

Dipartimento di Biotecnologie e Bioscienze – UNIMIB

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Systemic inflammation induced by lipopolysaccharide: Good or Bad for Alzheimer's disease progression?

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Abstract: Systemic inflammation and infections exacerbate the risk and progression of Alzheimer's disease (AD). Recent evidence suggest that is in part mediated by lipopolysaccharide (LPS), a potent pro-inflammatory endotoxin produced by the gut, found in excess levels in AD where it associates with neurological hallmarks of pathology. In this context, we have investigated the impact of systemic inflammation induced by LPS on the progression of amyloid plaque load and tau hyperphosphorylation, the main neurological hallmarks of AD pathology, using mouse models expressing either of these features. In an attempt to identify brain mechanisms contributing to the disease-exacerbating effect of LPS, we also focused on metabolic changes occurring in the brain in the early hours following inoculation with LPS, also looking at the sex-dependency of the metabolic response. We have found that while tau hyperphosphorylation was reduced after LPS, amyloid plaque load was increased, albeit in different mouse models. Four hours after LPS administration, pathways regulating energy metabolism, immune and oxidative stress responses were simultaneously recruited in the hippocampi of 4.5-month-old mice with a more protective response in females despite their pro-inflammatory and pro-oxidant metabolic signature in the absence of immune stimulation and greater plaque load. This questions the contribution of LPS to AD progression.

Host: **Silvia Barabino**

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