Brain Sleep Clearance of Amyloid-Beta Peptides Study (Brain SCRAPS)

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USA

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Brain Sleep Clearance of Amyloid-Beta Peptides Study (Brain SCRAPS)

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Research Abstract

? DESCRIPTION (provided by applicant): Alzheimer’s disease (AD) is a common neurodegenerative disease characterized by the accumulation of amyloid plaques and neurofibrillary tangles. Recent studies support the hypothesis that amyloid beta (Aß) dynamics

in the brain are influenced by the sleep-wake cycle, with increases in the production of soluble Aß during wakefulness and decreases during slow wave sleep (SWS). In this model, prior to amyloid deposition, brain soluble Aß levels may be relatively increased in the elderly primarily due to loss of total sleep time and slow wave sleep (SWS) that occur with normal aging and/or secondarily, due to sleep disturbances such as Sleep Disordered Breathing (SDB) or insomnia that are common in late life. We have preliminary evidence showing that: a) SDB advances cognitive decline in normal elderly; b) SDB increases cerebrospinal fluid (CSF) Aß42 levels in middle age adults; and, c) increased CSF Aß42 is associated with reduced SWS in normal elderly. Our goal is to test this hypothesis in 20 cognitively normal elderly with no SDB or brain amyloid (Aim 1), and a group of 22 middle age adults with severe SDB treated with therapeutic continuous positive airway pressure (CPAP) and good treatment compliance (Aim 2). In the elderly group, we will evaluate the relationship between SWS and CSF Aß42/Aß40 ratio in the absence of SDB or amyloid burden (measured with a 18F-florbetaben PET scan). In the middle age group, we will disrupt sleep by withdrawing CPAP on one night and allow participants to sleep with therapeutic CPAP on a second night. A morning lumbar puncture will be performed in both visits to evaluate the effect of disrupting sleep by acute CPAP withdrawal on CSF Aß42 levels. This project will be the first to explore the protective effect of SWS on Aß42 dynamics in a group of elderly subjects as well as the effect of acute sleep disruption by CPAP withdrawal on CSF Aß42 levels in a well- characterized clinical sample of severe middle age obstructive SDB patients. This proposal may identify: 1) evidence of age-related SWS loss effects on CSF Aß42 dynamics; 2) a mechanism by which a highly prevalent sleep disorder may contribute to AD pathology; and, 3) SWS as new therapeutic target for AD prevention.

Further information available at:

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