Review of: "FGF8 induces chemokinesis and regulates condensation of mouse nephron progenitor cells"

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NPC condensation is essential for nephron formation, but underlying regulation mechanisms remain elusive. Previous studies have demonstrated the important role of Fgf8 for the survival of NPCs. In this manuscript, Sharma et al reveal a novel function of Fgf8. By using mouse models, quantitative imaging assays, and data-driven computational modeling, they demonstrated the crucial role of Fgf8 signaling for the coordination of NPCs behaviors to the UB, especially for NPC condensation.

Generally speaking, the manuscript was well organized and written. The experiments and analysis were well done. However, I have following concerns:

1. Fig 3, I don't understand why the control lost Six2 expression (Fig 3A). Fig 3E is not consistent withFig 3d, which showed that the treatment of Fgf8 antibody significantly decreases the number of live cells. Lastly, the 3D culture matrix experiments did not provide evidence on the role of Fgf8 for NPC condensation.

2. At the end of "A model based on Fgf8-induced motility leads to robust condensation of NPC" part, there is not a conclusive sentence.

3. Whole kidney qPCR results are not enough to support the claim of incomplete deletion of Fgf8 in mouse models. Protein staining or mRNA detection in section is required to support the claim. In addition, clear explanation is required on how the phenotypes of Fgf8 KO mice are associated the function of Fgf8 for NPC condensation.

4. I can not understand the last sentence well "Further work is required to reveal how Fgf8 along with its receptors and inhibiting factors orchestrates NPC condensation, its ....."

It is novel to study the role of Fgf8 for NPC condensation as a chemokine.