SPECIFIC COMMENTS TO AUTHORS

In this manuscript titled "Aryl Hydrocarbon Receptor Dynamics in ESCC: Unmasking the Immune Evasion Strategies," the authors review the multiple roles of aryl hydrocarbon receptors in esophageal squamous cell carcinoma. The manuscript is well finished, but I think it needs the following revisions:

1. The authors should revise the title of the manuscript. In fact, the authors' review of the role of AhR in ESCC is not limited to immune escape. Authors may consider broadening the scope of their title description to better attract potential readers.

2. The author mentioned AhR agonists and anti-tumor effects. Take, for example, indole-3-carbinol, which the authors describe as a modulator in the manuscript. But indole-3-carbinol should be an AhR agonist. Meanwhile, the main development direction described in the manuscript is AhR antagonists. I think readers may be confused whether the two have the same effect?

3. The description of dietary therapy may need to be more rigorous. The important reason is that there are too few studies on the metabolic kinetics of permeability and transport, which results in the possible limited role of dietary therapy.

Point-by-point response to Reviewer #1:

We would like to express our sincere gratitude for your helpful comments and constructive feedback, which have significantly contributed to the improvement of our manuscript.

1. Thank you for your valuable feedback regarding the manuscript title. We appreciate your suggestion to broaden the scope of the title of our review beyond immune escape. In response to your comments, we have revised the title. The revised title is "Aryl Hydrocarbon Receptor dynamics in ESCC: From immune modulation to therapeutic opportunities".

2. Thank you for your comment regarding the distinction between AhR agonists and antagonists in our manuscript. We acknowledge the importance of clarifying this

distinction to avoid confusion, especially regarding indole-3-carbinol (I3C). In response to your feedback, we have carefully revised the manuscript to differentiate between AhR agonists and antagonists more clearly. Specifically, where appropriate, we have used terms such as "ligand" or "agonist" to specify the role of AhR activators like I3C, which we highlighted for their potential anticancer effects. We have also expanded on why certain AhR agonists, including I3C, may exhibit anticancer properties.

3. Thank you for your feedback regarding the description of dietary therapy in our manuscript. We acknowledge the importance of a more rigorous discussion on the limitations of dietary AhR modulators. In response to your comment, we have added the following text to the article:

"However, the effectiveness of dietary AhR modulators is not well-established by the lack of comprehensive studies on the metabolic kinetics, permeability, and transport of dietary compounds. Future research should focus on these aspects to better understand and optimize the role of these compounds in ESCC treatment."

These changes have been made and highlighted in the text accordingly.