



Neural Stem Cell Development and Cell Adhesion Molecules

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DESCRIPTION

Different neural cell types emerge and migrate in a precise temporospatial manner in the mammalian Central Nervous System. Neurogenesis peaks at Embryonic day 14 (E14) and declines at Embryonic day 18 (E18) in the mouse brain, when neurons start appear around Embryonic day 12 (E12). The quantity of astrocytes begins to develop at E18 and peaks in the neonatal period. Oligodendrocytes are produced after birth when neurogenesis has mostly stopped.

In vitro cultured neural precursors demonstrated improved neuronal differentiation along with decreased astroglialogenesis and unaltered oligodendrocyte genesis, regardless of whether they were co-grown with fibroblasts overexpressing L1 or cultured on substrate coated with L1. Additional research on several neuronal subtypes that separated from NS/PCs reveals that L1 suppresses cholinergic neuron differentiation but increases GABAergic neuron differentiation.

Ectopic L1 expression in NS/PCs or overexpression of L1 on ESC-derived neural precursors improves neuronal development but inhibits astrocytic differentiation in an *in vitro* culture system. When GABAergic neurons were transplanted into striatum that had been damaged, L1 overexpression also caused a greater yield of these neurons. The SVZ, hippocampus, and rostral migratory stream are among the adult CNS's neurogenic areas that express PSA-NCAM, another significant IgSF CAM that has been identified as a neural precursor marker (RMS).

The development of the mouse brain depends on the migration and differentiation of neural precursors under the control of Polysialic Acid (PSA). In the OB, where they differentiate into interneurons that do not express PSA, SVZ precursors travel along the RMS. It's interesting to note that migratory failure and premature differentiation occur when PSA is specifically deleted from SVZ precursors using Endoneuraminidase-N (Endo N). Nestin-positive cells do not migrate and do not acquire the characteristics of neuronal cells, such as long neurites. These cells also exhibit positive Tyrosine Hydroxylase (TH). Further research showed that cell-cell contact, which is made possible by

NCAM activating p59fyn and MAPK p44/42 activation, is necessary for PSA-dependent differentiation.

After PSA was removed using a double-knockout strategy for the PSA synthases ST8SiaII and ST8SiaIV, there were more astrocytes that were positive for the Glial Fibrillary Acidic Protein (GFAP), both *in vitro* and *in vivo*. TAG-1 interacts with the amyloid precursor protein to suppress neurogenesis (APP). When TAG-1 and APP connect to one another, the APP Intracellular Domain (AICD) is released, which causes the APP signalling pathway to be activated and negatively inhibit neurogenesis. These two molecules are co-localized in the neurogenic niche and neural progenitors. Fe65 has been discovered to be a downstream component of TAG-1-APP signalling during the control of neurogenesis and is expressed in the neurogenic VZ as well.

β 4-integrin plays an important role in NS/PC differentiation. Under *in vitro* culture system, reduced of endogenous β 4-integrin inhibits cell differentiation and decreases of FGFR2 action, while overexpression of β 4-integrin in NS/PCs promotes differentiation. The FNIII domain 6-8 of ECM molecule tenascin-R, through interactions with β 1-integrin, stimulates astrocytic differentiation, while stopping differentiation into neurons and oligodendrocytes; Contrariwise, Epidermal Growth Factor Like (EGFL) domain of tenascin-R promotes neuronal differentiation, while reducing differentiation into astrocytes and oligodendrocytes, through interactions with β 1-integrin that can be inhibited with application of β 1-integrin antibody.

CONCLUSION

The parts of cadherins on neural diversity are still not well assumed. Although neural differentiation appears to occur normally in N-cadherin-deficient mice, a recent study knocking down N-cadherin expression by *in utero* eletroporation caused an enhanced neuronal differentiation, likely through β -catenin signaling as it can be saved by presenting a stabilized active form of β -catenin.

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