CONVEGNO MONOTEMATICO SIF

GLIAL CELLS AND THERAPEUTIC PERSPECTIVES: FROM MALADAPTIVE PLASTICITY TO NEURORESTORATION

Firenze
Aula Magna del Rettorato, Piazza San Marco 4
29 giugno 2018

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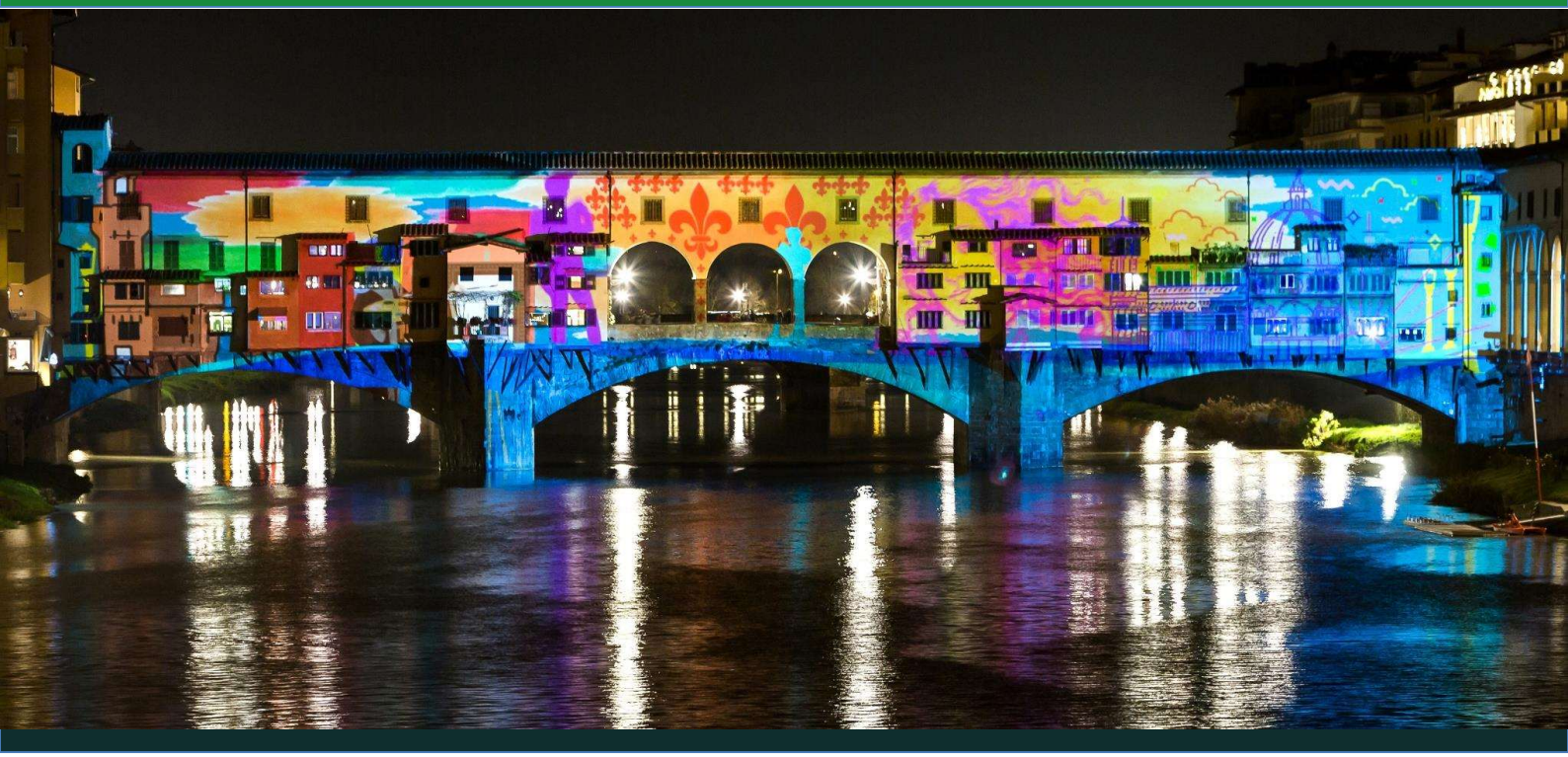

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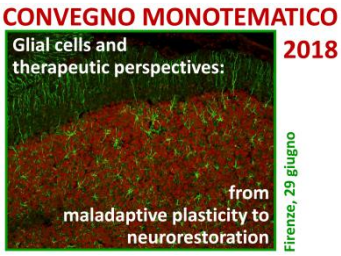

CONVEGNO MONOTEMATICO

"Glial cells and therapeutic perspectives: from maladaptive plasticity to neurorestoration"

