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To cite this version:
François Mouton-Liger, Anne-Sophie Rebillat, Clarisse Pace, Sarah Gourmaud, Mariko Taga, et al.. Peripheral inflammation increases PKR activation, Tau phosphorylation and amyloid β production in wild-type mice. Molecular Neurodegeneration, 2013, 8 (Suppl 1), pp.P32. 10.1186/1750-1326-8-S1-P32 . inserm-00868758

HAL Id: inserm-00868758
https://inserm.hal.science/inserm-00868758
Submitted on 1 Oct 2013

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Peripheral inflammation increases PKR activation, Tau phosphorylation and amyloid β production in wild-type mice

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From Molecular Neurodegeneration: Basic biology and disease pathways Cannes, France. 10-12 September 2013

Background
Systemic inflammation is correlated with dementia progression. Pro-inflammatory molecules can communicate from the periphery to the central nervous system to induce neuroinflammation and neurodegeneration. Our protein of interest is the pro-apoptotic kinase PKR (the double strand-RNA dependent protein kinase). Increased activated PKR levels were found in AD patients brain and cerebrospinal fluid. PKR activation can be triggered by inflammatory stresses and induces neurotoxicity in vitro. Is in vivo PKR-mediated inflammation involved in AD neurodegenerative process?

Learning objective
To investigate whether PKR-mediated neuroinflammation could play a role in AD neurodegenerative process.

Methods
C57BL/6 wild type mice were injected intraperitoneally with LPS (1mg/kg) versus saline once a day for 3 days to induce PKR activation and neuroinflammation (LPS is the bacilli gram negative endotoxin lipopolysaccharide).

Brains were collected and dissected; immunohistochemistry and western blotting were performed for neuroinflammation, PKR activation and AD pathological hallmarks (as Tau hyperphosphorylation).

Results
Mice showed endotoxin-induced sickness behaviour including body weight loss and elevated serum cytokine levels.

Microglial activation, neuronal apoptosis, increase of PKR, GSK3β and Tau phosphorylation and amyloid β production were found in hippocampus and cortex of LPS-treated mice.

Conclusions
PKR could be involved in the signalling of neurofibrillary tangles formation after a systemic inflammatory challenge.

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Published: 13 September 2013

doi:10.1186/1750-1326-8-S1-P32
Cite this article as: Mouton-Liger et al: Peripheral inflammation increases PKR activation, Tau phosphorylation and amyloid β production in wild-type mice. Molecular Neurodegeneration 2013 8(Suppl 1):P32.

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