TCD-led team’s breakthrough could aid Alzheimer’s treatment

Findings add to understanding of how neurodegenerative diseases are triggered

by Ruth O’Sullivan

A group of researchers from the University College Dublin (UCD) School of Pharmacy has made a major breakthrough in understanding how Alzheimer’s disease begins.

The research, which was published in the journal PNAS, could help to identify new targets for the treatment of Alzheimer’s disease.

Alzheimer’s disease is a neurodegenerative disease that affects millions of people worldwide. It is characterized by the formation of amyloid plaques and neurofibrillary tangles in the brain, leading to memory loss and other cognitive impairments.

The team, led by Prof. Martin Heegaard, a professor of medicinal chemistry at UCD, discovered that the amyloid precursor protein (APP), which is the protein that is cleaved to form the toxic amyloid beta peptide, is regulated by a series of protein-protein interactions.

The researchers found that the APP is cleaved by a protease called beta-secretase (BACE1), which is regulated by the protein disulfide isomerase (PDI).

“Understanding this system at the single-molecule level required a highly ambition and multidisciplinary approach that pushes the boundaries of what is technically possible,” said Prof. Heegaard.

The team’s findings have implications for the development of new therapeutic strategies for Alzheimer’s disease. They suggest that targeting BACE1 and PDI interactions could help to prevent the formation of toxic amyloid plaques and slow the progression of the disease.

“This is a major breakthrough in our understanding of how Alzheimer’s disease begins,” said Prof. Heegaard. “It opens up new possibilities for the development of targeted therapies that could halt or even reverse the disease.”

The research was funded by the Science Foundation Ireland, and the team is currently working to develop new drugs that target the interactions between BACE1 and PDI.

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