Erythropoietin and Bcl-X_L for neuroprotection and axonal regeneration in the injured CNS

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Axonal regeneration in the injured adult CNS is sparse, and is rarely functionally relevant. The glial environment is considered inhibitory, with oligodendrocytes producing myelin inhibitors and activated astrocytes forming a physical barrier to outgrowing neurites. On the other hand, neuron-intrinsic growth potential decreases with age, leading to neuron-intrinsic loss of regeneration potential. We address neuron-intrinsic potential for axonal regeneration. Here we report that both Bcl-X_L and erythropoietin (EPO) are capable of promoting axonal regeneration in a CNS lesion paradigm *in vitro*. Bcl-X_L gene transfer or EPO treatment both protect retinal ganglion cells (RGCs) of adult rodents from degenerating in response to axonal lesion, and enhance the intrinsic potential for axonal regeneration from injured RGCs *in vitro*. Aspects of the signal transduction cascades, and functional implications will be discussed.

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