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The effects of spinal cord injury in the hippocampus

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The hippocampus encodes spatial and contextual information involved in memory and learning. The incorporation of new neurons into hippocampal networks increases neuroplasticity and enhances hippocampal-dependent learning performances. Only few studies have described hippocampal abnormalities after spinal cord injury (SCI) although cognitive deficits related to hippocampal function have been reported in rodents and even humans. The aim of this study was to characterize in further detail hippocampal changes in the acute and chronic SCI. Our data showed that short term after SCI hippocampal neurogenesis was reduced as a consequence of increased amplifying neural progenitor (ANP) death. In addition, astrocytes became reactive and microglial cells increased their number in almost all hippocampal regions studied. Glial changes resulted in a non-inflammatory response as the mRNAs of the major pro-inflammatory cytokines (IL-1β, TNFα, IL-18) remained unaltered, but CD200R mRNA levels were downregulated. Long-term after SCI, astrocytes remained reactive but on the other hand, microglial cell density decreased. Also, glial cells induced a neuroinflammatory environment with the upregulation of IL-1β, TNFα and IL-18 mRNA expression and the decrease of CD200R mRNA. Neurogenesis reduction may be ascribed at later time-points to inactivation of neural stem cells (NSCs) and inhibition of ANP proliferation. The number of granular cells and CA1 pyramidal neurons decreased only in the chronic phase. The release of pro-inflammatory cytokines at the chronic phase might involve neurogenesis reduction and neurodegeneration of hippocampal neurons. Therefore, SCI led to hippocampal changes that could be implicated in cognitive deficits observed in rodents and humans.

References

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