Reviewer #1:
Scientific Quality: Grade B (Very good)
Language Quality: Grade A (Priority publishing)
Conclusion: Accept (General priority)
Specific Comments to Authors: No comments.

We thank you for your feedback and time reviewing our manuscript.

Reviewer #2:
Scientific Quality: Grade A (Excellent)
Language Quality: Grade A (Priority publishing)
Conclusion: Accept (High priority)
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.

We thank you for your positive feedback and your time in reviewing our manuscript.