Aspirin prevents cell damage induced by Aβ1-42 on astrocytes in primary culture

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Introduction: Astrocytes increase neuronal viability and mitochondrial biogenesis, protecting from oxidative stress and inflammation induced by toxic amyloid peptide (Aguirre et al. 2015 a, b). Aspirin has been used as anti-inflammatory and anti-aggregate for decades but the precise mechanism(s) of action after the presence of the toxic peptide Aβ1-42 in cultured astrocytes remains poorly resolved. Here we use low-doses of aspirin (10-7 M) in astrocytes in primary culture in presence or absence of Aβ1-42 toxic peptide.

Results: It was observed an increase of cell viability and proliferation with or without Aβ1-42 peptide presence in aspirin treated cells. In addition, a decrease in apoptosis, determined by Caspase 3 activity and the expression of Cyt c and Smac/Diablo, were detected. Also, aspirin diminished necrosis process (LDH levels).

Conclusions: Taken together, our results show that aspirin, at low doses increases astrocytes viability and proliferation, decreases apoptosis (Caspase 3, Cyt c and Smac/Diablo) and necrosis (LDH), in the presence or absence of Aβ1-42 peptide.