Protective effects of Chondroitin Sulfate against glutamate-induced excitotoxicity in astrocytes

Sandra Regina Perosa a, Gustavo José da Silva Pereira b, Bruno Palmiere de Souza b, Soraya Soubhi Smaili b and Maria da Graça Naffah Mazzacoratti a,c

a Departamento de Neurologia/Neurocirurgia, Universidade Federal de São Paulo, Brazil; b Departamento de Farmacologia-INFAR, Universidade Federal de São Paulo, Brazil; c Departamento de Bioquímica, Universidade Federal de São Paulo, Brazil

e-mail: perosa.nexp@epm.br; gujspharma@gmail.com; b.palmiere@gmail.com; ssmaili@unifesp.br; naffahmazz.nexp@epm.br

tel:+55-11-55-76-4846

Background: Chondroitin sulfate (CS) is a glycosaminoglycan (GAG) linked to several types of proteoglycans in high concentration in the extracellular matrix of the central nervous system (CNS). They have critical roles in the brain development, plasticity and post-injury response. Recent evidences have revealed that CS may have therapeutic interest in CNS diseases due to its anti-inflammatory and anti-apoptotic properties. Aims: Since neuroinflammation has been implicated in different brain pathologies, this study was planned to investigate how CS could modulate the inflammatory response and cell death in CNS. Methods: Primarily astrocytes were stimulated with glutamate (Glu) and were investigated the expression of kinin B1 and B2 receptors (B1BK and B2BK), the ratio between bands p42/p44 of MAPK and apoptosis markers, employing Western Blotting and immunofluorescence techniques. Results: Cell viability assay showed that at 10µM CS was able to increased the astrocytes viability, when exposed to Glu (1mM) after 24 h of treatment (Glu: 79.0±4.74; Glu+CS: 97.0±2.14 cells p<0.01), and reduced the expression of B1BK receptor (Glu: 0.534±0.019; Glu+CS: 0.382±0.025 p=0.02) as well as decreased the levels of p53 in (Glu:
3.76±0.33; Glu+CS: 2.67±0.04) and phosphorylation of p53 (Glu: 1.89±0.01; Glu+CS: 1.12±0.1) as pro-apoptotic proteins. However, there is not involvement in expression of B2BK receptor and p42/44 MAPK. **Conclusion:** These results indicate that CS reduces excitotoxicity, reducing B1BK receptors levels and protect astrocytes against Glu-induced cell death, *in vitro.*

**Information on ethical approval:** The animal experiments were performed under Institutional Ethical Approval of protocol (CEP:1469/10) and all efforts were made to minimize animal suffering.

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