ALMA MATER STUDIORUM
UNIVERSITÀ DI BOLOGNA

DIPARTIMENTO
DI FARMACIA
E BIOTECNOLOGIE

AVVISO DI SEMINARIO

Il giorno **5 dicembre 2025**
alle ore **15.00**

Dr. Luca Masin

Ricercatore PostDoc, Department of Biology, Animal Physiology and Neurobiology Section, KU Leuven, Leuven Brain Institute, Lovanio, Belgio
(ospite del Prof. C. Bergamini)

terrà un seminario in lingua inglese dal titolo:

Local glycolysis as an intrinsic and evolutionarily conserved feature of axonal regeneration

Area tematica: Neuroscience, Biochemistry

in presenza:
AULA B Anatomia, Via Irnerio 48, Bologna

Colleghi e studenti sono cordialmente invitati

ABSTRACT

The adult mammalian central nervous system (CNS) has a very limited regenerative capacity. Axonal regrowth following injury is an extremely costly process that requires an effective allocation of energy. Injury has been reported to depolarize local mitochondria in axons, and mitochondria are predominantly stationary in the adult mammalian CNS. While their remobilization is a viable strategy to sustain axonal regeneration, how axons can withstand the initial disruption in energy production caused by the injury, and subsequently initiate regrowth, is poorly understood. We uncovered that retinal ganglion cells co-deleted for Pten and Socs3 promptly undergo a switch in the expression of metabolism-related genes towards glycolysis after optic nerve crush injury. Using in vitro microfluidic cultures, we confirmed that injury-induced axonal regeneration is associated with mitochondrial transport within the axons and that this is enhanced after injury upon deletion of Pten and Socs3. Interestingly, we found that co-deleted neurons upregulate glycolysis locally in the distal axonal compartment immediately after axotomy. RNAseq data of sorted RGCs from the spontaneously-regenerating adult zebrafish, show a comparable upregulation of glycolytic genes after optic nerve crush injury. Together, these observations reveal that glycolysis, combined with sustained mitochondrial transport, is a core evolutionarily-conserved mechanism essential for injury-induced axonal regrowth. The manipulation of glycolysis could provide new therapeutic avenues to unlock the regeneration potential of the mammalian CNS.

BIOGRAPHICAL SKETCH

Luca Masin graduated in Pharmaceutical Chemistry and Technology at the University of Bologna in 2018, with a master thesis under the supervision of Prof. Romana Fato and Prof. Christian Bergamini. He then went to pursue a PhD at KU Leuven under the supervision of Prof. Lieve Moons. During his PhD, he developed an in vitro microfluidic culture of retinal murine neurons, which was used to the study of injury-induced axonal regeneration and of the molecular and metabolic players underlying it, with a particular focus on the ones involved in mitochondrial trafficking and ATP production. As a postdoc, Luca expanded his research areas, focusing on evolutionarily conserved features underlying axonal regeneration in both murine and fish models.