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Microglia promotes Amyloid beta accumulation and tau hyperphosphorylation in neurons after exposure to LPS from Porphyromonas gingivalis

A growing body of evidence highlights microglia as key regulators of neurodegenerative processes in Alzheimer's disease (AD), with bacterial components emerging as potential modulators of disease pathology. LPS from Porphyromonas gingivalis (P.gLPS), an oral pathogen–derived virulence factor, has been identified in AD brains, yet its functional role in disease progression is not fully defined. Here, we demonstrate that P.gLPS exposure provokes a robust microglial inflammatory response characterized by elevated secretion of pro-inflammatory mediators such as IL-1β and TNF-α. These cytokines differentially influence neuronal pathology: IL-1β enhances amyloidogenic processes via a cathepsin B–associated mechanism, whereas TNF-α facilitates tau pathology through activation of glycogen synthase kinase 3β signaling. In vivo, systemic administration of P.gLPS leads to sustained cognitive impairment in middle-aged wild-type mice, accompanied by increased intracellular accumulation of amyloid β. In contrast, in transgenic AD models with established amyloid burden, P.gLPS primarily exacerbates disease severity by promoting tau-related neurodegenerative changes. Together, these results delineate a pathway by which infection-related factors modulate microglial activity to differentially impact amyloid and tau pathology across disease stages. Interventions targeting inflammation triggered by chronic oral infection may therefore offer therapeutic potential for delaying or mitigating AD progression.

Keywords: microglia, cytokines, LPS from Porphyromonas gingivalis LPS, amyloid beta, tau hyperphosphorylation.

Biography

Dr. Gui received her PhD in 2025 and studies microglia-mediated neuroinflammation in Alzheimer's disease. Her work reveals that Porphyromonas gingivalis–derived LPS activates microglia to drive amyloid-β accumulation and tau pathology, uncovering key mechanisms by which infection-triggered immune responses accelerate AD onset and progression, and identifying potential therapeutic targets.

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