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Alzheimer's disease - tau proteins – Amyloid - Neurofibrillary tangles - glycogen synthase kinase-3ß

Research
Alzheimer's disease (AD) is the most common form of dementia in our industrialized countries and is characterized by two pathological hallmarks, senile plaques and neurofibrillary tangles. Senile plaques and neurofibrillary tangles are mainly composed of Aβ amyloid peptide and hyperphosphorylated Tau protein, respectively.
Our research interests are focused on the mechanisms responsible for the development of tau pathology and the functional consequences of these cellular lesions. We have studied more specifically the role of glycogen synthase kinase-3ß in the development of tau pathology. We recently studied new transgenic lines expressing mutant tau proteins as models of tauopathies and these transgenic lines were used in experimental paradigms aimed at reducing tau pathology.
In order to investigate the potential link between Aβ peptide and Tau protein, we are also currently studying how reducing the expression of tau modulates the development of amyloid pathology in transgenic models expressing mutants APP and PS1 proteins. These models should improve our understanding of the interactions between amyloid or APP and tau proteins and their effects on cellular and behavioural phenotypes.

Selected publications
Schindowski, K., Bretteville, A., **Leroy, K**, Bégard, S., Brion, J. P., Hamdane, M., and Buée, L. (2006). Alzheimer disease-like tau neuropathology leads to memory deficits and loss of functional synapses in a novel mutated tau transgenic mouse without any motor deficits. American Journal of Pathology, 169: 599-616.

Leroy K, Yilmaz Z, Brion, JP (2007) Increased level of active GSK-3ß in Alzheimer's disease and accumulation in argyrophilic grains and in neurons at different stages of neurofibrillary degeneration, Neuropathology and Applied Neurobiology, 33: 43-55.

Leroy K, Bretteville A, Schindowski K, Gillissen E, Authelet M, De Decker R, Yilmaz Z, Buée L, Brion JP (2007) Early axonopathy preceding neurofibrillary tangles in mutant Tau transgenic mice. The American Journal of Pathology, 171: 976-992.

Leroy K, Ando K, Héraud C, Yilmaz Z, Authelet M, Boeynaems JM, Buée L, De Decker R, Brion JP (2009) Lithium treatment arrests the development of neurofibrillary tangles in mutant tau transgenic mice with advanced neurofibrillary pathology. *Journal of Alzheimer's Disease, 18, 1–18.*