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Better understanding of the cause of Alzheimer's disease: New suggestion for a possible treatment

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Alzheimer's disease is the most common form of dementia, affecting over 35 million people worldwide. It is generally assumed that the clumping of beta-amyloid (Aß) protein causes neuronal loss in patients. Medication focuses on reducing Aß42, one of the most common proteins and the most harmful. University of Twente PhD student Annelies Vandersteen is refining the current approach. She explains: "The results of my research provide a broader understanding of the processes that lead to Alzheimer's disease and in this way may help to bring about new medication".

The Aß protein occurs in the body in various lengths, ranging from 33 to 49 amino acids. The shorter varieties are regarded as 'safe', unlike the longer ones – Aß42 and longer – which are highly aggregating. Current therapeutic strategy tries to reduce the clumping of Aß42, and its harmful effects, by limiting the release of Aß42. Reducing Aß42 production at the same time results in a rise in Aß38 levels. Vandersteen comments: "One of the findings of my research is that small amounts of Aß38 can in fact increase or temper the clumping and toxic effects of longer Aß proteins. The processes that result in Alzheimer's disease are determined by the whole spectrum of Aß proteins. So the picture is far less black and white than has been assumed so far, and less common forms of Aß are far less harmless than we thought."

Vandersteen examined the protein mixtures in a laboratory situation. She

devised a series of experiments based on a computer-calculated hypothesis. The behaviour of the various Aß proteins and mixtures was studied in detail and described using various biophysical techniques. The influence of the various Aß proteins and mixtures on neurons was then studied in a cell culture.

Annelies Vandersteen's PhD research was carried out as part of a triple degree at the University of Twente, the Catholic University of Leuven and the Vrije Universiteit Brussel. The study falls within the work of the MESA+ and MIRA research institutes of the University of Twente, Faculty of Science and Technology, Nanobiophysics Group. The thesis 'Aggregation and toxicity of amyloid-beta peptide in relation to peptide sequence variation' is available on request.

Provided by University of Twente