

Damaged protein identified as early diagnostic biomarker for Alzheimer's disease

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Researchers at NYU School of Medicine have found that elevated cerebrospinal fluid levels of phosphorylated tau231 (P-tau231), a damaged tau protein found in patients with Alzheimer's disease, may be an early diagnostic biomarker for Alzheimer's disease in healthy adults.

The study published this month online by *Neurobiology of Aging* shows that high levels of P- tau231 predict future <u>memory decline</u> and loss of brain gray matter in the medial temporal lobe- a key memory center. Prior studies found the medial temporal lobe to be the most vulnerable brain region in the early stages of Alzheimer's disease accumulating damaged tau proteins in the form of neurofibrillary tangles. Tangles are one of the signature indicators of Alzheimer's disease, in addition to beta amyloid plaques.

"Our research results show for the first time that elevated levels of P-tau 231 in normal individuals can predict memory decline and accompanying brain atrophy," said lead author Lidia Glodzik MD, PhD, assistant research professor, Department of Psychiatry at the Center for Brain Health and Center of Excellence on Brain Aging at NYU School of Medicine. "Our findings suggest that P-tau231 has the potential to be an important diagnostic tool in the pre-symptomatic stages of Alzheimer's disease."

Researchers evaluated 57 cognitively healthy <u>older adults</u> and studied the relationships between baseline cerebrospinal fluid biomarkers, longitudinal memory performance and longitudinal measures of the



medial temporal lobe gray matter using <u>Magnetic Resonance Imaging</u>, or MRI. Two years later, researchers found that 20 out of 57 healthy adults showed decreased memory performance. The group with worsened memory had higher baseline levels of P-tau231 and more atrophy in the medial temporal lobe. The higher P-tau231 levels were associated with reductions in medial temporal lobe <u>gray matter</u>. Authors concluded that elevated P-tau231 predicts both memory decline and medial temporal lobe atrophy.

"Indentifying people at risk for Alzheimer's disease is the necessary first step in developing preventive therapies," said co-author Mony de Leon, EdD, professor, Department of Psychiatry and director of the Center for Brain Health at the Center of Excellence on Brain Aging at NYU School of Medicine and Research Scientist at the Nathan S. Kline Institute for Psychiatric Research. "This study shows that <u>Alzheimer's disease</u> pathology may be recognized in the normal stages of cognition. This observation may be of value in future studies investigating mechanisms that cause or accelerate dementia".

Provided by New York University School of Medicine

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