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Study identifies toxic key to Alzheimer's disease memory loss

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Using new scientific techniques, scientists have unlocked the cascade of molecular events that lead to Alzheimer's disease. The scientific findings published in the latest edition of Nature Medicine suggest a potential new target for the development of drug therapies to fight the irreversible and degenerative disease which affects some 29.8 million people worldwide. The total worldwide societal cost of dementia was estimated at somewhere in the region of US$315.4 billion in 2005.

Alzheimer's disease is marked by the build-up of plaques consisting of beta-amyloid protein fragments, as well as abnormal tangles of tau protein found inside brain cells. Early in the disease, Alzheimer's pathology is first observed in the hippocampus, the part of the brain important to memory, and gradually spreads to the cerebral cortex, the outer layer of the brain.

The team of Irish and international researchers have identified that the accumulation of a particular protein (called amyloid ß-protein - Aß) in the brain initiates Alzheimer's disease and that it directly alters the structure and function of brain cells. The findings place a significant emphasis on the development of new therapeutic strategies targeted at the reduction of the formation of Aß as opposed to the reduction of the plaque burden associated with the disease.

"Alzheimer's disease is a major personal and societal tragedy," says Professor Ciaran Regan from the UCD School of Biomolecular and Biomedical Science, University College Dublin, one of the co-authors of

the report. “The disease progression is torturously long and debilitating, extorting a huge emotional and economic cost.”

“The onset of the disease is insidious with the earliest symptoms often manifested as subtle and intermittent deficits of episodic memory,” explains Professor Dominic Walsh, associate Professor of Pharmacology at the UCD Conway Institute, University College Dublin, another co-author of the report.

“Our findings support the growing theory that Alzheimers’s disease memory deficits may result from loss of dendritic spines and that this process is mediated by amyloid ß–protein (Aß) oligomers, not monomer or plaque Aß as previously considered.”

Source: University College Dublin