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Specific immune tolerance to spinal antigens as a protective strategy in rats with traumatic spinal cord injury

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Spinal cord injury (SCI) elicits a robust intra-spinal inflammatory response with potentially devastating consequences. Immune cells present in the injury site may sequester cell debris and carry spinal antigens (SAgs) to secondary lymphoid organs. There, SAgs may be processed and presented by antigen presenting cells to lymphocytes, triggering lymphocyte activation. In clinical and experimental SCI, only a few autoantigen targets have been documented. ACAID (anterior chamber associated immune deviation) is an antigen-specific form of peripheral immune tolerance (IT) that is induced against exogenous antigens placed in the anterior chamber (AC) of the eye. It is characterized by the inhibition of delayed hypersensitivity reactions to the AC-injected antigens. This IT is elicited by AC-induced CD4+ CD25+ antigen-specific regulatory T cells. Since neural degeneration after SCI includes a strong inflammatory component triggered by reactivity against multiple antigens derived from the neural tissue, this project proposes to induce IT against SAgs as a neuro-protective strategy in SCI. Results to date indicate that it is possible to induce IT to a cocktail of SAgs obtained from healthy rat spinal cord by ACAID. Neuroprotection of the spinal cord will be evaluated by histological and immuno-fluorescence techniques and further functional evaluations will be done with sensorimotor tests as indicators of spinal cord tissue preservation.

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