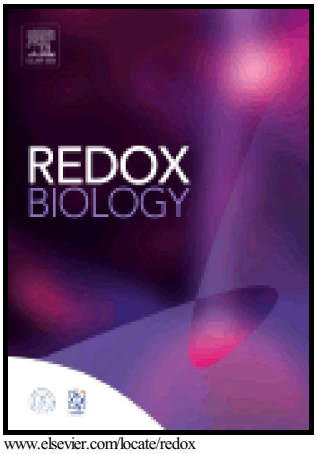

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PII: S2213-2317(16)30094-5
DOI: http://dx.doi.org/10.1016/j.redox.2016.09.001
Reference: REDOX460

To appear in: *Redox Biology*

Received date: 1 August 2016
Revised date: 7 September 2016
Accepted date: 9 September 2016

Cite this article as: Gabriella Testa, Erica Staurenghi, Chiara Zerbinati, Simona Gargiulo, Luigi Iuliano, Giorgio Giaccone, Fausto Fantò, Giuseppe Poli Gabriella Leonarduzzi and Paola Gamba, Changes in brain oxysterols at differen stages of Alzheimer's disease: their involvement in neuroinflammation, *Redox Biology,* http://dx.doi.org/10.1016/j.redox.2016.09.001

Changes in brain oxysterols at different stages of Alzheimer's disease: their involvement in neuroinflammation

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Abstract

Alzheimer's disease (AD) is a gradually debilitating disease that leads to dementia. The molecular mechanisms underlying AD are still not clear, and at present no reliable biomarkers are available for the early diagnosis. In the last several years, together with oxidative stress and neuroinflammation, altered cholesterol metabolism in the brain has become increasingly implicated in AD progression. A significant body of evidence indicates that oxidized cholesterol, in the form of oxysterols, is one of the main triggers of AD. The oxysterols potentially most closely involved in the pathogenesis of AD are 24-hydroxycholesterol and 27-hydroxycholesterol, respectively deriving from cholesterol oxidation by the enzymes CYP46A1 and CYP27A1. However, the possible involvement of oxysterols resulting from cholesterol autooxidation, including 7-ketocholesterol and 7β-hydroxycholesterol, is now emerging. In a systematic analysis of oxysterols in *post-mortem*

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