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SEMINAR CYCLE

of the PhD in Neuroscience of Turin

4nd Appointment

Prof. Marco Peviani

University of Pavia

**“Harnessing the heterogeneity of
neuroinflammatory responses to tackle
neurodegeneration in Amyotrophic Lateral
Sclerosis”**

28th April, 2023 h 11:30 AM

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

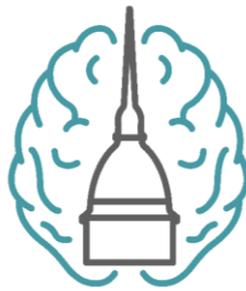
Host: Prof. Maurizio Giustetto



Aula C Anatomia - Corso Massimo D'Azeglio 52

Link: <https://bit.ly/3JzKKWg>

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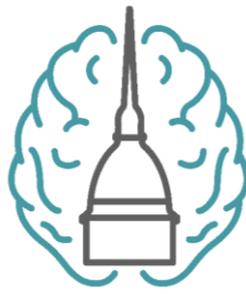
SEMINAR CYCLE

of the PhD in Neuroscience of Turin

PROF. MARCO PEVIANI

Marco Peviani received his PhD in 2010 from the Open University (UK) and Mario Negri Institute (Italy), studying preclinical models of Amyotrophic Lateral Sclerosis (ALS). As a post-doc at the University of Pavia, he validated Sigma-1 receptor as a therapeutic target for ALS. In 2014, he moved to San Raffaele Institute, and then he joined the gene therapy program at Dana-Farber/Boston Children's hospital and Harvard Medical School as an instructor, to optimize a new hematopoietic stem cell gene therapy approach for neuronal ceroid lipofuscinosis. In 2019, he joined the Department of Biology and Biotechnology at the University of Pavia as Assistant Professor, and then Associate Professor of Neuropsychopharmacology since 2021. His major research focus is on the development of new gene/drug-delivery tools and MRI/PET tracers for neurodegenerative disorders.

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ABSTRACT

Amyotrophic lateral sclerosis (ALS) is a fatal neurodegenerative disorder affecting 1-3/100,000 individuals annually that still lacks a cure. ALS is a complex pathology: i) the neurodegeneration is chronic and progressive; it starts in specific regions of brain and spinal cord, and eventually spreads to several districts of the central nervous system (CNS); ii) despite brain and spinal cord motor neurons are the primary target of the pathology, other cell types (i.e glial and immune system cells) are actively involved in the disease process, in such a way that modulation of their responses can affect disease progression. We addressed this complexity by exploiting single-cell transcriptome and functional profiling in the animal model to obtain better insights into the heterogeneity of glial-cell responses in ALS; and to develop, at the same time, strategies to obtain targeted engagement of specific reactive glial cell subtypes. In this seminar I will give an update on novel potential therapeutic targets and biomarkers we have identified, and share our plans for future clinical translation of our results.

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