Pathogenicity of neuronally-derived tau in exosomes

https://neurodegenerationresearch.eu/survey/pathogenicity-of-neuronally-derived-tau-in-exosomes/

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Research Abstract

Project Summary/Abstract Alzheimer’s disease (AD) is the most common form of dementia and is characterized neuropathologically by the presence of ?-amyloid (A?)-containing plaques and neurofibrillary tangles (NFTs) composed of phosphorylated tau protein. Symptomatic treatments for AD have been developed, but effective disease- modifying intervention is still needed.

Targets have been identified for disease-modifying drugs, but the results of clinical trials have been disappointing. Biomarkers of AD may improve clinical trial design and analysis, increasing the likelihood of successful drug development. The precise mechanisms of Tau release in AD are not completely understood, however some studies indicates that Tau might be released as aggregates in clear vesicles or membrane free. Of them, recent studies suggest that pathogenic forms of Tau might be released in exosomal vesicles that are positive for the L1 cell adhesion protein (LI CAM), suggesting that they are shed from neuronal cells from where they can traffic to the CSF and blood. Without specific preparation and handling for isolation of exosomes, these neuronally-derived exosomes (L1NE) containing Tau are not detectible in blood. We hypothesize that the trafficking of Tau in exosomes from the CNS to CSF and/or blood is an important pathway in identifying stages of AD and can serve to identify patients in preclinical stages when pathology is developing and no overt cognitive effects are seen.

Further information available at:

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