Presentation Abstract

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Presentation Title: Copolymer-1 induces neurogenesis in a model of cerebral ischemia with reperfusión.

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Abstract: Cerebral ischemia is a disease caused by either temporal or permanent alterations in blood supply, this causes the development of a whole series of autodestructive events including excitotoxicity, apoptosis or inflammation that lead to
neural death. Immunization with copolymer-1 (COP-1), proved to have neuroprotective effects after cerebral ischemia since, preserves tissue and improves neurological recovery. COP-1 modulates the immune response by inducing a Th-2 phenotype. These lymphocytes release anti-inflammatory cytokines but also neurotrophic factors such as BDNF and NT-3 that in turn, could promote neuroprotection and neural restoration. That is why we investigated if COP-1 is capable of promoting neurogenesis after cerebral ischemia in rats. For this purpose, we conducted two experiments, in the first, the amounts of trophic factor proteins (BDNF and NT-3; ELISA assays) were determined at three and
seven days after focal cerebral ischemia (FCI). The ischemic procedure was performed by using the middle cerebral artery occlusion (MCAO) model. To verify MCAO severity, regional cerebral blood flow was determined by laser-Doppler flowmetry. After a 90 minutes occlusion the suture was withdrawn and cerebral blood flow recovered. In the second study, we determined the ability of COP-1 to induce neurogenesis by performing a double labeling (BrdU/DCX; immunohistochemistry assays) of neurons at the dentate gyrus, subventricular zone and cerebral cortex of the brain at seven days and two months after FCI. In both experiments, the neurological recovery of animals was evaluated. The results showed that COP-1-immunization induced a better neurological recovery and a significant increase of NT-3 but not of BDNF. As well, COP-1 increased the neurogenesis in all brain regions, mainly at the subventricular zone. These findings suggest that immunization with COP-1 promotes the expression of NT-3 which could be essential for the induction of neurogenesis in the model of cerebral ischemia with reperfusion.

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