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Protective effect of Sodium propionate in Aβ1-42 -Induced Neurotoxicity and Spinal Cord Trauma

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Abstract

Sodium propionate (SP) is one of the main short chain fatty acids (SCFA) that can be produced naturally through host metabolic pathways. SP have been documented and include the reduction of pro-inflammatory mediators in an in vivo model of colitis. The aim of this study is to evaluate the neuroprotective effects of SP in reducing inflammatory process associated to neurological disorders. We performed both in vitro model of Alzheimer's disease, induced by oligomeric Abeta1-42 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 SH-SY5Y cell line was first differentiated with retinoic acid (100 µM) for 24 hours and then stimulated by oligomeric Abeta1-42 (1µg/ml) and treated with SP at 0.1- 1- 10 µM concentrations for another 24 hours. Instead, the in vivo model of SCI was induced by extradural compression of the spinal cord at T6-T8 levels, and animals were treated with SP (10-30-100 mg/kg o.s) 1 and 6 h after SCI. 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. In conclusion, our results demonstrated that SP possesses neuroprotective effects, suggesting it could represent a target for therapeutic intervention in neuroinflammatory disorders.

Biography

Irene Paterniti, PhD, is a post doc fellow at the Department of Chemical, Biological, Pharmaceutical and Environmental Sciences of Messina University. She is a author of 90 scientific research papers (H index: 25) published on international refereed journals. Her area of interest is the role of inflammatory process and oxidative stress in different pathology all aimed at protecting the health of the community.

Publications

1. FeTPPS Reduces Secondary Damage and Improves Neurobehavioral Functions after Traumatic Brain Injury
2. Traumatic Brain Injury Leads to Development of Parkinson's Disease Related Pathology in Mice
3. KU0063794, a Dual mTORC1 and mTORC2 Inhibitor, Reduces Neural Tissue Damage and Locomotor Impairment After Spinal Cord Injury in Mice.
4. Emerging pharmacotherapy for treatment of traumatic brain injury: targeting hypopituitarism and inflammation.
5. Phosphodiesterases as a New Therapeutic Targets for the Treatment of Spinal Cord Injury and Neurodegenerative Diseases.
6. Phosphodiesterase as a new therapeutic target for the treatment of spinal cord injury and neurodegenerative diseases
7. Exogenous T3 administration provides neuroprotection in a murine model of traumatic brain injury.
8. GW0742, a high-affinity PPAR-δ agonist, mediates protection in an organotypic model of spinal cord damage.
9. Effect of Apocynin, an inhibitor of NADPH oxidase, in the inflammatory process induced by an experimental model of spinal cord injury.
10. MK801 attenuates secondary injury in a mouse experimental compression model of spinal cord trauma.

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