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Passive immunization with novel monoclonal anti-PrP antibody TW1 in an Alzheimer's mouse model

Alzheimer's disease (AD) is a progressive neurodegenerative disorder with two hallmarks: Plaques seniles (PS) and Neurofibrillary tangles (NFTs). Recently, there has been much more focus on tau related pathology with the published failure of several amyloid β targeted therapeutic approaches in phase III clinical trials. Previously, we published the first active and passive immunization targeting tau pathology that was reproduced later by Citron's group at Eli Lilly as well as by other academic groups. It has been also reported that oligomeric species of tau are thought to be critical for the "prion-like" spread of pathology and for neuronal toxicity. We have first generated a novel monoclonal anti-PrP antibody (TW1) and developed a new transgenic model for AD that express mutated PS1 and all human tau isoforms on a murine tau knockout background (hTau/PS1 Tg mice). This model has an earlier onset (at < 3 months of age) and more rapid progression of tau pathology than prior htau mouse model. In this study, passive immunization with our new monoclonal antibody TW1 didn't show any difference in sensorimotor tests including traverse beam, rotarod and locomotor activity but improves cognitive decline in two cognitive tasks: short term memory (recognition object) mice treated spent more time with the novel object compared to the old object and differed significantly between both groups treatment effect between treated and non-treated animals showed a significant difference, One tailed t-test students p=0.025, and with closed field symmetrical maze (Day 1 two tailed, t-test P=0.0001; Day2 two tailed, t-test P=0.0015; Day3 two tailed, t-test P=0.0002). Reduction of tau pathology was observed in the motor cortex, hippocampus and piriform cortex of the animal treated with TW1 compared to controls. No significant difference was observed between both groups with GFAP antibody. Further analysis is underway. Passive immunization with a novel monoclonal anti-PrP decreases tau pathology by immunotherapy and improves cognitive decline in AD mouse model.

Biography

Allal Boutajangout obtained a PhD in Neuropathology from Free University of Brussels (ULB-Erasme Hospital), School of Medicine. He has completed his Postdoctoral training at New York University School of Medicine. He is a Research Associate Professor of Neurology and Neuroscience and Physiology. He is also the Chief of the Laboratory of Neurodegeneration and Drug Discovery Program within Center for Cognitive Neurology at NYU. He has received prestigious award Margaret M Cahn for his outstanding research in the field of Alzheimer's and other awards from: Alzheimer association, NIH pilot grant, Toyama Company, Revalesio Company and co-investigator in several RO1 NIH grants. He has published more than 30 papers in reputed journals and serves as a reviewer for many scientific journals.

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