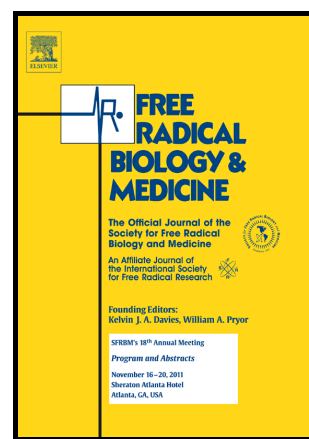


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Reactive oxygen species released from astrocytes treated with amyloid beta oligomers elicit neuronal calcium signals that decrease phospho-Ser727-STAT3 nuclear content

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SUMMARY

The transcription factor STAT3 has a crucial role in the development and maintenance of the nervous system. In this work, we treated astrocytes with oligomers of the amyloid beta peptide (A β O_s), which display potent synaptotoxic activity, and studied the effects of mediators released by A β O_s-treated astrocytes on the nuclear location of neuronal serine-727-phosphorylated STAT3 (pSerSTAT3). Treatment of mixed neuron-astrocyte cultures with 0.5 μ M A β O_s induced in neurons a significant decrease of nuclear pSerSTAT3, but not of phosphotyrosine-705 STAT3, the other form of STAT3 phosphorylation. This decrease did not occur in astrocyte-poor neuronal cultures revealing a pivotal role for astrocytes in this response. To test if mediators released by astrocytes in response to A β O_s induce pSerSTAT3 nuclear depletion, we used conditioned medium derived from A β O_s-treated astrocyte cultures. Treatment of astrocyte-poor neuronal cultures with this

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