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Intracisternal antisense oligonucleotide to growth associated protein-43 blocks the recovery-promoting effects of basic fibroblast growth factor after focal stroke.

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Focal infarction (stroke) of the lateral cerebral cortex of rats (including the sensorimotor cortex) produces deficits in sensorimotor function of the contralateral limbs that recover partially over time.

In previous studies, we found that the intracisternal injection of basic fibroblast growth factor (bFGF), a potent neurotrophic growth factor, starting at 1 day after stroke, significantly enhanced recovery of sensorimotor function of the contralateral forelimb and hindlimb. Moreover, immunoreactivity (IR) for growth-associated protein-43 (GAP-43), a molecular marker of new axonal growth, was increased in the intact contralateral sensorimotor cortex following bFGF treatment. In the current study, we found that the intracisternal administration of antisense, but not missense, oligonucleotide to GAP-43 blocked the recovery-enhancing effects of bFGF and blocked the increase in GAP-43 IR in the contralateral cortex.

These results suggest that upregulation of GAP-43 expression and consequent enhanced axonal sprouting in intact uninjured parts of the brain are likely mechanisms for the recovery-promoting effects of bFGF.

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