal of language polishing [ ] Grade D: Rejection
Conclusion	[ ] Accept (High priority) [ ] Accept (General priority) [Y] Minor revision [ ] Major revision [ ] Rejection
Re-review	[Y] Yes [ ] No
Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous Conflicts-of-Interest: [ ] Yes [Y] No

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SPECIFIC COMMENTS TO AUTHORS

The title of the manuscript is appropriate to the subject matter discussed in the paper. The authors have used the TCGA HCC dataset to find out the expression level of PLK1 and BIRC5 in a total of 374 HCC data sets. Authors found that both PKL1 and BRIC5 overexpressed in a subset of HCC data set. They discovered that co-expression of these proteins is frequently detected in HCC with p53 mutation, which also correlated with poor clinical outcome. They went on to use inhibitors of either PKL1 or BRIC5 to target Huh7 carrying Y220C mutation effectively, but HepG2 cell with WTp53 was not affected. Specific comments: 1. What is the expression status of these proteins in HCC tumors having wild type p53? 2. HuH7 carries a p53 mutation. The mutant p53 is not inactive in HuH7 cells (http://dx.doi.org/10.1128/JVI.00729-15). 3. Do these proteins have an inhibitory effect on p53 function? Inhibition of either of these proteins may activate p53 function and induce apoptosis. 4. Protein-protein interaction between these protein and wild type p53 should be done using immunoprecipitation and Western blotting. 5. The status of other proteins such as FuBP1 which is overexpressed in 80 % of HCC tumors with CHC background, and strongly inhibits p53 function.