AGE-RELATED BIOMARKET CHANGES IN CEREBROSPINAL FLUID

https://www.neurodegenerationresearch.eu/survey/age-related-biomarket-changes-in-cerebrospinal-fluid/

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AGE-RELATED BIOMARKET CHANGES IN CEREBROSPINAL FLUID

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Research Abstract

? DESCRIPTION (provided by applicant): Age-related longitudinal change in cerebrospinal fluid biomarkers of Alzheimer disease Alzheimer disease (AD) is the most common cause of dementia in older adults. AD-related brain pathology, which includes the accumulation and deposition of amyloid-ß peptide and tau protein, begins ~10- 20 years before the onset of

dementia. Multiple failed drug trials for AD demonstrate that treatment late in the disease course, when patients have significant neuronal damage and dementia, is unlikely to be helpful. There is hope that treatment of early AD will be more effective. Significant efforts are underway to identify patients with “”pre-clinical AD,”” those who have early AD-related brain pathology but are still cognitively normal, using fluid biomarkers or imaging tests. Leading fluid biomarkers for AD include low levels of amyloid-ß 42 (Aß42) and high levels of tau and phosphotau181 (ptau) in the cerebrospinal fluid (CSF). However, there is large person-to-person variation in levels of CSF Aß42, tau and ptau, and no clear cut-offs for diagnostic purposes. Measuring CSF biomarkers at multiple time points, then quantifying their rates of change, may correct for some of the individual variation in baseline CSF biomarker levels and provide a superior predictor of AD-related brain pathology. In this study, levels of CSF Aß40, Aß42, tau and ptau will be measured in individuals who have undergone two or more CSF collections. CSF biomarker trajectories will be plotted for individuals representing the entire adult lifespan (age 20-84). Th time course of CSF biomarker changes will be defined, including when CSF Aß42 levels begin to decline (consistent with amyloid deposition) and when CSF tau and ptau levels begin to increase (reflecting neuronal injury). Statistical methods will be used to determine whether the rate of change in biomarker levels, rather than single baseline measures, better predict neuronal injury, poor performance on cognitive tests, and progression to AD dementia. If the rate of change in biomarker levels, rather than baseline concentrations alone, better predicts AD dementia and related measures, this would impact efforts to identify individuals with pre-clinical AD for clinical trials and may affect clinical diagnostic testing for AD dementia.

Further information available at:

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