Firenze, 29 giugno 2018

Aula Magna del Rettorato, Piazza San Marco 4

ore 9:00 -9:30 Accoglienza partecipanti - Registrazione e Welcome Coffee

ore 9:30-10:00 Saluto Autorità

ore 10:00-10:30 **Claudia Verderio**
(introduce: Maria Angela Sortino)

Multimodal microglia modulation of neuronal function via extracellular vesicles: implications in neuroinflammatory diseases

ore 10:30-12:45 Sessione I: Glial cells, single phenotypes and cell-cell interactions

(moderatori: Mariagrazia Grilli, Anna Pittaluga)

Astrocyte heterogeneity in health and disease: insight into mechanisms regulating the neurogenic activation of parenchymal astroglia and astrocyte-dependent cerebellar functions
Buffo A.

Role of the cross-talk between microglia and oligodendroglial progenitors in cerebral ischemia
Fumagalli M.

Role of NPC-astrocyte crosstalk in Down syndrome
Salvalai M.E.

The neuron-astrocyte-microglia triad in rodent models of neurodegeneration
Giovannini M.G.

Neuroinflammation: Glia, Mast Cells, and Their Interactions
Zusso M.

G protein coupled receptor heterodimers on astrocytes: presence and function
Marcoli M.

Role of astrocytes in PBMCs migration through an in vitro model of blood brain barrier
Spampinato S.F.

CCL5-glutamate cross-talk in astrocyte-neuron communication in mammal CNS
Pittaluga A.

Sphingosine 1-phosphate receptor subtype 1 (S1PR1) as a therapeutic target for brain trauma
Paterniti I.

12.45 -13.45 Pranzo

13.45 -14.15 **Annamaria Vezzani**
(introduce: Sabatino Maione)

Innate immunity and inflammation in epilepsy: the pathophysiological role of glial cells

14.15 -14.30 Saluti del Magnifico Rettore

14.30 -16.45 Sessione II: Therapeutic approaches and cross-talk between peripheral and central glial cells

(moderatori: Emanuela Masini, Salvatore Cuzzocrea)

Brain plasticity in chronic pain: glial cells as potential pharmacological targets
Ceruti S.

Targeting HCAR2 to treat neuropathic pain
Boccella S.

Glia and Alzheimer's disease: the pharmacological manipulation as promising tool against pathology progression
Scuderi C.

N-palmitoylethanolamide prevents Parkinsonian phenotypes in aged mice
Crupi R.

Exosome-shuttled miRNAs derived from mesenchymal stem cells modulate in-vitro the reactive phenotype of amyotrophic lateral sclerosis glial cells
Milanese M.

Antibiotic-induced microbiota perturbation causes gut endocannabinoidome changes, hippocampal neuroglial reorganization and depression in mice
Iannotta M.

Protease and protease-activated receptors as regulators of peripheral nerve regeneration
Fabrizi C.

Schwann cell TRPA1 mediates neuroinflammation that sustains macrophage-dependent neuropathic pain in mice
De Logu F.

Parabrachial nucleus astrocytes modulate pain perception: a protective skill
Di Cesare Mannelli L.

16.45 Conclusioni (C. Ghelardini)

Exosome-shuttled miRNAs derived from mesenchymal stem cells modulate in-vitro the reactive phenotype of amyotrophic lateral sclerosis glial cells.

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Amyotrophic lateral sclerosis (ALS) is neurodegenerative disease characterized by degeneration of motor neurons (MNs), caused by cellular and molecular alterations involving also glial cells, such as microglia and astrocytes.
We demonstrated that intravenous mesenchymal stem cells (MSCs) administration prolongs survival probability, ameliorates motor skills and reduces reactive gliosis in the $SOD1^{G93A}$ mouse model of ALS. We postulated that MSCs produce their beneficial effects via the modulation of glial cell reactive phenotypes, through specific microRNAs (miRNAs) shuttled by MSCs-secreted exosomes. Indeed, in-vitro experiments showed that MSCs-derived exosomes can induce a positive switch of the reactive phenotype in microglia and astrocyte cell cultures derived from symptomatic $SOD1^{G93A}$ ALS mice. Of note, also specific mimics of miRNAs (such as 466q and 467f), which have been found up-regulated in MSC-derived exosomes, are able to affect the reactive phenotype of ALS glial cells. In detail, after treatment with MSCs-derived exosomes we registered a significant reduction of specific markers for reactive gliosis/neuroinflammation (IL1β, TNFα, IL6, NLRP3, GFAP, vimentin) paralleled by an increased expression of neuroprotective/anti-inflammatory factors (CX3CL1, NR4A2, IL-10).
These results suggest that exosome-shuttled miRNAs can modulate the reactive phenotype of glial cells and represent a promising therapeutic approach to be tested in pre-clinical trial.