

SEMINAR CYCLE

of the PhD in Neuroscience of Turin

2nd Appointment

Prof. Benedikt Berninger

King's College London, UK and
University Medical Center Mainz, Germany

“Engineering neurogenesis in the postnatal cerebral cortex by lineage reprogramming”

31th January, 2025 h 2:00 PM

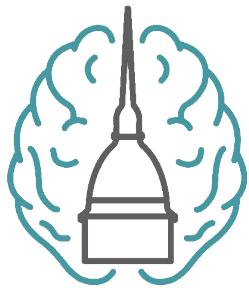
The lecture will last 1 hour and it will be followed by discussion

Host: Prof. Federico Luzzati

Seminar Room, NICO, Ospedale San Luigi Gonzaga, Regione
gonzole 10, Orbassano (TO)

Link: unito.webex.com/meet/federico.luzzati

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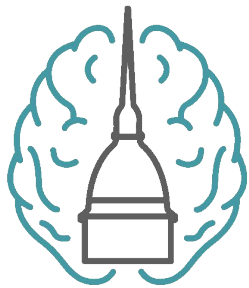


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PROF. BERNINGER

Benedikt Berninger is Professor of Developmental Neurobiology at the Centre for Developmental Neurobiology at King's College London, UK and at University Medical Center Mainz, Germany. His lab's research primarily focuses on how to re-ignite neurogenesis in the mammalian brain after developmental neurogenesis has come to an end. They do this by converting glia into induced neurons, using in vitro and in vivo virus-based models. They also study adult neural stem cells to learn how newly generated neurons integrate within existing networks.

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ABSTRACT

Lineage reprogramming of glia into neurons emerges as an experimental strategy to regenerate neurons lost to disease. We explore the possibility of using proneural transcription factors and mutant variants thereof to convert cortical glia (astrocytes and oligodendrocyte progenitor cells) into induced neurons with subtype specific properties. For example, we could show that forced expression of a phospho-deficient form of achaete-scute complex like 1 (Ascl1), referred to as Ascl1SA6, together with the cell death regulator Bcl2 can promote the conversion of early postnatal astrocytes into induced neurons that feature hallmarks of fast-spiking parvalbumin-expression interneurons (Marichal et al., 2024). Here, I will discuss how much we have learned about the transcriptional programmes underlying the conversion process, how much induced neurons resemble/differ from their endogenous counterparts, how we aim at closing the gap between induced and endogenous neurons, and to which induced neurons succeed to integrate into functional cortical circuits.

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