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PEER-REVIEW REPORT

Name of journal: *World Journal of Gastrointestinal Oncology*

Manuscript NO: 75248

Title: Dissecting novel mechanisms of hepatitis B virus related hepatocellular carcinoma using meta-analysis of public data

Provenance and peer review: Unsolicited manuscript; Externally peer reviewed

Peer-review model: Single blind

Reviewer's code: 02936529
Position: Editorial Board
Academic degree: FRCS (Hon), MD, PhD
Professional title: Professor, Surgical Oncologist
Reviewer's Country/Territory: Brazil
Author's Country/Territory: United States

Manuscript submission date: 2022-01-20

Reviewer chosen by: AI Technique

Reviewer accepted review: 2022-01-20 12:32

Reviewer performed review: 2022-01-23 01:02

Review time: 2 Days and 12 Hours

Scientific quality	[Y] Grade A: Excellent [ ] Grade B: Very good [ ] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish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

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Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous Conflicts-of-Interest: [ ] Yes [Y] No

SPECIFIC COMMENTS TO AUTHORS

The authors explored specific oncogenic gene expression pathways hepatitis B-related HCC. The authors employed Search, Tag, Analyze, Resource platform to conduct a meta-analysis of public data from NCBI's Gene Expression Omnibus (meta-analysis of 155 tumor versus 185 adjacent non-tumor samples) with Ingenuity Pathway Analysis. Results of metanalysis: - LXR/RXR activation and FXR/RXR activation as top canonical pathways amongst others; - Top upstream regulators identified included the Ras family gene RABL6; - RABL6 mediates pathogenesis of HBV-related HCC through promotion of genes related to cell division, epigenetic regulation, and Akt signaling; - Survival analysis demonstrated increased mortality with higher RABL6 expression; - HOXA10 was a top upstream regulator and was strongly upregulated; HOXA10 has recently been demonstrated to contribute to HCC pathogenesis in vitro. Our causal analysis suggests an in vivo role through downregulation of tumor suppressors and other mechanisms. The present study has impecable methodology with illustrative figures, and a very concise discussion of the possible implications of RABL6 and HOXA10 in the carcinogenic pathways for hepatitis B-related HCC and the possible future possibilites of their role as therapeutic targets.

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Reviewer's code: 03647881
Position: Editorial Board
Academic degree: MD, PhD
Professional title: Associate Professor, Attending Doctor
Reviewer's Country/Territory: Taiwan
Author's Country/Territory: United States

Manuscript submission date: 2022-01-20

Reviewer chosen by: AI Technique

Reviewer accepted review: 2022-01-28 12:22

Reviewer performed review: 2022-02-03 00:30

Review time: 5 Days and 12 Hours

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

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Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous Conflicts-of-Interest: [ ] Yes [Y] No

SPECIFIC COMMENTS TO AUTHORS

No comments.