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Editorial

Quantitative Polymerase Chain Reaction (Q-PCR)

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INTRODUCTION

Play a key role in neurogenesis, both under physiological and pathological conditions, since they are responsible of tissue homeostasis and repair to brain damage after injury. Though mostly quiescent, they can proliferate under certain circumstances and differentiate into the three main neural cell lineages: neurons, oligodendrocytes and astrocytes. A wide array of studies in progress is still aimed at identifying NSC specific markers, with GFAP and nestin being commonly recognized as non-specific markers of NSC. Indeed, GFAP is also a marker of astrocytes, while nestin of Neuronal Progenitor Cells (NPCs). In neurogenesis, GFAP+ cells include both the stem cell source generating NPCs and astroglial cells providing trophic and instructing support to migrating NPCs and neuronal differentiation. GFAP is a protein of Intermediate Filaments (IFs) which represent a master player in the cytoskeletal organization, cell migration and adhesionwith multiple splicing variants that have been identified and variably related to different areas of expression and functions. In particular, two peculiar cytoskeletal proteins have been associated to NSCs-derived glial cells and related neurodegenerative diseases like Alzheimer's Disease (AD): gelsolin andHuman is a GFAP protein isoform that GFAP-δ is encoded by an alternative splice variant of the GFAP gene. Previous studies have shown that GFAP is specifically expressed by a subpopulation of astrocytes located in the subpial zone of the cerebral cortex, the Subgranular Zone (SGZ) of the hippocampus and by a ribbon of astrocytes following the ependymal layer of the cerebral ventricles. Therefore, GFAP specifically marks the astrocytes in the SVZ containing the NSCs. Interestingly, its expression has been found upregulated in SVZ of patients of AD, most likely as a consequence of constitutive splicing than of astrogliosis. Indeed, given the ability of GFAP to change the assembly properties of GFAP filaments, its role has been related to the modulation of the volume and/or cellular location of IFs in astrocytes. Van Den Berge, et al. have also shown that GFAP is specifically expressed in long-term quiescent cells in the human SVZ. Gelsolin, a cytoskeletal protein, is a founding member of a family of actin binding proteins involved in controlling the organization of the actin cytoskeleton in cells . Its expression seems to be involved in the regulation of membrane ruffling and chemotaxis but it may also act as an inhibitor of Aβ fibrillization in AD patients and as an antioxidant and anti-apoptotic protein . In particular, cytoplasmic gelsolin (c-gelsolin) has been shown upregulated in cells under oxidative stress through a mechanism involving PKC activity as determinant for its upregulation. Moreover, Kronenberg, et al. have demonstrated that gelsolin deficiency does not affect proliferation or neuronal differentiation of adult NPCs but causes a delay of their migration ability in vitro, consistently with slowing the emigration of newly generated cells from the SVZ to the olfactory bulbs in vivo. Considering that the morphological changes mediated by cytoskeletal dynamics are associated to the neural differentiation of NSC and their migration through the physiological neurogenic pathways and, at last instance, to aging and cell death, we analysed the co-expression of gelsolin and GFAP in human neural cell lines both in vitro and in vivo. In particular, we investigated the expression and localization of GFAP and gelsolin in different sources of NPCs and found that the two proteins tend to segregate in separate subcellular compartments and, with differentiation, in different astroglial cells; this dichotomy was maintained in vivo after transplantation of hNSC in the SVZ of healthy such as in lysolecitine or ischemia lesioned rats. In this study, we show how GFAP and gelsolin are co-expressed in undifferentiated cells but never co-localize in the same structure and we hypothesize they could have specific and complementary roles in stem cell differentiation and migration.

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