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Spatial and temporal expression of developmentally regulated proteins during spinal cord formation

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Background: Between 250,000 and 500,000 persons suffer a spinal cord injury every year. Previous proteomics analysis in the lab has identified proteins that are specifically expressed in the developing spinal cord of rats and may be important for the generation of the corticospinal tract (CST).

Aim: The aim of this study was to characterize the timing and location of expression of these proteins in the developing rat to relate them to spinal cord formation.

Methods: Cross sections from postnatal day (P) 0, 3, 7, 10, 14, 17 rat cervical spinal cord were incubated with antibodies against stathmin1 p38, DPYSL3, BLBP and CRABP1. Then, they were "stained" with an appropriate 594 (RED) fluorescent secondary antibody.

Results: While the expression of CRABP1, phosphorylated stathmin 1 (p38) and DPYSL3 appear restricted to the CST at specific stages of development, BLBP appears to be more globally distributed throughout the spinal cord.

Conclusion: This characterization offers insights into the role that certain developmentally regulated proteins may play in spinal cord formation and the potential importance of these proteins in future therapies for spinal cord injury.

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New therapeutic modalities for Alzheimer's disease

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Genetic analysis of familial forms of Alzheimer's disease (AD) causally links the proteolytic processing of the amyloid precursor protein (APP) and AD. However, the specific type of amyloid and mechanisms of amyloid pathogenesis remain unclear. We conducted a detailed analysis of intracellular amyloid with an aggregation specific conformation dependent monoclonal antibody, M78, raised against fibrillar Aß42. M78 immunoreactivity colocalizes with Aß and the carboxyl terminus of APP (APP-CTF) immunoreactivities in perinuclear compartments at intermediate times in 10 month 3XTg-AD mice, indicating that this represents misfolded and aggregated protein rather than normally folded APP. At 12 months, M78 immunoreactivity also accumulates in the nucleus. Neuritic plaques at 12 months display the same spatial organization of centrally colocalized M78, diffuse chromatin and neuronal nuclear NeuN staining surrounded by peripheral M78 and APP-CTF immunoreactivity as observed in neurons, indicating that neuritic plaques arise from degenerating neurons with intracellular amyloid immunoreactivity. The same staining pattern was observed in neuritic plaques in human AD brains, showing elevated intracellular M78 immunoreactivity at intermediate stages of amyloid pathology (Braak A and B) compared to no amyloid pathology and late stage amyloid pathology (Braak 0 and C, respectively). These results indicate that intraneuronal protein aggregation and amyloid accumulation is an early event in AD and that neuritic plaques are initiated by the degeneration and death of neurons by a mechanism that may be related to the formation of extracellular traps by neutrophils.

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