Proposal of Master Research Project / Proyecto fin de Master for the academic year 2025-26

Group name: Control molecular de la mielinización axonal
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Group web:

Title of the MRP/TFM: The Immune Role of OPCs: Modulating inflammation and remyelination in the adult CNS

Summary of the Project:

Myelin is essential for neuroprotection and adequate central nervous system (CNS) function. In different neurodegenerative diseases (e.g. Multiple Sclerosis), myelin is lost, leading to axonal degeneration. Oligodendrocyte Progenitor Cells (OPCs) are the main drivers of myelin repair in the CNS. Myelin regeneration efficiency declines with chronic inflammation, highlighting the important interplay between inflammation and OPCs. OPCs are essential for CNS homeostasis and their functions in the adult CNS go beyond myelin repair. Recent studies have shown that OPCs are not only recipients of immune signals, but also active agents acquiring an immune role and developing a disease-associated state in inflammatory contexts. Thus, our work focuses on understanding the dynamics of this disease-associated OPC state and how OPCs may modulate inflammatory responses in the CNS.

This project aims to investigate whether OPCs promote or limit CNS inflammation in the healthy and inflamed CNS. To do so, we will combine an *in vivo* OPCs depletion model paired acute inflammation and address the following objectives:

A) Investigate if OPCs regulate immune cell infiltration in the CNS
B) Determine if OPC depletion changes CNS inflammatory profile
C) Study whether OPC depletion changes the response of other CNS resident cells (microglia, astrocytes) to acute inflammation

This project will explore novel OPC-mediated immune functions and investigate the recently described interplay between OPCs and inflammation. In the long-term, identifying OPC immune modulatory functions will open new therapeutic approaches to limit CNS inflammation and enhance myelin repair.

Methods and technology involved in the MRP/TFM Project:

Animal Models: We will use an acute model of systemic inflammation that induces CNS inflammation transiently in C57BL6/J transgenic mice in which we can deplete OPCs.

Histology: We will perform histology analysis (cryosection, immunostaining and fluorescent microscopy) to investigate changes in the CNS in our model

Molecular biology: RT-qPCR will be used to determine changes in inflammatory cytokines and chemokines in the CNS.

Member/s of the lab who will act as tutor/co-tutor of the project (if different from the group IP; PhD required to be tutor / co-tutor):

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