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BOOK OF ABSTRACTS

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Inhibition of miR-21 promotes cellular senescence in NT2-derived astrocytes

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Astrocytes are the main homeostatic cells in the central nervous system (CNS) that provide mechanical, metabolic, and trophic support to neurons. Disruption of their physiological role or acquisition of senescence-associated phenotype can contribute to the CNS dysfunction and pathology. However, molecular mechanisms underlying the complex physiology of astrocytes are explored insufficiently. Recent studies have shown that miRNAs are involved in the regulation of astrocyte function through different mechanisms. Although miR-21 has been reported as an astrocytic miRNA with an important role in astrogliosis, no link between this miRNA and cellular senescence of astrocytes has been identified. To address the role of miR-21 in astrocytes, with special focus on cellular senescence, we used NT2/A (astrocytes derived from NT2/D1 cells). Downregulation of miR-21 expression in both immature and mature NT2/A by the antisense technology induced the arrest of cell growth and premature cellular senescence, as indicated by senescence hallmarks such as increased expression of cell cycle inhibitors p21 and p53 and augmented senescence-associated β-galactosidase activity. Additionally, in silico analysis predicted many of the genes, previously shown to be upregulated in astrocytes with the irradiation-induced senescence, as miR-21 targets. Taken together, our results point to miR-21 as a potential regulator of astrocyte senescence. To the best of our knowledge, these are the first data showing the link between miR-21 and cellular senescence of astrocytes. Since senescent astrocytes are associated with different CNS pathologies, development of novel therapeutic strategies based on miRNA manipulation could prevent senescence and may improve the physiological outcome.