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## The NG2 proteoglycan is an endogenous modulator of adverse glial responses and counteracts neurological deficits following traumatic brain injury

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Traumatic brain injury (TBI) is a major cause of death and disability and therapeutic options are limited. The underlying pathophysiology is characterized by secondary changes in cerebral blood flow and metabolism, progressive brain tissue damage, activation of glia cells as well as the CNS inflammation. Taking advantage of gene-deficient mice, we studied the role of the NG2 proteoglycan in response to TBI using controlled cortical impact as a model system. Seven days after TBI behavioral analysis, brain damage volumetry, assessment of blood brain barrier integrity as well as immunohistochemical analysis of astrocytes and microglia demonstrated an exacerbated response of NG2-deficient compared to wild-type mice. Moreover, PCR array screening revealed a striking TBI-induced up-regulation of the Cxcl13 chemokine in NG2-deficient mice. CXCL13, known to attract immune cells to the inflamed brain was expressed by activated perilesional microglia/macrophages and enhanced CD45+ leukocyte infiltration was observed thirty days after TBI. Thus, NG2-deficiency exacerbates neurological deficits after TBI and associates with abnormal activation of astrocytes, microglia/macrophages and increased leukocyte recruitment to the injured brain. These findings suggest that NG2 may counteract neurological deficits and adverse glial responses in TBI

### Biography

Michael K E Schaefer has completed his PhD in Biology and he is currently a Professor for Experimental Anesthesiology at the University Medical Center Mainz, Germany. He is also affiliated to the Research Focus Program Translational Neurosciences in Mainz. His work is focused on neurodevelopment disorders as well as acute and chronic neurodegeneration in response to brain injury.

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