Preventing M2-Polarized Macrophages in a HIF2-Dependent Manner

Nghia Hoang[1,2], Jasper R. Chen[2], and Cullen M. Taniguchi MD-PhD[2]
[1]Franklin & Marshall College, Lancaster, PA, USA
[2]Department of Radiation Oncology, The University of Texas MD Anderson Cancer Center, Houston, TX, USA

THE UNIVERSITY OF TEXAS MD Anderson Cancer Center
Making Cancer History®

Background/Introduction

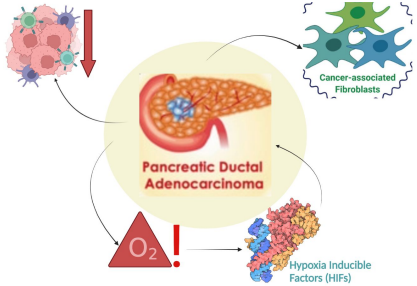
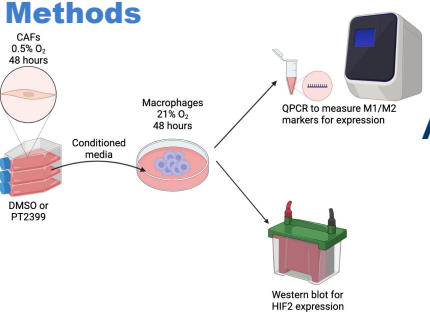


Figure 1. Characteristics of Pancreatic Ductal Adenocarcinoma (PDAC).

Methods



Graphical Abstract

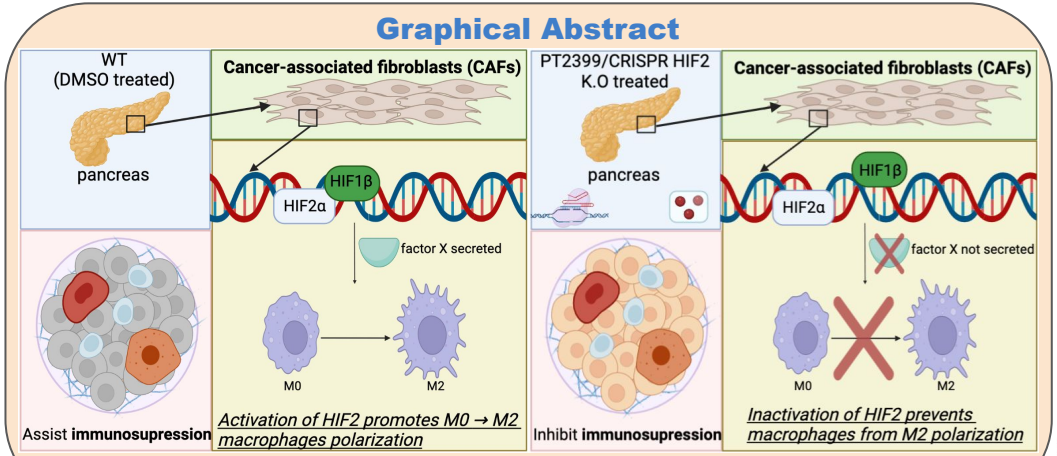


Figure 2a. Schematic diagram outlining the wild-type CAFs control.

Figure 2b. Schematic diagram outlining the experimented CAFs subjected to the treatment of PT2399, a HIF2 transcription factor inhibitor.

Results

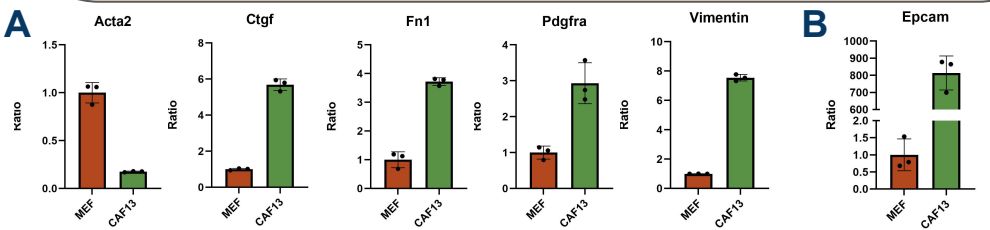


Figure 3. Characterization of Mouse Embryonic Fibroblast (MEF) and CAF13 cell lines via RT-quantitative PCR of 5 fibroblast **(A)** and 2 epithelial **(B)** marker genes. Data was normalized by 18s reference gene.

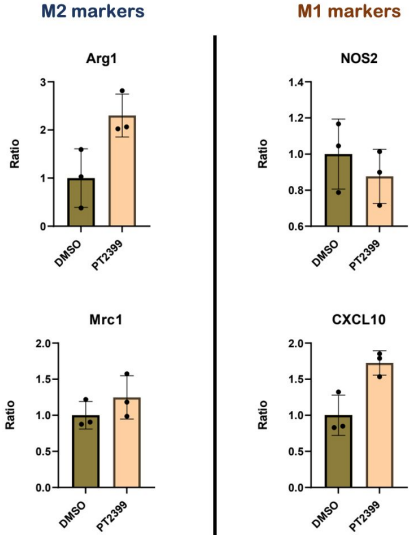


Figure 4. Characterization of macrophages in conditioned media via RT-quantitative PCR using 4 M1/M2 biomarker genes. Conditioned media from CAF13 cells were either treated with DMSO or PT2399. Data was normalized by 18s reference gene.

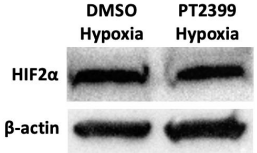


Figure 5. Western blot analysis of HIF2α antibodies with hypoxic CAF lysates, treated with either DMSO (WT) or PT2399.

Conclusions/Future Directions

- Knocking out HIF2 using PT2399 inhibitor prevents M0 macrophages from polarizing to M2, reducing immunosuppressive effects.
- FD: Identification of factor X by means of mass spectrometry.

Acknowledgements

I would like to give special thanks to Dr. Taniguchi and Jasper Chen for welcoming me into the lab, providing me with great guidance throughout the project, and for being such kind and helpful mentors.

References

1. Taniguchi, Cullen M. et al. *Gastroenterology* (**2022**).
2. Huang, Yanqing et al. *Science China. Life sciences* (**2017**).