The AP-1 transcription factor C-Jun is required for efficient axonal regeneration

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Nerve injury triggers numerous changes in the injured neurons and surrounding non-neuronal cells that ultimately result in successful target reinnervation or cell death. C-Jun is a component of the heterodimeric AP-1 transcription factor and c-Jun is highly expressed in response to neuronal trauma. Here we have investigated the role of c-jun during axonal regeneration using mice lacking c-jun in the central nervous system. After transsection of the facial nerve, the absence of c-Jun caused severe defects in several aspects of the axonal response including perineuronal sprouting, lymphocyte recruitment and microglial activation. C-Jun-deficient motoneurons were atrophic, resistant to axotomy-induced cell death and showed reduced target muscle reinnervation. Expression of CD44, galanin and alpha7beta1 integrin, molecules known to be involved in regeneration, was greatly impaired, suggesting a mechanism for c-Jun-mediated axonal growth. Taken together, our results identify c-Jun as an important regulator of axonal regeneration in the injured central nervous system.