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Intracellular trafficking in neurones and glia of fibroblast growth factor-2, fibroblast growth factor receptor 1 and heparan sulphate proteoglycans in the injured adult rat cerebral cortex.

Leadbeater WE, Gonzalez AM, Logaras N, Berry M, Turnbull JE, Logan A.
Molecular Neuroscience Group, Division of Medical Sciences, University of Birmingham, Birmingham, UK. w.leadbeater@bham.ac.uk

Abstract
The potent gliogenic and neurotrophic fibroblast growth factor (FGF)-2 signals through a receptor complex comprising high-affinity FGF receptor (FGFR)1 with heparan sulphate proteoglycans (HSPGs) as co-receptors. We examined the intracellular dynamics of FGF-2, FGFR1 and the HSPGs syndecan-2 and -3, glypican-1 and -2, and perlecan in neurones and glia in and around adult rat cerebral wounds. In the intact cerebral cortex, FGF-2 and FGFR1 mRNA and protein were constitutively expressed in astrocytes and neurones respectively. FGF-2 protein was localized exclusively to astrocyte nuclei. After injury, expression of FGF-2 mRNA was up-regulated only in astrocytes, whereas FGFR1 mRNA expression was increased in both glia and neurones, a disparity indicating that FGF-2 may act as a paracrine and autocrine factor for neurones and glia respectively. FGF-2 protein localized to both cytoplasm and nuclei of injury-responsive neurones and glia. There was weak or no staining of HSPGs in the normal cerebral neuropil and glia nuclei, with a few immunopositive neurones. Specific HSPGs responded to injury by differentially co-localizing with trafficked intracellular FGF-2 and FGFR1. The spatiotemporal dynamics of FGF-2-FGFR1-HSPG complex formation implies a role for individual HSPGs in regulating FGF-2 storage, nuclear trafficking and cell-specific injury responses in CNS wounds.

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