





MASTER THESIS PROJECT:

Unraveling the role of PHGDH in normal and pathogenic T cell immunity

About us

In the Immunobiology Lab we are interested in understanding how metabolism regulates T cell responses during autoimmune diseases and antitumor immunity. Our aim is to identify how metabolic pathways regulate post-transcriptional and epigenetic programs directing T cell fate and differentiation, in order to manipulate these pathways in autoimmune diseases and antitumor immunity.

Project outline

We recently found that phosphoglycerate dehydrogenase (PHGDH), the key enzyme of the serine synthesis pathway, also functions as an RNA-binding protein (RBP), and interacts with RNAs that are critical for stemness, cell fate, viability and mitochondrial homeostasis. In this project, we will explore how the RNAbinding activity and catalytic activity of PHGDH orchestrate normal and pathogenic T cell-mediated immunity using in vitro and in vivo models of T cell differentiation and function, in combination with cutting edge -omics approaches.

We offer

- Dynamic environment that encourages networking, learning and the development of your own ideas.
- Opportunity to work and learn with highly qualified experts in the fields of immunology and metabolism.
- You will gain experience in flow cytometry, microscopy, cell culture, cutting-edge techniques and more.

Your profile

- We are looking for a very curious and motivated Master's student with excellent communication skills and interest in immunology and/or metabolism.
- The ideal candidate should want to learn to work independently and have the initiative to develop a research project under the guidance of a junior researcher fellow, Dr. Francesc Baixauli, and the Principal Investigator, Prof Christoph Hess.

How to apply

Please send us an updated CV, including previous lab experience and a motivation letter to francesc.baixauli@unibas.ch and christoph.hess@unibas.ch.

References

- Lotscher, J. et al. Magnesium sensing via LFA-1 regulates CD8(+) T cell effector function. Cell 185, 585-602 e529 (2022). https://doi.org:10.1016/j.cell.2021.12.039
- Baixauli, F. et al. An LKB1-mitochondria axis controls TH17 effector function. Nature (2022). https://doi.org:10.1038/s41586-022-05264-1