Effect of Steroid and Non-steroid Anti-inflammatory Compounds on S100B Secretion in Primary Astrocyte Cultures Exposed or Not To LPS

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S100B is a protein produced and secreted by astrocytes in SNC, and is involved in several neuroinflammatory disorders, like Alzheimer’s disease (DA). One of the current therapeutic approaches to DA is the use of commercial anti-inflammatories and the development of anti-inflammatories which act selectively in SNC. Lipopolysaccharide (LPS) is used in models of neuroinflammation, however, there are not studies showing the effect of LPS in the secretion of S100B. The aim of this work is investigate the S100B secretion in primary astrocyte cultures exposed to steroid and non-steroid anti-inflammatory compounds in presence or not of LPS. Primary astrocyte cultures were prepared from cerebral cortex of newborn Wistar rats and were allowed to grow to confluence. S100B secretion was determined in culture medium by ELISA at 1 and 24h of exposure to different anti-inflammatory compounds (dexametasone 0.1µM, acetilsalicilic acid 100µM, and sodium diclofenac 100µM) in presence or not of LPS 0.1µg/mL. Acetilsalicilic acid was able to reduce S100B secretion (while sodium diclofenac and dexametasonse increases S100B secretion in 1h of treatment). However, in 24h of anti-inflammatory exposure, only dexametasonse was able to induce a decrease in S100B secretion. After 1h of exposure to LPS 0.1µg/mL acetilsalicilic acid and sodium diclofenac were able to increase S100B secretion. However, during 24h of exposure to LPS only dexametasonse was able to decrease S100B secretion. The effect of different anti-inflammatory compounds in astrocytes, particularly on S100B secretion, in presence or not from LPS reinforce the possibility of astrocytes became therapeutic target in neuroinflammatory diseases.

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