Allal Boutajangout et al., J Neurol Neurophysiol 2016, 7:4 (Suppl)
http://dx.doi.org/10.4172/2155-9562.C1.035

Passive immunization with new Monoclonal antibody improves behavioral tasks and reduces tau pathology in Alzheimer's disease model

Allal Boutajangout[1,2,4], Hamed Habib[5], Haythum Tayeb[5], Hazem Atta[5], Abdulaziz Awwad[5], Ismail Almokayd[1], Kholod Alotaibi[1] and **Thomas Wisniewski[1,2,3]**
New York University Langone Medical Center, Departments of Neurology[1], Psychiatry[2], Pathology[3] and Physiology &Neuroscience[4]
King Abdulaziz University, School of Medicine, Jeddah, Saudi Arabia[5]

Background: Immunotherapy is emerging as promising approach to treat Alzheimer's disease. We have previously shown that prophylactic active and passive immunization clears tau aggregates from different area of the brain and prevent improves cognitive decline. Here we assessed if our new monoclonal antibody against PHF-Tau would be efficacious in tangles model of Alzheimer's disease. Methods: Homozygous P301L mice (2-2.5 months of age) were injected intraperitoneally one per week with our new monoclonal antibody 11B7 and controls received PBS only (200ug/125 µl; n=10 per group) for a total of 13 injections. Results: Treated animals (n=10) didn't show a significant difference on sensorimotor tasks (Traverse beam, Locomotor activity) while a significant difference was observed with the rotarod test (One tailed, t-student p=0.0489). Significant statistical differences were observed with the novel object recognition test (75% time spent with a novel objects (One tailed, t-student p=0.0004) 25% for controls (One tailed, t-student P>0.05), and with the closed field symmetrical maze (Day1 one tailed, t-test P=0.0001; Day2 one tailed t-student P=0001; Day3 one tailed t test P=0.0003). Immunohistochemistry analysis with PHF1 and CP13 showed a reduction of tau pathology in the cortex and in the hippocampus which correlates with the behavioral results. By Western blotting, the antibody therapy reduces tau pathology in the cortex and in the hippocampus. Further analysis is underway. Conclusion: passive immunization with our new monoclonal antibody decreases tau pathology by immunotherapy, biochemistry and improves cognitive decline in P301L mice.

Biography

Allal Boutajangout graduated from Free University of Brussels (ULB-Erasme Hospital), School of Medicine (PhD in Neuropathology). He completed his Postdoctoral training at New York University School of Medicine. He is a Research Associate Professor of Neurology and Neuroscience & Physiology and Psychiatry. He is also the chief of Neurodegeneration and Drug Discovery Program within Center for Cognitive Neurology at NYU. He 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 5 RO1 NIH grants. He has published more than 30 papers in reputed journals and serves as a reviewer for many scientific journals.

Allal.Boutajangout@nyumc.org

Notes: