Reviewer's report

Title: Epidermal growth factor induces HCCR expression via PI3K/Akt/mTOR signaling in PANC-1 pancreatic cancer cells

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Reviewer: Young Jung

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[Major Compulsory Revision]

The author suggested that EGF upregulates HCCR via Akt and mTOR signaling. The data showing in this manuscript for Akt signaling are very convincing. However, they used only one chemical inhibitor (rapamycin) for mTOR signaling. To prove the involvement of the mTOR signaling, some molecular approach (dominant-negative or overexpressed constructs / siRNA) are needed.

[Minor Essential Revisions]

1. In the section of “Materials and methods”, the origin of the Akt construct and siRNA targeted HCCR are addressed to be gifted other scientists. In that case, it should be cited the previous paper describing the detailed methods. Or they are developed in this lab, the detailed methods are missed.

2. Fig 2 B, C, D, E: a control for un-stimulated cell is missing and should be included in these figures in order to see the solely effect of the siRNA and overexpressed HCCR.

3. The author should discuss the particular reasons for using PANC-1 cells in this manuscript rather than other pancreatic cancer cells.

Level of interest: An article of importance in its field

Quality of written English: Acceptable

Statistical review: No, the manuscript does not need to be seen by a statistician.

Declaration of competing interests:

I declare that I have no competing interests