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Microglia-Mediated Neuroinflammation Drives Neuropathology in Alzheimer's Disease during *P. gingivalis* Infection

Alzheimer's disease (AD) is the leading cause of dementia, yet its underlying drivers remain incompletely understood. Increasing evidence identifies microglia-mediated neuroinflammation as a central mechanism linking environmental risk factors to AD neuropathology. In this talk, I will present our findings demonstrating that *Porphyromonas gingivalis* (P.g) infection drives AD-related neuropathology through microglial activation. Using experimental models of systemic P.g infection and exposure to its lipopolysaccharide (P.gLPS), we show that infection-induced neuroinflammation promotes neuronal amyloid beta (A β) accumulation, tau hyperphosphorylation, and synaptic loss, key features associated with cognitive decline. In parallel, P.g infection enhances peripheral A β generation, suggesting a systemic contribution to brain pathology and highlighting a potential link between peripheral infection and central neurodegeneration. Mechanistically, we identify key enzymes, including cathepsin B and glycogen synthase kinase 3 β , as critical mediators of microglia-driven inflammatory pathways that exacerbate AD neuropathology. These findings provide insight into the molecular mechanisms by which infection-triggered immune responses influence disease progression. Together, our results support a model in which P.g infection amplifies microglia-mediated neuroinflammation, thereby accelerating the progression of AD neuropathology. Targeting infection-associated neuroinflammatory pathways may therefore represent a promising strategy for the prevention and early intervention of AD, particularly at stages when therapeutic efficacy is most achievable.

Keywords: microglia, neuroinflammation, *Porphyromonas gingivalis*, amyloid beta, Cathepsin B, glycogen synthase kinase 3 β

Biography

Dr. Wu holds an MD and PhD and studies how systemic inflammation drives neurodegeneration. Her research focuses on microglia-mediated neuroinflammation and the roles of enzymes such as cathepsins in immune signaling. Her work highlights how *Porphyromonas gingivalis* infection contributes to Alzheimer's disease and identifies potential targets for early intervention.