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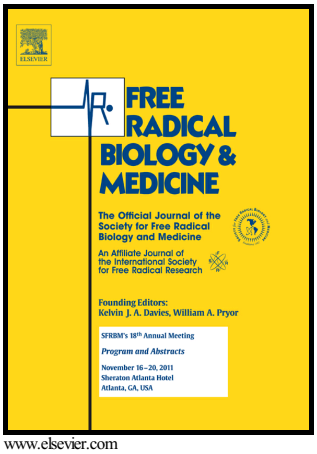

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Reactive oxygen species released from astrocytes treated with amyloid beta oligomers elicit neuronal calcium signals that decrease phospho-Ser727-STAT3 nuclear content

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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 (AβOs), which display potent synaptotoxic activity, and studied the effects of mediators released by AβOs-treated astrocytes on the nuclear location of neuronal serine-727-phosphorylated STAT3 (pSerSTAT3). Treatment of mixed neuron-astrocyte cultures with 0.5 µM AβOs 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 AβOs induce pSerSTAT3 nuclear depletion, we used conditioned medium derived from AβOs-treated astrocyte cultures. Treatment of astrocyte-poor neuronal cultures with this