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POSTER PRESENTATION

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# Peripheral inflammation increases PKR activation, Tau phosphorylation and amyloid $\beta$ production in wild-type mice

François Mouton-Liger<sup>1,2\*</sup>, Anne-Sophie Rebillat<sup>1</sup>, Clarisse Pace<sup>1</sup>, Sarah Gourmaud<sup>1</sup>, Mariko Taga<sup>1,3</sup>, Claire Paquet<sup>1,2</sup>, Jacques Hugon<sup>1,2</sup>

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## 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 (1mk/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, GSK3 $\beta$  and Tau phosphorylation and amyloid  $\beta$  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.

## Authors' details

<sup>1</sup>Inserm UMR-S839, Paris, France. <sup>2</sup>Hopital Lariboisière APHP, Paris, France. <sup>3</sup>University of Southampton, Southampton, UK.

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<sup>1</sup>Inserm UMR-S839, Paris, France

Full list of author information is available at the end of the article