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SOX transcription factors – choosing between stemness and neuronal differentiation

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The *SOX* genes show properties of both classical transcription factors and architectural components of chromatin. As a main part of regulatory networks that consist of transcription factors, epigenetic modifiers and microRNAs, they govern diverse cellular processes during embryonic and adult neurogenesis, such as maintaining the multipotency of neural stem cells, cell fate decision and terminal cell differentiation.

The sequential expression of the SoxB, SoxC, SoxD, SoxE and/or SoxF group members in differentiating and mature neurons, oligodendrocytes and astrocytes is essential for their identity and survival. Several studies revealed that deregulated expression of these proteins had significant impact on the neuronal plasticity. Results of our research using both *in vivo* and *in vitro* model systems to study neurogenesis in different brain pathologies, demonstrated decreased expression of SOXB transcription factor in neural stem/progenitor cells in early stages of neurodegeneration or following ischemia-related injury that has significant impact on regeneration capacity. Recently, it has been demonstrated that neural stem cells from SVZ are cells of origin of glioblastoma, the most common and devastated brain tumor. Our investigation of SOXB showed deregulated expression of SOX3 protein also in glioblastoma samples, compared to the expression in non-tumoral brain tissues while exogenous overexpression of this gene promoted the malignant properties of glioblastoma cells.

Despite numerous data implicating the key role of *Sox/SOX* genes in regulation of neurogenesis, contribution of *SOX* genes in various brain pathologies is still not clear. Future studies are needed to evaluate them as a potential targets in therapeutic and regenerative strategies.

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