NEUROPROTECTIVE EFFECT OF SODIUM PROPIONATE IN NEUROINFLAMMATION MODELS

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Introduction: Sodium propionate (SP) is one of the main short chain fatty acids (SCFA) that can be produced naturally through host metabolic pathways. The physiological effects of SP have been documented and include the reduction of pro-inflammatory mediators in an vivo model of colitis. Thus, the aim of this study is to evaluate the neuroprotective effects of SP in reducing inflammatory process associated to neurological disorders.

Materials and methods: We performed both in vitro model of Alzheimer's disease, induced by oligomeric A_{1-42} stimulation, and in in vivo model of spinal cord injury (SCI) in which neuroinflammation plays a crucial role. For in vitro model, the human neuroblastoma SHSY-5Y cell line was first differentiated with retinoic acid (100 μM) for 24 hours and then stimulated by oligomeric A_{1-42} (1μg) and treated with SP at three different concentrations (0.1- 1- 10 μM) for another 24 hours. Instead, the in vivo model of SCI was induced by extradural compression of the spinal cord at T6-T7 levels, using an aneurysm clip, and animals were treated with SP (10-30-100 mg/kg o.s) 1 and 6 h after SCI.

Results: Our results demonstrated that both in in vitro neuroinflammatory model and in vivo model of SCI the treatment with SP significantly reduced NF-κB nuclear translocation and IκBα degradation, as well as decreases COX-2 and iNOS expressions evaluated by Western blot analysis. Moreover, we showed that SP treatment significantly ameliorated histopathology changes and improved motor recovery in a dose-dependent manner.

Discussion and conclusion: In conclusion, our results demonstrated that SP possesses neuroprotective effects, suggesting it could represent a target for therapeutic intervention in inflammatory disorders such as neurodegenerative disease and central nervous system injury.